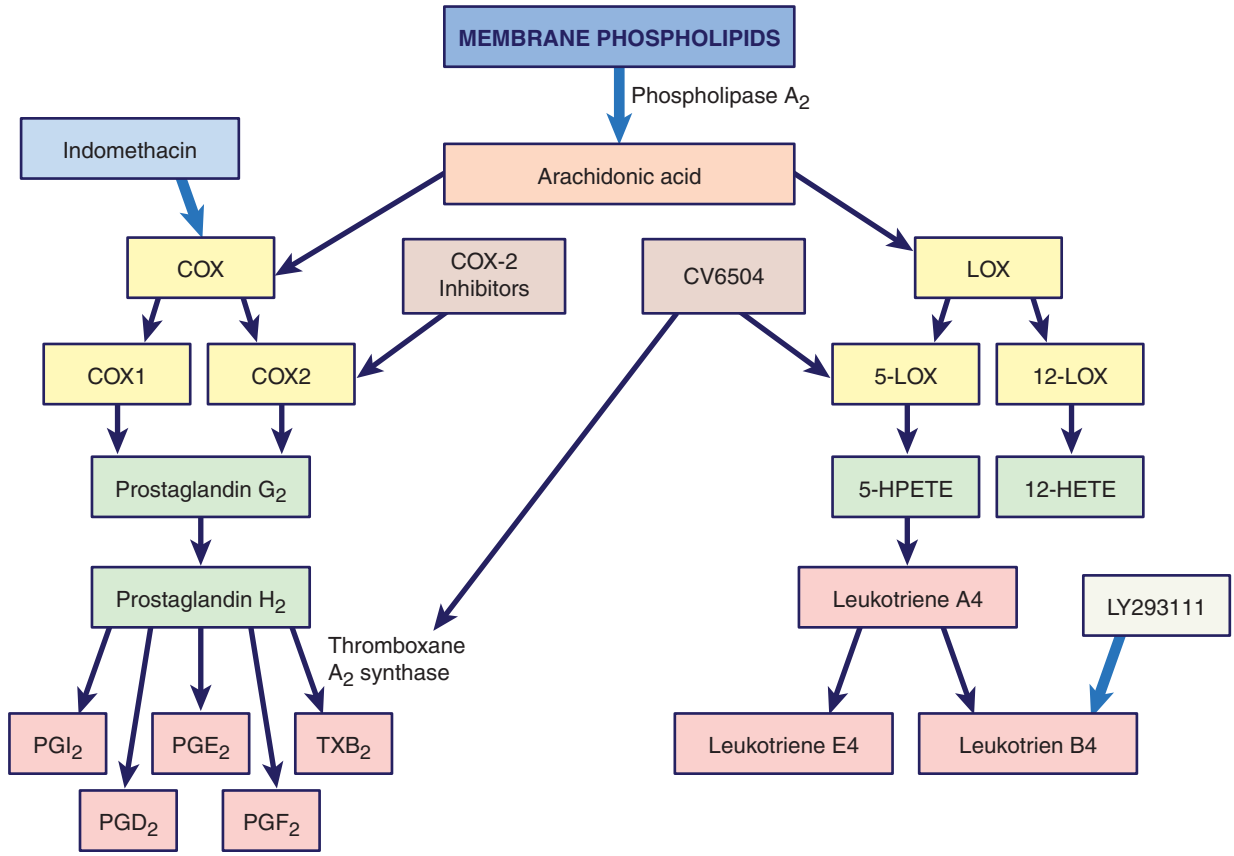

acid-derived eicosanoids (prostaglandins, thromboxanes, leukotrienes, and other oxidized derivatives), other inflammatory agents (e.g., reactive oxygen species), and adhesion molecules (Calder, 2006). Three major types of omega-3 fatty acids are ingested in foods: alpha linolenic acid (ALA), eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA). The body converts ALA to EPA and DHA, which are readily used by the body. Omega-3 fatty acids help reduce inflammation, while omega-6 fatty acids tend to promote inflammation. The precursor ALA does not appear to exert anti-inflammatory effects at achievable intakes (Calder, 2006).

A balance between omega-3 and omega-6 fatty acids in the diet is needed. The proper balance helps maintain and even improve health; one to four times more omega-6 fatty acids than omega-3 fatty acids is desirable, yet people who follow a Western diet consume a higher percentage of omega-6 fatty acids than they should.

Long-chain omega-3 polyunsaturated fatty acids (PUFAs) act by replacing arachidonic acid as an eicosanoid substrate, inhibiting arachidonic acid metabolism; by altering the expression of inflammatory genes through effects on transcription factor activation; and by leading to anti-inflammatory mediators known as resolvins (Calder, 2006).

Role of Phytochemicals and Total Diet

Phytochemicals known for their ability to protect tissue also appear to block the activity of an enzyme that triggers inflammation in joints. See Table 11-2.

Complementary and Alternative Medicine (CAM) Therapies

Controlled scientific studies of many patients can prove that a particular treatment is beneficial or that an apparent improvement is incidental. The important consideration is that treatment should do no harm.

Some studies have been done in alternative therapies, particularly diet in the treatment of arthritis, but none have shown any real long-term benefit. Patients often do benefit from complementary therapies, either because the treatment truly works or because of psychological (placebo) effects. While there is evidence of benefit for vitamin C, vitamin D, and nutraceuticals such as glucosamine, chondroitin, *S*-adenosylmethionine, ginger, and avocado/soybean unsaponifiables (McAlindon, 2006), specific diets and herbal or botanical products should only be used with medical consultation.

While the best nutrition-based strategy for promoting optimal health and reducing the risk of chronic disease is to wisely choose a wide variety of foods, additional nutrients from supplements can help some people meet their nutrition needs (American Dietetic Association, 2009). Physicians reported familiarity with acupuncture (80%), yoga (74%), and Tai-Chi (72%) yet almost all of their patients use CAM therapies (Mak et al, 2009). It is logical, then, that dietetics practitioners must keep up to date on the efficacy, safety, and the regulatory issues in order to provide the best advice.

TABLE 11-2 Phytochemicals and Dietary Factors Affecting Rheumatic Disorders

Component	Foods or Ingredients	Role
Cruciferous vegetables: broccoli, cauliflower, cabbage, bok choy	Sulforaphane	Boost phase 2 enzymes
Dairy products, low fat	To be identified; vitamin D?	Protective factors against gout (Choi, 2005).
Fruits: pomegranate, cranberry	Anthocyanins, tannins; ellagic acid; resverarol; quercetin; vitamins A, C; selenium	Potent anti-inflammatory activity (Rasheed et al, 2009).
Long-chain polyunsaturated fatty acids	EPA and DHA	Replace arachidonic acid as an eicosanoid substrate, inhibiting arachidonic acid metabolism. Alter expression of inflammatory genes through effects on transcription factor activation, leading to anti-inflammatory mediators termed resolvins (Calder, 2006).
Mediterranean diet	Resveratrol, olive oil, lower intake of red meat	Protects against severity of rheumatoid arthritis (Choi, 2005).
Spices	Turmeric (curcumin) Red pepper (capsaicin) Cloves (eugenol) Ginger (gingerol) Cumin, anise, and fennel (anethol) Basil, rosemary (ursolic acid) Garlic (diallyl sulfide, ajoene, *S*-allylmercaptocysteine)	Interrupts pathway for transcription factor-κB (Aggarwal and Shishodia, 2004).
Vitamin D	Hormone affects over 2000 genes	Needed for healthy immune system, gene expression, strong bones.
Total protein and purine-rich vegetables	Neutral	Do not tend to promote gout (Choi, 2005).
Vitamin E, beta-carotene, and retinol.	Neutral	Have NOT been shown to halt the progression of rheumatic disorders
Red meats, seafood, beer, and liquor	Undesirable	Tend to promote symptoms of gout, inflammatory polyarthritis, or rheumatoid arthritis (Choi, 2005).

Sources: Aggarwal BB, Shishodia S. Suppression of the nuclear factor-kappa B activation pathway by spice-derived phytochemicals: reasoning for seasoning. *Ann N Y Acad Sci.* 1030:434, 2004.

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McAlindon TE. Nutraceuticals: do they work and when should we use them? *Baillieres Best Pract Res Clin Rheumatol.* 20:99, 2006.

Nash PT, Florin TH. Tumour necrosis factor inhibitors. *Med J Aust.* 183:205, 2005.

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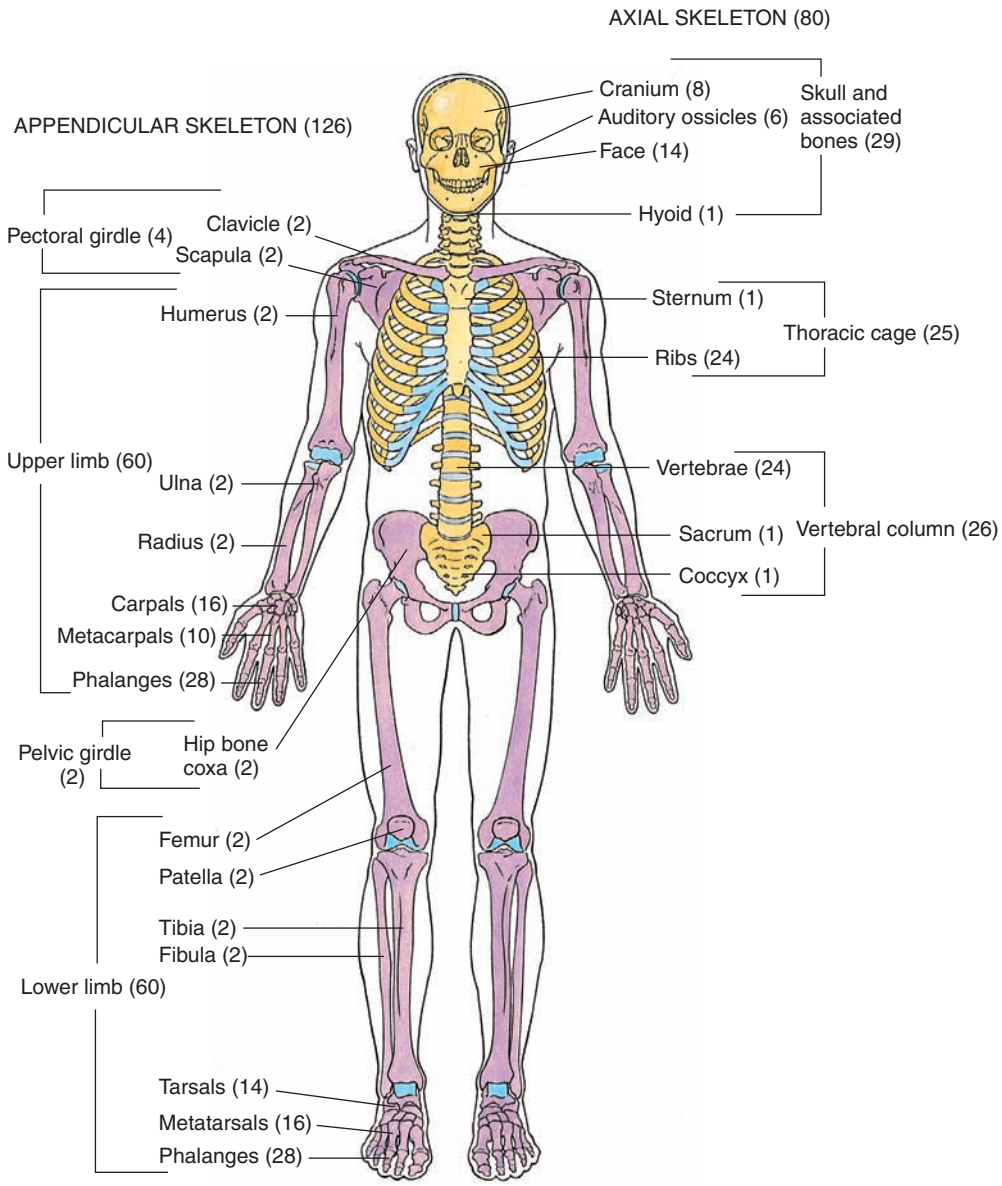
OVERVIEW—BONE DISORDERS



Adapted from: Moore KL, Agur AMR. *Essential Clinical Anatomy,* 2nd ed. Baltimore. Lippincott Williams & Wilkins

Bones are living, growing, and changing parts of the body. The human skeletal system consists of bones, cartilage, ligaments, and tendons and accounts for about 20% of the body weight. Osteoblasts are bone-forming cells, osteoclasts resorb or break down bone, and osteocytes are mature bone cells. The osteoblast is an endocrine cell type.

There is a reciprocal regulation of bone and energy metabolism by leptin and osteocalcin. Leptin inhibits insulin secretion by beta cells while osteocalcin favors it (Hinoi et al, 2009). Leptin deficiency leads to increased osteoblast activity and increased bone mass. Expression of the Esp gene, exclusive to osteoblasts, regulates glucose homeostasis and adiposity through controlling osteoblastic secretion of osteocalcin (Wolf, 2008). Osteocalcin deficiency leads to decreased insulin and adiponectin secretion, insulin resistance, higher serum glucose levels, and increased adiposity (Wolf, 2008). This recently understood concept has implications for diabetes and the metabolic syndrome.

There are 206 bones in the adult skeleton. The two types of bone tissue (compact and spongy) differ in density. Bone strength is derived from quantity (density and size) and quality (structure, consistency, and turnover). Bone mass is dependent upon individual genetic background. Adequate nutrient intake is needed from birth to achieve maximal bone mass and to prevent osteoporosis later in life.

The trace elements, calcium and phosphorus, are involved in skeletal growth. Parathyroid hormone (PTH) regulates calcium and bone homeostasis; it is expressed in the placenta, regulates the placental expression of genes involved in calcium and other solute transfer, and may directly stimulate placental calcium transfer (Simmonds et al, 2010).

Magnesium and fluoride are matrix constituents while zinc, copper and manganese are components of enzymatic systems involved in matrix turnover. A sufficient protein intake, along with adequate calcium, supports stronger

TABLE 11-3 Recommendations for Prevention of Osteoporosis

Get the recommended amounts of calcium and vitamin D_3 for age and sex; use supplements when diets are inadequate.

Maintain a healthy weight and be physically active 30+ minutes a day for adults and 60+ minutes a day for children, including weight-bearing activities to improve strength and balance.

Minimize the risk of falls by removing items that might cause tripping, improving lighting, and encouraging regular exercise and vision tests to improve balance and coordination.

Risks for patients of all ages should be evaluated by health care professionals. Obtain bone density tests for women over the age of 65 and for any man or woman who suffers even a minor fracture after the age of 50. "Red flags" for someone is at risk include a history of multiple fractures, those who take certain medications, and those who have a disease that can lead to bone loss.

A BMD test is used to detect osteoporosis before fractures occur, predict chances of future fractures, or determine rate of bone loss and monitor the effects of treatment. The DEXA scan is most common.

- Normal BMD: within 1 standard deviation (SD) of a "young normal" adult.
- Low bone mass (osteopenia): BMD is between 1 and 2.5 SD below that of a "young normal" adult.
- Osteoporosis: BMD is 2.5 SD or more below that of a "young normal" adult.

bone density; this fact contradicts past suggestions that high-protein diets deplete bone strength.

Changes in bone turnover markers may become accurate predictors of fracture risk. Assessing risk factors for low bone mass is important in monitoring the etiology of fracture in older individuals (Kelsey et al, 2006). In general, women's bone health has been studied more extensively than that of men. Studies on the predictors of fractures in men are needed, such as bone architecture, morphology, biochemical markers of bone turnover, and hormonal levels (Szulc et al, 2005).

Vitamins are important. Vitamin D_3 plays a role in calcium metabolism. Vitamins C and K are cofactors of key enzymes for skeletal metabolism. Another indicator of bone health is heart health. There are similar pathophysiological mechanisms underlying cardiovascular disease (such as dyslipidemia, oxidative stress, inflammation, hyperhomocysteinemia, hypertension, and diabetes) and low bone mineral density (BMD). Sufficient folic acid, vitamins B_6 and B_{12} can help improve bone health by lowering elevated homocysteine levels. Antioxidant nutrients, including vitamins A and C and selenium, play a role in bone health.

While calcium is widely recognized for bone health, other minerals are equally important. Iron promotes production of collagen in bone structure; 18 mg is most protective for women but balance is also critical as too much iron may throw off calcium balance. Finally, silicon in the form of choline-stabilized orthosilicic acid is the bioavailable form that enhances calcium and vitamin D_3 in bone health.

Omega-3 fatty acids such as EPA help increase levels of calcium in the body, deposit calcium in the bones, and improve bone strength. People who are deficient in EFAs EPA and gamma linolenic acid (GLA) are more prone to bone loss.

Former U.S. Surgeon General Richard H. Carmona (2005) warned in a landmark report that, by 2020, half of all American citizens older than 50 would be at risk for fractures from osteoporosis and low bone mass if immediate action is delayed by individuals at risk, doctors, health systems, or policymakers. At least 10 million Americans over the age of 50 have osteoporosis, another 34 million are at risk for developing osteoporosis, and roughly 1.5 million people have suffered a bone fracture related to osteoporosis. About 20% of senior citizens who suffer a hip fracture die within a year of fracture; another 20% of individuals with a hip fracture end up in a nursing home. Hip fractures account for 300,000 hospitalizations each year. See Table 11-3 for recommendations to prevent osteoporosis.

For More Information

- American Academy of Orthopaedic Surgeons
  http://www.aaos.org/
- American Academy of Physical Medicine and Rehabilitation
  http://www.aapmr.org
- American Autoimmune-Related Diseases Association (AARDA)
  http://www.aarda.org/
- American College of Rheumatology
  http://www.rheumatology.org/
- American Osteopathic Association
  http://www.do-online.osteotech.org/
- American Pain Foundation
  http://www.painfoundation.org/
- American Society for Bone and Mineral Research
  http://www.asbmr.org/
- Arthritis Foundation
  http://www.arthritis.org/
- Autoimmunity Resources
  http://www.aarda.org/links.php
- CAM Therapy Resources
  http://nccam.nih.gov/health/bydisease.htm
- CDC—Calcium for Bone Health
  http://www.cdc.gov/nutrition/everyone/basics/vitamins/calcium.html
- Clinical Trials Research Trials
  http://www.aarda.org/links.php
- Drug List
  http://www.rxlist.com/alternative.htm
- Journal of Immunology
  http://www.jimmunol.org/
- National Institute of Arthritis and Musculoskeletal and Skin Disorders
  http://www.niams.nih.gov/hi/index.htm
- Quack Watch for Unproven Remedies
  http://www.quackwatch.com/
- Rheumatic Diseases Internet Journal
  http://www.rheuma21st.com/

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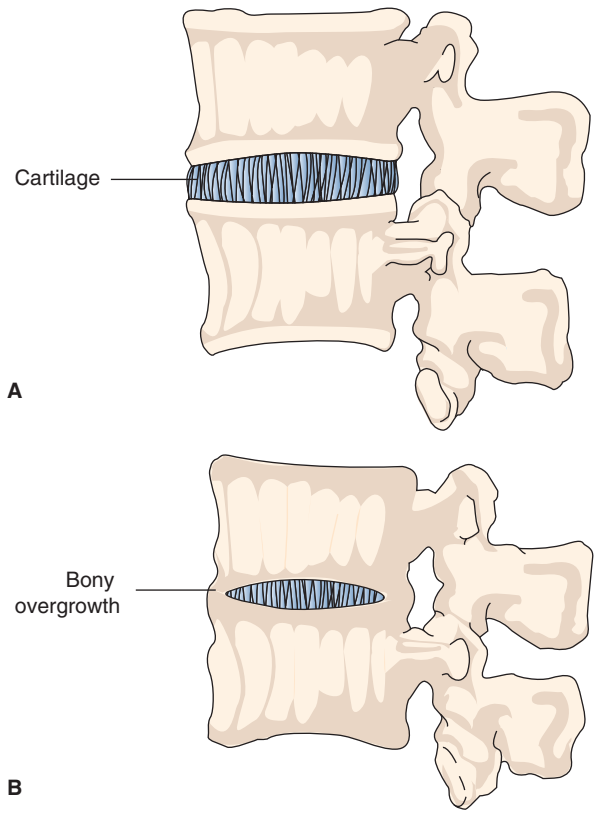
ANKYLOSING SPONDYLITIS (SPINAL ARTHRITIS)

NUTRITIONAL ACUITY RANKING: LEVEL 1



Carol Mattson Porth, *Pathophysiology Concepts of Altered Health States*, 7th ed. Philadelphia: Lippincott Williams & Wilkins, 2005.

DEFINITIONS AND BACKGROUND

Among the 100 different rheumatic diseases that affect the joints and muscles is a group of five called **spondyloarthropathies**. These include ankylosing spondylitis, reactive arthritis (Reiter's syndrome), psoriatic arthritis or spondylitis, spondylitis of inflammatory bowel disease, and undifferentiated spondyloarthropathy. Spondylitis is inflammation of the joints linking the vertebrae (a fused spine is not uncommon). Spondylitis affects about 300,000 Americans and is more common in Caucasians than in African Americans. The condition is most common in men aged 16–35 years and may run in families.

In **ankylosing spondylitis**, inflammation of connective tissue recedes but leaves hardened and damaged joints that fuse together the bones of the spinal column. The sacroiliac joints generally are affected first. Symptoms and signs include chronic lower back pain, early morning stiffness in the lower back where the lower spine is joined to pelvis, vague chest pains, tender heels, weight loss, anemia, anorexia, slight fever, recurring iritis or reddened eyes, valvular heart disease. Pain may occasionally start in the knees and shoulders. There is a strong link between the bowel and the osteo-articular system, notably with the HLA-B27 gene where there are symptoms such as abnormal antigen presentation, the presence of autoantibodies against specific antigens shared by the colon and other extra-colonic tissues, increased intestinal permeability, osteoporosis and osteomalacia secondary to IBD (Rodriguez-Reyna et al, 2009).

Elevated tumor necrosis factor alpha (TNFα) is believed to be one of the causes of inflammation and bone destruction (Braun et al, 2006); therefore, anti-TNF therapy is effective (Barkham et al, 2005). Exercise to strengthen muscles that tend to cause pain on stooping or bending may be useful to relieve lower back pain. Attention to good posture will reduce some types of pain. Surgery may be needed to replace a joint or to relieve pain.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Genetic marker HLA-B27 can be detected in these individuals.

Clinical/History

- Height
- Weight
- Body mass index (BMI)
- Weight changes
- Anorexia
- Fever?
- Lower back pain
- Pain in knees or shoulders
- Iritis or reddened eyes
- X-rays

Lab Work

- HLA-B27 gene test (positive in 90%)
- Erythrocyte sedimentation rate (ESR) (high)
- C-reactive protein (CRP)
- Ca^{++}, Mg^{++}
- Na^{+}, K^{+}
- Alkaline phosphatase (Alk phos)
- Blood urea nitrogen (BUN)
- Creatinine (Creat)
- Phosphorus (P)
- Hemoglobin and hematocrit (H & H)
- Aspartate aminotransferase (AST)
- Alanine aminotransferase (ALT)
- Serum folate and B_{12}
- Homocysteine levels
- Vitamin D_3 status (serum 25-OHD)

INTERVENTION

OBJECTIVES

- Reduce pain, inflammation, and disease activity; support improved functioning and ability to work or to maintain quality of life.
- Correct anorexia, nausea, poor intake or weight loss, anemia, or fever where present.

SAMPLE NUTRITION CARE PROCESS STEPS

Unintentional Weight Loss

Assessment: Loss of 15 lb this past 6 months, much pain and inflammation with ankylosing spondylitis, taking numerous medicines that cause GI distress and anorexia.

Nutrition Diagnosis (PES): *Unintentional weight loss* (NC-1.4) related to pain, inflammatory processes, and GI distress and evidenced by 15-lb unplanned weight loss in past 6 months.

Interventions:
Food and Nutrient Delivery: ND 1.2 Alter diet as tolerated. ND 32.3 and 32.4 Initiate vitamin and mineral supplementation.

Education: E-1.2 Discuss ways to increase energy and nutrient density in food choices.

Counseling: C 2.2 Agree to goal of consuming only nutrient-dense foods for the coming month until next visit. C-2.3 Keep a food diary for one month.

Monitoring and Evaluation: Review food diary after 1 month. Monitor weight for resolution of weight loss; goal is gain of 1–2 lb weekly.

- Improve ability to participate in physical activities of choice to maintain lean body mass.

FOOD AND NUTRITION

- A normal diet is useful. Support gradual weight loss, if needed, to normalize weight. Some patients claim relief while using a vegetarian diet with less red meat.
- Preferred foods should be offered to stimulate appetite.
- Increase intake of foods rich in antioxidants such as vitamins E and C, selenium, and fish oils for rich sources of omega-3 fatty acids. Sufficient calcium and vitamin D are also important.
- Include phytochemicals derived from spices such as turmeric (curcumin); red pepper (capsaicin); cloves (eugenol); ginger (gingerol); cumin, anise, and fennel (anethol); basil and rosemary (ursolic acid); garlic (diallyl sulfide, *S*-allylmercaptocysteine, ajoene); and pomegranate (ellagic acid) (Aggarwal and Shishodia, 2004).

Common Drugs Used and Potential Side Effects

- Sulfasalazine, methotrexate, azathioprine, cyclosporine, leflunomide, and tumor necrosis factor-alpha blocking agents can be considered as first-line therapy but there are possible harmful effects on intestinal integrity, permeability, and even on gut inflammation (Rodriguez-Reyna et al, 2009).
- Etanercept (Enbrel), an anti-TNF therapy, may improve mobility and quality of life (Braun et al, 2006; Davis et al, 2005; Temel et al, 2005). Infliximab (Remicade), another monoclonal antibody, also targets TNFα and provides clinical improvement. Upper respiratory infections, psoriatic rashes, and allergic reactions can occur.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician. Ginger, corn, pineapple, and pigweed have been recommended; no clinical trials prove efficacy.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Exercise is crucial, especially swimming, to relieve back pain.
- Patient should practice deep breathing exercises for pain relief. Stretching and strengthening exercises also are important.
- Patient will likely find that sleeping on a hard bed, supine, is most helpful.
- Discuss role of energy intake for weight control.

Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

For More Information

- Ankylosing Spondylitis International Federation
  http://www.asif.rheumanet.org/
- National Ankylosing Spondylitis Society (NASS)–United Kingdom
  http://www.nass.co.uk/
- Spondylitis Association of America
  http://www.spondylitis.org

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Davis JC, et al. Reductions in health-related quality of life in patients with ankylosing spondylitis and improvements with etanercept therapy. *Arthritis Rheum.* 53:494, 2005.

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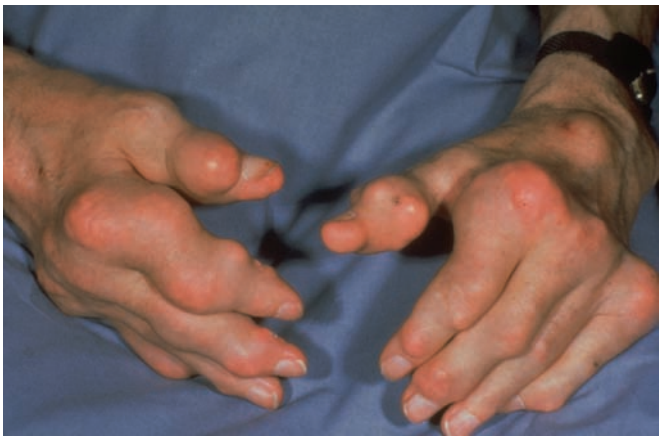
GOUT

NUTRITIONAL ACUITY RANKING: LEVEL 2



Adapted from: Rubin E MD and Farber JL MD. *Pathology,* 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 1999.

DEFINITIONS AND BACKGROUND

Uric acid is the end product of purine metabolism. Because humans have lost hepatic uricase activity, this leads to uniquely high serum uric acid concentrations when compared with other mammals. About 70% of daily urate disposal occurs via the kidneys; in 5–25% of the human population, impaired renal excretion leads to hyperuricemia.

Gout is a disorder of sudden and recurring attacks of painful arthritis with inflamed joints (usually the big toe, ankle, knees, and feet). Hyperuricemia promotes deposition of monosodium urate crystals in the joints and tendons. Gout affects more than 1% of adults in the United States and is the most common form of inflammatory arthritis among men (Saag and Choi, 2006). The disease tends to affect men between the ages of 30 and 50 years and is often hereditary.

Risks include genetic factors; high intake of seafood and red meats (Choi et al, 2005; Johnson et al, 2005) as well as beer and fructose (Doherty, 2009). Higher intakes of coffee, low-fat dairy products, and vitamin C are associated with lower risk (Doherty, 2009). See Table 11-4 for other etiologies of hyperuricemia. Gout prevalence increases in direct association with age, metabolic syndrome, hypertension, and use of thiazide diuretics (Saag and Choi, 2006). There is increased incidence in postmenopausal women, with polyarticular onset, hand involvement, and development of tophi (Ene-Stroescu and Gorbien, 2005). Tophi are hard lumps of urate crystals that are deposited under the skin around the joints and may be permanent.

Acute attacks may be triggered by surgery, sudden and severe illness, fasting, chemotherapy, or joint injury. Acute gout most commonly affects the first metatarsal joint of the foot, but other joints may also be involved. The joint swells, and skin turns warm, red, purplish, and shiny. Severe pain usually occurs, more so at night.

Gout progresses from asymptomatic hyperuricemia to acute gouty arthritis, intercritical gout (intervals between acute attacks), and finally to chronic tophaceous gout. Tophi may develop if the condition goes untreated. Although attacks of gout can subside in a few days, repeated attacks can cause permanent joint damage, and the disease often results in substantial disability and frequent medical care.

Treatment includes the pain-relieving NSAIDs and, for more serious outbreaks, corticosteroids. Most patients with gout eventually require long-term treatment with medications that lower blood uric acid levels. Patients with asymptomatic hyperuricemia should lower their urate levels by changes in diet or lifestyle.

TABLE 11-4 Acquired Causes of Hyperuricemia

Cause	Description
Increased urate production	
Nutritional	Excess ethanol or fructose intake
Hematological	Myeloproliferative and lymphoproliferative disorders, polycythemia
Drugs	Ethanol, cytotoxic drugs, vitamin B_{12} (treatment of pernicious anemia)
Miscellaneous	Obesity, psoriasis, hypertriglyceridemia
Decreased renal excretion of urate	
Drugs	Ethanol, cyclosporine (Sandimmune), thiazides, furosemide (Lasix) and other loop diuretics, ethambutol (Myambutol), pyrazinamide, aspirin(low-dose), levodopa (Larodopa), nicotinic acid (Nicolar)
Renal	Hypertension, polycystic kidney disease, chronic renal failure (any etiology)
Metabolic/endocrine	Dehydration, lactic acidosis, ketosis, hypothyroidism, hyperparathyroidism
Miscellaneous	Obesity, sarcoidosis, toxemia of pregnancy

Adapted from: Harris M, et al. Gout and hyperuricemia. *Am Fam Physician.* 59:925, 1999.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: About 10% of people with hyperuricemia develop gout. Genetic variants within the transporter gene, SLC2A9 (GLUT9), affect both fructose and uric acid transport. Other renal urate transporters have been identified, including URAT1.

Clinical/History

- Height
- Weight
- BMI
- Obesity
- Swollen, painful big toe (podagra)
- Arthritis
- Urate crystals in urine
- Use of thiazide diuretics?

Tophus, suspected or proven
Asymmetrical swelling within a joint on X-ray

Birefringent crystals in the synovial fluid
BUN (increased)
Cholesterol (Chol)
Triglycerides (increased?)

Ca^{++}, Mg^{++}
Na^{+}, K^{+}
Albumin (Alb)
Creat
Glucose (Gluc)
AST, ALT
Vitamin D_3 status (serum 25-OHD)

Lab Work

CRP
Uric acid (increased)

INTERVENTION

OBJECTIVES

- Lower bodily stores of uric acid crystal deposits to prevent the inflammatory processes and structural alterations. Increase excretion of urates and force fluid intake to prevent uric acid kidney stones.
- Data from NHANES III show a remarkably high prevalence of the metabolic syndrome among individuals with gout, along with an increased risk of myocardial infarction and cardiovascular mortality (Hak and Choi, 2008). Encourage lifestyle changes including reduction in energy intake, weight, alcohol intake, red meat intake.
- Promote gradual weight loss. In the obese, controlled weight management has the potential to lower serum urate (Schlesinger, 2005).
- Correct any existing dyslipidemia and prevent complications such as renal disease, hypertension, and stroke.

FOOD AND NUTRITION

- A low-fat, high-carbohydrate (CHO) diet increases excretion of urates. Vegetables such as peas, mushrooms, cauliflower, and spinach yield a protective effect (Choi, 2005).
- Develop a weight loss plan if needed.
- Avoid excessive intake of seafood such as anchovies, sardines, caviar, and herring. Reduce intake of beef, pork, duck, bacon, turkey, and ham.
- Ensure a high-fluid intake, especially water and skim milk. Nonfat milk, low-fat yogurt, dairy products, fruits such as cherries, and high intakes of vegetable protein may reduce serum urate (Schlesinger, 2005).
- Use of 4+ cups of coffee per day should be recommended (Choi and Curhan, 2007).
- Exclude alcoholic beverages (Schlesinger, 2005) and fructose or sugar-sweetened soft drinks (Choi et al, 2008).
- Use antioxidant-rich foods such as pomegranate, raspberries, and strawberries.

SAMPLE NUTRITION DIAGNOSES

Excessive Alcohol Intake

Assessment Data: Diet history and food records, medication history, alcohol and fluid intake.

Nutrition Diagnosis (PES): Excessive alcohol intake related to consuming large amounts of alcohol (36 oz whiskey daily) as evidenced by recent painful flare of gout with hyperuricemia.

Intervention: Food-nutrient delivery—Decrease alcohol intake (ND 3.3). Education: Discuss role of proteins, alcohol, diet, fluid intake, and medications in managing gout. Counseling: Motivational interviewing and goal setting with patient (C 2.1, 2.2) to implement recommended lifestyle modifications into daily plan.

Monitoring and Evaluation: Evaluation of alcohol intake records; improvement in symptoms of gout. Monitor need for additional education/counseling. Evaluate for decrease in uric acid levels and lower frequency of gouty attacks.

Common Drugs Used and Potential Side Effects

- Uricosuric drugs: Probenecid (Benemid) and sulfinpyrazone (Anturane) block renal absorption of urates. Serum uric acid levels should be kept below 360 μmol/L (6 mg/dL). Use adequate fluid.

  Anorexia, nausea, vomiting, and sore gums may result.
- The medication febuxostat (Uloric) shows promise.
- Xanthine oxidase inhibitors: Allopurinol (Aloprim) blocks uric acid formation. Adequate intake of fluid is needed. Mild gastrointestinal (GI) upset, taste changes, or diarrhea can occur; take after meals. Febuxostat is even more effective than allopurinol; side effects are transient (Schumacher, 2005).
- During more serious outbreaks, NSAIDs, colchicine (Colcrys), and corticosteroids (prednione) may be prescribed for short-term use.
- Medications that can increase uric acid levels include hydrochlorothiazide (a diuretic) and some transplantation medications (cyclosporine and tacrolimus). Monitor for signs of gout.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Celery, avocado, turmeric, cat's claw, chiso, and devil's claw have been recommended; there are no clinical trials that prove efficacy.
- Vitamin C shows some effectiveness; 1000 mg may be beneficial in preventing gouty attacks (Gao et al, 2008).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- The inflammatory response may be suppressed by omega-3 fatty acids from fish oils and from walnuts, flaxseed, and cherries. Use these foods several times a week.
- Alcohol, beef, sardines, anchovies, and pork may precipitate a gouty attack (Choi et al, 2005). Otherwise, there is little need for a traditional "low purine" diet (Hayman and Marcason, 2009).

- Weight loss may be helpful, but avoid fasting. Instruct patient to lose weight gradually.
- Discuss the importance of adequate fluid ingestion. Recommend coffee intake (Choi and Curhan, 2007). Avoid sugar-sweetened soft drinks and fructose, but diet soft drinks are acceptable (Choi et al, 2008; Hak and Choi, 2008).
- Aim to drink at least a half gallon of water and skim milk daily.

Patient Education—Food Safety

If enteral or parenteral nutrition is needed, sanitation and handwashing are essential.

For More Information

- American College of Rheumatology
  http://www.rheumatology.org/
- Arthritis–Gout
  http://www.arthritis.org/conditions/diseasecenter/gout.asp
- Diet for Gout
  http://www.gout.com/diet_gout/gout_friendly_foods.aspx
- Mayo Clinic—Gout
  http://www.mayoclinic.com/health/gout/DS00090/rss=1

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IMMOBILIZATION

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Extended periods of immobilization, for various reasons, may be nutritionally depleting. Patients with orthopedic injuries may lose 15–20 lb from stress, immobilization, trauma, and bed rest. Prolonged immobilization and nonuse of lower and upper limb muscles may cause atrophy. Nitrogen depletion can be extensive. A large nitrogen loss and high protein oxidation can be related to extensive injury and elevated energy expenditure.

Unloading of weight-bearing bones induced by immobilization has significant impacts on calcium and bone metabolism. Immobilization hypercalcemia involves nausea, vomiting, abdominal cramps, constipation, headache, and lethargy.

Persons with physical disabilities frequently are nonambulatory and have bone loss due to immobility. Prevention of osteoporosis and related fractures in this population includes calcium and vitamin D supplementation and risk-based screening. With careful attention to functional capacity enhancements, bone mass can be restored (Rittweger et al, 2005).

In older individuals, sarcopenia is the result of excessive loss of muscle mass and strength, loss of mobility, neuromuscular impairment, and balance failure. Falls and fractures can lead to immobilization, which induces more loss of muscle mass.

One final group at risk for the consequences of immobilization are those individuals who are in intensive care units (ICU) for a prolonged period. There is a need for physical therapy, as possible, to avoid a long recovery.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Immobilization is usually from injury or other nongenetic causes, but may be a side effect of certain diseases with a genetic origin, such as spina bifida.

Clinical/History		Lab Work
Height or arm length/knee length	Dual-energy x-ray absorptiometry (DEXA)	H & H
Weight	Decreased range of motion?	Alb
BMI	Contractures; stiff joints?	Transthyretin, retinol-binding protein (RBP)
Weight changes	Blood clots	CRP
Triceps skinfold (TSF)	Pressure ulcers	Nitrogen (N) balance
Midarm muscle circumference (MAMC)	Constipation	Ca^{++} (increased?)
Midarm circumference (MAC)	Indigestion, anorexia?	Parathormone (PTH)
	Depression?	
	Change in quality of life?	

Urinary Ca^{++} (high?)	Alk phos	BUN, Creat
Vitamin D_3 status (serum 25-OHD)	Mg^{++}	Na^+, K^+
	Red blood cell (RBC) count	

INTERVENTION

OBJECTIVES

- Correct negative nitrogen balance from increased losses (perhaps up to 2–3 g of nitrogen per day) to prevent pressure ulcers and infections. Moderate exercise is beneficial in altering the inflammatory milieu associated with immobility, and in improving muscle strength and physical function (Truong et al, 2009).
- Correct anorexia, indigestion, constipation.
- Prevent deossification and osteoporosis of bones. Prevent hypercalcemia from low serum levels of albumin, which normally binds calcium.
- Prevent kidney and bladder stones, urinary tract infections.
- Provide adequate fluid intake to aid excretion of nutrients.
- Prevent constipation, impactions, and obstruction.
- Prevent anemias that result from inadequate nitrogen balance.
- Prevent venous thrombosis (McManus et al, 2009).
- Improve or sustain a positive quality of life.

FOOD AND NUTRITION

- Diet should provide adequate intake of high–biological value proteins to correct nitrogen balance. An intake of 1.2 g protein/kg body weight is often recommended. Provide adequate energy to spare protein; use sufficient carbohydrates and fats, including 1–2% total kilocalories as essential fatty acids (EFAs).
- Encourage adequate intake of calcium since a high-protein diet raises the body's calcium requirements. Increased intake of phosphorus during the first few weeks may be useful.
- Diet should provide a high-fluid intake.
- Intake of vitamin C and zinc should be adequate to protect against skin breakdown.
- Diet should provide adequate amounts of fiber to prevent constipation. Avoid overuse of fiber in cases where there is impaction.

SAMPLE NUTRITION DIAGNOSES

Physical Inactivity

Assessment Data: Diet history, food records, medication history, fluid intake. New paraplegia following motorcycle accident.

Nutrition Diagnosis (PES): Physical inactivity (NB 2.1) related to paraplegia as evidenced by inability to walk voluntarily after motorcycle accident.

Intervention: Food-nutrient delivery—Offer food preferences to maintain desired intake; monitor calcium and protein intake in particular. Education: Discuss importance of physical therapy and nutrition in maintaining as much lean body mass as possible.

Monitoring and Evaluation: Evaluate ability to tolerate sufficient physical therapy to maintain adequate skin integrity, muscle mass, and urinary tract function; ability to achieve desirable nitrogen and calcium balance.

Common Drugs Used and Potential Side Effects

- Medications may be used to treat underlying conditions; they may have side effects that contribute to nutrient losses.
- Take pain medications as directed to maintain relief of pain, rather than only taking then when you feel very badly.
- Immobilization-induced hypercalcemia affects bone metabolism in Parkinson's disease; this inhibits secretion of PTH, which in turn suppresses 1,25-dihydroxyvitamin D production (Sato et al, 2005). These abnormalities may be corrected by the suppression of bone resorption with bisphosphonate; supplementations of calcium and vitamin D should be avoided in these patients (Sato et al, 2005).

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain that calcium and nutrient intakes will have to be monitored for patients who will be tube fed or on a liquid diet for extended periods of time.
- Explain the need for adequate fiber and fluid (2–3 L) to prevent constipation, urinary tract infections, and so on. Early ambulation is the best treatment possible.
- Because prolonged bed rest in the ICU affects the development of ICU-acquired weakness, early mobility requires a reduction in heavy sedation and bed rest (Truong et al, 2009). Identify strengths and limitations, and alternate rest periods with activity. Do range of motion exercises every day.
- Monitor and report to a physician any symptoms such as pain and fatigue upon movement, new numbness in legs or arms, loss of motor strength, increased weakness, loss of bowel or bladder control, increased pain on movement.

Patient Education—Food Safety

If enteral or parenteral nutrition is needed, sanitation and handwashing are essential.

For More Information

- Family Care Research Program—Immobility and Movement
  http://www.cancercare.msu.edu/patients-caregivers/symptoms/immobility.htm
- Rehab Classworks
  http://www.rehabclassworks.com/mobility.htm

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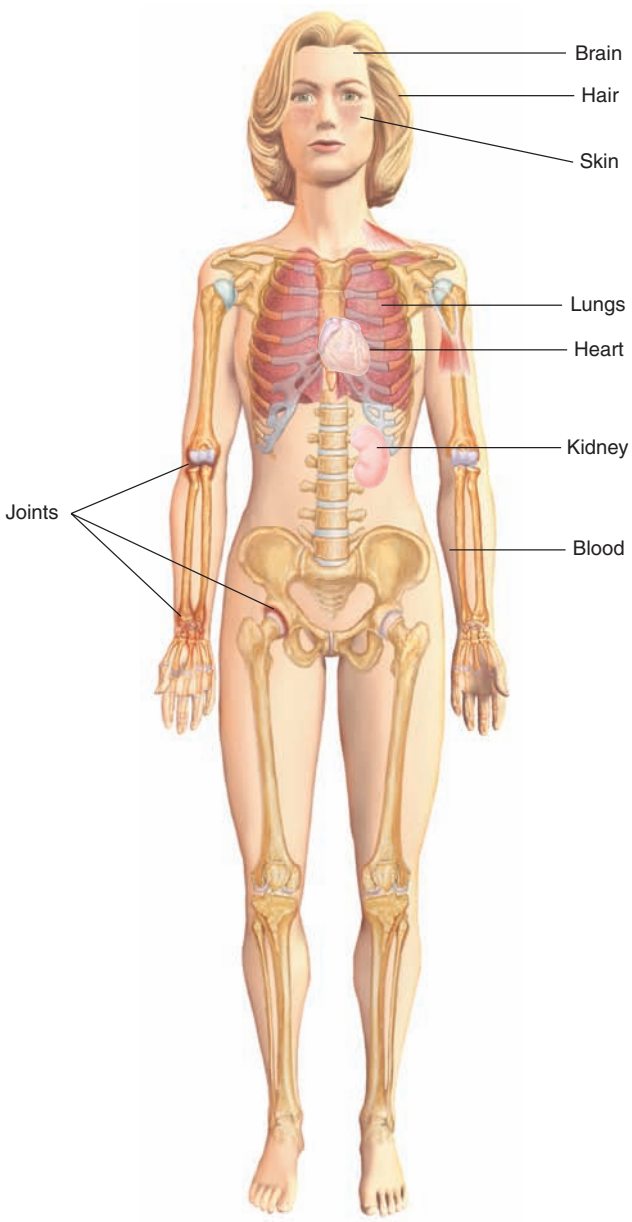
LUPUS

NUTRITIONAL ACUITY RANKING: LEVEL 2



Asset provided by Anatomical Chart Co.

DEFINITIONS AND BACKGROUND

Lupus is an autoimmune disorder that involves areas of inflammation of the joints, tendons, other connective tissues, and skin. A pathologic CD4+T cell subset with impaired extracellular signal-regulated kinase (ERK) pathway signaling, DNA hypomethylation, and consequent aberrant gene expression contributes to disease pathogenesis (Gorelik and Richardson, 2010).

There are four types of lupus: neonatal, discoid, systemic, and drug induced. The systemic form (SLE) is the most common. One to two million people have lupus, especially Latino, African American, and Native American women, with onset in the late teens to thirties. For most people, lupus is a mild disease affecting only a few organs; for some, it may cause serious and even life-threatening problems. Because lupus has symptoms that mimic other disorders, careful diagnosis is important. Lupus may show symptoms similar to those of celiac disease.

Infections can bring on a lupus flare, increasing the risk of even more infections. Other environmental factors that may trigger the disease include antibiotics (especially sulfa and penicillin), other drugs, and exposure to phthalate in toys, plastics, and beauty products.

Active lupus contributes to coronary heart disease (CHD) risk (Haque et al, 2010). Premature cardiovascular disease in SLE patients is a consequence of inflammation. Type I interferons stimulate the cascade of atherosclerotic development, starting with endothelial damage and abnormal vascular repair (Von Feldt, 2008).

SLE is characterized by autoantibodies to nuclear antigens and immune complex deposition in organs such as the kidney (Gorelik and Richardson, 2010). Lupus nephritis is the term for this form of kidney disease that occurs. About a third of patients with lupus will develop it, requiring medical evaluation and nutritional management.

A cure for lupus is not yet possible, but treatments allow a more normal life. The use of methotrexate can reduce the dependency on steroids, which is desirable (Fortin et al, 2008). Antioxidant interventions have been studied extensively and show promise. Finally, supplementation with fish oil may reduce symptomatic disease activity.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Persons with close family members who have lupus have a 10 times greater frequency than the general population. Alleles in the TYK2 gene have been associated with SLE as well as multiple sclerosis.

Clinical/History

Height
Weight
BMI
BP
I & O
Fever over 100°F
Seizures and cognitive dysfunction
Butterfly rash across cheeks and nose
Skin rashes, red raised patches
Photosensitivity
Painless mouth or nose ulcers
Pale or purple fingers from cold or stress (Raynaud's syndrome)
Unusual hair loss
Pleuritis or pericarditis
Fatigue, prolonged
Achy joints (arthralgia)
Swollen and painful joints (nonerosive arthritis)
Protein or cellular casts in urine
Swollen ankles
Dry eyes
Easy bruising

Lab Work

LE prep
ESR or CRP (elevated?)
Complement protein test (C3, C4, CH50, CH100)
INR, abnormal blood clotting
Positive anti-nuclear antibody test (ANA)
Antibodies to double-stranded DNA
Serum copper (increased)
Total protein (decreased)
WBC (decreased)
Gluc (increased)
Low platelet count
H & H, serum ferritin (decreased)
Transferrin
Chol (increased)
BUN, Creat
Specific gravity, urine (decreased)
Alb, transthyretin
Transferrin
Ca^{++}, Mg^{++}
Na^{+}, K^{+}
Vitamin D_3 status (serum 25-OHD)

INTERVENTION

OBJECTIVES

- Counteract steroid therapy; replenish potassium and nutrient reserves.
- Reduce fever and replace nutrient losses and weight loss.
- Control disease manifestations.
- Manage cardiac effects. Accelerated atherosclerosis and premature CHD are recognized complications (Haque et al, 2010). Pericarditis is also common, with shortness of breath and chest pain.
- Rule out gluten intolerance.

SAMPLE NUTRITION CARE PROCESS STEPS

Drug–Nutrient Interaction

Assessment Data: Weight and medical histories; medications; altered lab values for calcium, potassium. Complaints of swollen ankles and fluid retention.

Nutrition Diagnosis (PES): Drug–nutrient interaction related to prolonged use of corticosteroids for lupus as evidenced by osteopenia, low serum calcium and potassium, negative nitrogen balance and sodium-fluid retention.

Intervention: Food-nutrient delivery—Alter dietary intake to increase protein-rich foods, sources of potassium and calcium; decrease sodium intake. Education about the importance of managing specific nutrients while taking steroid medications (i.e., protein, calcium, potassium) and decreasing sodium-rich foods. Counseling about how to apply the DASH diet principles, which may be helpful.

Monitoring and Evaluation: Fewer complaints of swollen ankles and fluid retention; improved lab values related to calcium, potassium, and nitrogen balance studies.

- Prevent or manage infections, such as urinary tract infections, shingles, respiratory infections such as colds, yeast infections, salmonella and herpes.

FOOD AND NUTRITION

- Diet should be adequate in protein and energy during fever.
- When renal disease is present, diet should be adjusted. Check lab values regularly.
- Alter diet, if needed, to lower blood pressure (BP) levels or excess weight. Mildly restrict sodium intake and monitor for potassium and phosphorus changes.
- Dietary nutrients may modify clinical course of disease. Vitamin C intake may prevent the occurrence of active disease; use a multivitamin–mineral supplement.
- Anemia is often present. Vitamin B_{12}, dietary fiber, iron, calcium, and folate may be low in the diets of lupus patients. However, avoid excessive doses of supplements; use DRI levels.
- Use a nutrient-rich diet that includes nuts, fish and fish oils, olive oil, fruits, vegetables, and whole grains that are rich in phytochemicals, omega-3 fatty acids, and antioxidants. Include phytochemicals derived from spices (see Table 11-2).
- If gluten intolerance is present, provide a gluten-free nutrition plan.

Common Drugs Used and Potential Side Effects

- Benlysta (belimumab), is a new drug developed specifically for people with systemic lupus. Many other drugs are in clinical trials.

- Steroid therapy may cause sodium retention, hyperglycemia, potassium and calcium depletion, and negative nitrogen balance. Side effects include weight gain, a round face, acne, easy bruising, fractures or osteoporosis, hypertension, cataracts, hyperglycemia or onset of diabetes, increased risk of infection, and stomach ulcers. Fish oil supplements may allow gradual reduction in use of steroids.
- Methotrexate (Rheumatrex) confers an advantage in participants with moderately active lupus by lowering daily prednisone dose and slightly decreasing lupus disease activity (Fortin et al 2008).
- Corticosteroid and cytotoxic drugs affect the immune system over time, making the individual prone to more infections. Immunosuppressive agents such as azathioprine (Imuran) and cyclophosphamide (Cytoxan) or methotrexate are used to control the overactive immune system but they have GI side effects.
- NSAIDs and acetaminophen may be useful.
- Sunscreens are needed to protect against the sun's harmful rays; there are no systemic side effects.
- Antimalarials, such as chloroquine (Aralen) or hydroxychloroquine (Plaquenil), may be used for skin and joint symptoms of lupus. Side effects are rare and consist of occasional diarrhea or rashes. Chloroquine can affect the eyes. Hydroxychloroquine may cause anorexia, nausea, abdominal cramps, and diarrhea.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Coumestrol, a natural phytoestrogen, may relieve some symptoms.
- Use of indoles, conjugated linolenic acid (CLA), and vitamins C, E, and D may be beneficial.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Ensure patient has an adequate intake of fluids during febrile periods.
- Explain which foods are sources of sodium and potassium in the diet.
- Adequate rest is needed during flare-ups.
- Cortisone creams may be needed for persistent skin rashes. Sunblock should be used outdoors.
- Discuss how to manage diet for elevated blood glucose; insulin may be needed. Carbohydrate counting may be useful.
- Regular doctor visits and lab tests are important, especially blood and urine testing.
- Dietary strategies for the prevention of obesity, osteoporosis, and dyslipidemia deserve attention. Weight loss plans may be needed.

Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

For More Information

- Lupus Alliance of America
  http://www.lupusalliance.org/
- Lupus Canada
  http://www.lupuscanada.org/
- Lupus Foundation of America
  http://www.lupus.org/
- Lupus Library
  http://www.lupusny.org/library.php
- Lupus Organizations
  http://www.lupusny.org/links.php#lupusorg
- SLE Foundation, Inc.
  http://www.lupusny.org/

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MUSCULAR DYSTROPHY

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Actually a group of nine disorders, muscular dystrophy (MD) involves a hereditary condition with progressive degenerative changes in the muscle fibers, leading to weakness and atrophy. Most of the disorders are described in Table 11-5.

Muscular biopsy is required for the definitive diagnosis of the specific congenital type. BMI should be used with caution for the evaluation of the nutritional status of patients with Duchenne MD (DMD); assessment of the compartmental distribution of muscle and fat are more sensitive. Extremely elevated serum creatine kinase (CK) levels may indicate muscle disease. In the late stages, fat and connective tissue may replace muscle fibers.

Patients with MD may be prone to nutrient deficiency due to mobility limitations or oropharyngeal weakness (Motlagh et al, 2005). Micronutrient requirements are yet to be determined but, as a result of corticosteroid treatment, vitamin D and calcium should be supplemented (Davidson and Truby, 2009).

Many patients demonstrate inadequate nutrient intake of protein, energy, vitamins (especially E), and minerals (calcium, selenium, and magnesium), and significant correlations exist between measures of strength and copper and water-soluble vitamins (Motlagh et al, 2005).

Delayed growth, short stature, muscle wasting, and increased fat mass are characteristics that impact on nutritional status and energy requirements (Davidson and Truby, 2009). There may be loss of muscle mass, wasting, which may be hard to see because some types of MD cause a build-up of fat and connective tissue that makes the muscle appear larger (pseudohypertrophy).

Gene therapy, gene silencing, and cell therapy are potential therapies for MD patients. Some evidence exists supporting supplementation with creatine monohydrate to improve muscle strength (Davidson and Truby, 2009). Creatinine as a marker of renal function has limited value in DMD because of reduced muscle mass. There is potential value of cystatin C as a biomarker for monitoring renal function (Violett et al, 2009).

The prognosis of MD varies according to type and progression. Some cases may be mild and very slowly progressive, with a normal lifespan. Other cases may have more marked progression of muscle weakness, functional disability, and loss of ambulation. Life expectancy often depends on the degree of progression and late respiratory deficit. In DMD, death often occurs in the late teens or early twenties. Rehabilitation, orthopedic, respiratory, cardiovascular, gastroenterology, nutrition, pain issues, as well as general surgical and emergency-room considerations are essential to address (Bushby et al, 2010).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Dystrophinopathies are due to a genetic defect of the protein dystrophin. Genetic counseling is advised when there is a family history of MD. Note that DMD can be detected by genetic studies performed during pregnancy.

Clinical/History

- Height
- Weight
- BMI (use only with other parameters)
- MAC, MAMC, TSF
- Muscle weakness
- Apparent lack of coordination
- Progressive crippling
- Scoliosis?
- Contractures of the muscles around the joints
- Clubfoot or clawhand?
- Hypotonia
- Loss of mobility
- Eyelid drooping (ptosis)
- Dysphagia?
- Drooling
- Chewing difficulty?
- Hand-to-mouth coordination
- Electromyography (EMG)
- Electrocardiography (ECG)
- DEXA

Lab Work

- Muscle biopsy
- Creatine phosphokinase (CPK), increased?
- Lactate dehydrogenase (LDH), increased
- Cystatin C
- Myoglobin-urine/serum
- Creat (often decreased)
- Aldolase, AST (altered)
- BUN
- N balance
- Alb, transthyretin
- CRP
- H & H, serum ferritin
- Serum P
- Gluc
- AST, ALT
- Ca^{++}, Mg^{++}
- Na^{+}, K^{+}
- Vitamin D_3 status (serum 25-OHD)

TABLE 11-5 Types of Muscular Dystrophy and Nutritional Implications

Type of MD	Comments	Nutritional Implications
Becker muscular dystrophy (BMD)	Very similar to DMD (see below), but onset is later (adolescence or adulthood). BMD patients live longer.	Weakness makes it difficult for self-feeding.
Congenital muscular dystrophy (CMD)	Caused by genetic mutations affecting some of the proteins necessary for muscles and some proteins related to the eyes and/or brain. Onset is at or near time of birth. Indicators include generalized muscle weakness with possible joint stiffness or looseness. Depending on the type, CMD may involve spinal curvature, respiratory insufficiency, mental retardation or learning disabilities, eye defects, and seizures.	Weakness makes it difficult for self-feeding.
Distal muscular dystrophy (DD)	DD is caused by a mutation in any of at least seven genes that affect proteins necessary to the function of muscles; it is usually passed on as an autosomal dominant or autosomal recessive trait. DD (Miyoshi form) first shows signs between ages 40 and 60 years, with weakness and muscle wasting of the hands, forearms, and lower legs; it progresses slowly.	Weakness and muscle wasting of the hands and forearms make self-feeding difficult.
Duchenne muscular dystrophy (DMD)	DMD primarily affects young boys, who inherit the disease through their mothers (X-linked recessive). Also called pseudohypertrophic. Caused by absence of dystrophin, a protein that helps keep muscle cells intact. DMD is the most severe form of dystrophinopathy. Aggressive forms appear in age 2–3, with frequent falls, difficulty in getting up from sitting or lying position. Generalized weakness and muscle wasting affect hip, thigh, shoulder, and trunk muscles first; large calf muscles. Weakness in lower leg muscles, resulting in difficulty running and jumping; waddling gait; mild mental retardation or scoliosis, in some cases. Survival is rare after the late twenties.	Facial muscles are involved; patient cannot suck, close lips, bite, chew, or swallow. DMD eventually affects all voluntary muscles, the heart, and lung muscles.
Emery-Dreyfus muscular dystrophy (EDMD)	EDMD is caused by gene mutations that produce emerin, lamin A, or lamin C, which are proteins in the membrane that surrounds the nucleus of each muscle cell. Onset in childhood, usually by age 10. Weakness and wasting of shoulder, upper arm, and shin muscles and joint deformities are common. Disease usually progresses slowly. Frequent cardiac complications are common, and a pacemaker may be needed.	Self-feeding becomes difficult.
Fascioscapulohumeral muscular dystrophy (FSHMD)	FSHMD (also called Landouzy-Dejerine) begins in childhood to early adulthood, with facial muscle weakness and weakness and wasting of the shoulders and upper arms. Caused by a missing piece of DNA on chromosome 4, it progresses slowly with some periods of rapid deterioration. Usually evident by age 20, it may span many decades. Inheritance is autosomal dominant, which means it can be passed on by either parent. It is considered to be the most common form.	Self-feeding becomes difficult; loss of skeletal muscle occurs. Abdominal muscles are affected.
Limb-girdle muscular dystrophy (LGMD)	LGMD is caused by a mutation in any of at least 15 different genes that affect proteins necessary for muscle function. LGMD has an onset in late childhood to middle age. Weakness and wasting affects shoulder and pelvic girdles first. Progression is slow, with cardiopulmonary complications often occurring in later stages of the disease. It is inherited as an autosomal recessive, autosomal dominant trait.	Self-feeding becomes difficult.
Myotonic dystrophy (MyD)	MyD (Steinert's disease) has onset anywhere from birth to middle age. Congenital myotonic form is more severe. Generalized weakness and muscle wasting affect the face, feet, hands, and neck first. Delayed relaxation of muscles after contraction. Progression is slow, sometimes spanning 50–60 years. Inheritance is autosomal dominant; there is a repeated section of DNA on either chromosome 19 or chromosome 3. Individuals with MyD have long faces and drooping eyelids; men have frontal baldness.	Progression is slow. Often complicated by diabetes. Prone to nutritional deficiencies from associated dysmotility of the entire GI. Handgrip is significantly lower; knee extension is higher compared to other dystrophies (Motlagh et al, 2005).
Oculopharyngeal muscular dystrophy (OPMD)	OPMD has onset in early adulthood to middle age. It affects muscles of eyelids (causing droopy eyelids) and throat. It progresses slowly, with swallowing problems common. Inheritance is autosomal dominant, and onset is usually in the fourth or fifth decade. The gene that is defective in OPMD is called the *poly(A) binding protein2* gene; extra amino acids in the protein made from the defective *PABP2* gene cause the protein to clump together in the muscle cell nuclei, interfering with cell function. OPMD can be diagnosed with a DNA test.	Swallowing difficulty is common. Tube feeding should be considered before wasting occurs.

From the Muscular Dystrophy Association, accessed November 16, 2009, at http://www.mdausa.org/publications/Quest/q65occup.html.

Type of CMD	Cause	Inheritance Pattern
Merosin-deficient CMD	Lack of merosin (laminin 2) or other defect leading to merosin deficiency	Chromosome 6 gene Other genes
Ullrich CMD	Abnormalities in collagen 6	Chromosome 2 or 21 genes, recessive or dominant
Bethlem myopathy	Abnormalities in collagen 6	Chromosome 2 or 21 genes, dominant
Integrin-deficient CMD	Lack of integrin alpha 7	Chromosome 12 gene, recessive
Fukuyama CMD (FCMD)	Lack of fukutin	Chromosome 9 gene, recessive
Muscle-eye-brain disease (MEB)	Lack of POMGnT1, fukutin or fukutin-related protein	Chromosome 1, 9, or 19 genes, recessive
Walker–Warburg syndrome (WWS)	Lack of POMT1, POMT2, fukutin or fukutin related protein	Chromosome 9, 14, or 19 genes, recessive
CMD with rigid spine syndrome	Lack of selenoprotein N1	Chromosome 1 gene, recessive

INTERVENTION

OBJECTIVES

- Encourage patient to lead a relatively active life; exercise programs can help prevent contractures.
- Prevent obesity, from inactivity; obesity complicates physical therapy.
- Encourage activities other than eating to prevent dependency on food as a source of pleasure.
- Malnutrition is a serious threat, especially with respiratory muscle weakness. Monitor nutritional intake and deficits on a regular basis. Prevent aspiration pneumonia or nasal regurgitation. Use a multidisciplinary approach, especially for feeding difficulties such as texture modification and supplemental feeding (Davidson and Truby, 2009).
- Avoid constipation because fecal impaction is frequent.
- Prevent osteoporosis and fractures, which can occur in this population.
- Manage long-term consequences, such as cardiomyopathy or respiratory failure.

SAMPLE NUTRITION CARE PROCESS STEPS

Abnormal GI Function—Constipation

Assessment Data: Weight and physical activity histories. Medical history, medications and lab values. Complaints of chronic constipation and GI discomfort. Diet hx showing low fiber and intake of <4 cups of fluid per day to avoid the need to urinate.

Nutrition Diagnosis (PES): Abnormal GI function related to constipation and physical inactivity as evidenced by infrequent evacuation, hard stools, and GI discomfort.

Intervention: Food and Nutrient Delivery—Increase fluid and fiber sources from tolerated foods such as whole grain cereals, fresh fruits, and vegetables. Educate about the desirable foods and fluid intake. Counsel about the importance of daily range of motion and physical exercises. Coordinate care with other disciplines according to needs, including PT, OT, nursing, medical team.

Monitoring and Evaluation: Alleviation of constipation. No further complaints of GI discomfort related to infrequent stooling pattern.

FOOD AND NUTRITION

- Work with the MyPyramid food guidance system as a basic guide. Check patient's BMI and adjust intake accordingly. Use a low-energy diet if necessary to control or lessen obesity. Some patients' requirements may be 30% lower than normal (Munn et al, 2005).
- Use foods that are easy to chew and swallow for DMD, such as pureed or blenderized foods. Tube feed only if necessary.
- Provide adequate fiber (prune juice, bran and other whole grains, fruits, and vegetables) if constipation becomes a problem.
- Ensure adequate intake of fluid to prevent fecal impaction, dehydration, and related effects.
- Adequate sodium chloride is important (Yoshida et al, 2006). Manage carefully if there are cardiac side effects or problems with BP.

Common Drugs Used and Potential Side Effects

- The myotonia (delayed relaxation of a muscle after a strong contraction) may be treated with medications

such as phenytoin or quinine. Side effects can include folic acid depletion.
- Early introduction of steroids can exacerbate weight gain in a population already susceptible to obesity (Davidson and Truby, 2009).
- It may be useful to try beta$_2$-adrenergic agonists, which can increase muscle mass. Albuterol may be needed for some individuals prior to exercise and strength training.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.

Approximately 50% of children are on herbal preparations, 30% of adolescents take herbal medications, and 70% of adults use some aspect of complementary medicine (Buehler, 2007).

- Green tea extract may improve muscle health by reducing or delaying necrosis by an antioxidant mechanism (Dorchies et al, 2006).
- Traditional Chinese medicine has been advocated for treatment of types of MD, but studies are needed to identify active ingredients.
- Supplementation with creatine monohydrate may improve muscle strength (Davidson and Truby, 2009).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Provide low-calorie snack tips for patients who are obese.
- Help patient modify food textures to meet needs.
- Discuss problems related to inactivity or weight gain.
- Discuss the importance of adequate fluid intake.
- Discuss methods to prevent aspiration and pneumonia.
- Comprehensive management strategies can improve function, quality of life, and longevity (Bushby et al, 2010). Work with the occupational therapist and other therapists to maintain optimal levels of function.

Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

For More Information

- Facioscapulohumeral Dystrophy (FSHD) Society
  http://www.fshsociety.org
- Muscular Dystrophy Association (MDA)
  http://www.mdausa.org/
- Muscular Dystrophy Association of Canada
  http://www.mdac.ca/
- Muscular Dystrophy Family Foundation
  http://www.mdff.org/
- National Institute—MD
  http://www.ninds.nih.gov/health_and_medical/disorders/md.htm
- Neuromuscular Disorders in the MDA Foundation
  http://www.mdausa.org/disease/
- Parent Project for Muscular Dystrophy Research
  http://www.parentprojectmd.org
- Rare Muscular Dystrophy types
  http://www.mdausa.org/publications/fa-rareMD.html#dd

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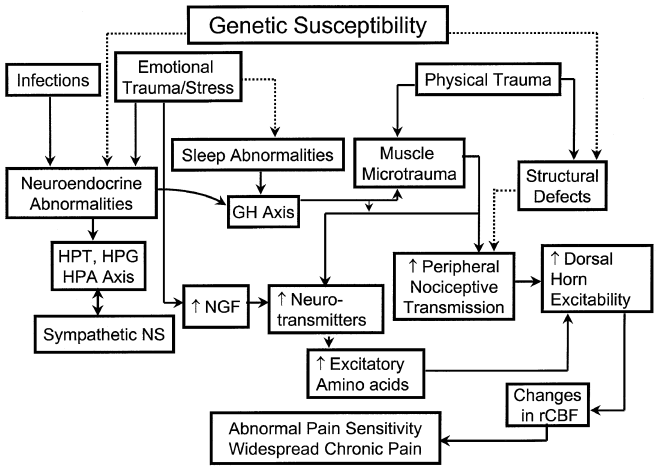
MYOFASCIAL PAIN SYNDROMES: FIBROMYALGIA AND POLYMYALGIA RHEUMATICA

NUTRITIONAL ACUITY RANKING: LEVEL 1–2



Adapted from: William J. Koopman, Larry W. Moreland, *Arthritis and Allied Conditions A Textbook of Rheumatology,* 15th ed. Philadelphia: Lippincott Williams & Wilkins, 2005.

DEFINITIONS AND BACKGROUND

Myofascial pain syndromes are a group of disorders characterized by achy pain and stiffness in soft tissues, including muscles, tendons, and ligaments. In the United States, **fibromyalgia (FM)** is estimated to occur in 2% of adults (Mease, 2005). Diagnosis is difficult, and the etiology is not clear. Corticotropin-releasing hormone (CRH) and substance P (SP) are found in increased levels in the cerebral spinal fluid (CSF) of FM patients, and increased interleukin (IL)-6 and IL-8 are found in the serum where they release proinflammatory and neurosensitizing molecules (Lucas et al, 2006).

FM, or "fibrositis," is a central sensitivity syndrome with abnormalities in the peripheral, central, sympathetic nervous systems, and the hypothalomo–pitutary–adrenal axis stress response system (Mease, 2005). Etiology theories abound, including inadequate sleep, physical or psychological trauma, or exposure to viruses such as hepatitis B or C, or HIV infection. Serotonin and dopamine levels may be lower than normal. Insulin-like growth factor-1 (IGF-1) levels may also be low; they are a surrogate marker for low growth hormone secretion during stage 3 and 4 of sleep, when tissue repair occurs (Rosenzweig and Thomas, 2009).

FM causes widespread pain and stiffness either throughout the body or localized along the spine. Persistent symptoms may be disruptive but are not life threatening. Symptoms include sleep disturbance, depression, fatigue, headaches, irritable bowel syndrome, numbness in the hands and feet, and mood disorders. Acupuncture may offer relief (Martin et al, 2006).

Polymyalgia rheumatica (PMR) affects people over age 70 years, usually women. It causes aching, severe muscle stiffness, and pain. Symptoms start suddenly and may affect several areas in the neck, shoulders, hips, and/or thighs. It usually goes away with treatment but may reoccur. Symptoms include mild joint stiffness and swelling, face pain, anemia, extreme fatigue, unintentional weight loss, and anorexia. The cause of PMR is not known but may be related to aging. Diagnosis is difficult. Many people with PMR also have giant-cell arteritis with double vision, severe headaches, or vision loss. Low-dose corticosteroids may be needed for up to a year (Hernandez-Rodriguez et al, 2009).

Treatment of myofascial pain disorders may include exercise, medications such as glucocorticoids and NSAIDs, a healthy diet rich in antioxidants, and adequate rest. Massage and cognitive behavioral therapy (CBT) are helpful (Mease, 2005). After a warm-water swimming program, a significant decrease in IL-8, IFNgamma, and CRP has been noted (Ortega et al, 2009). Use of a phyochemical-rich diet results in a decrease in joint stiffness and pain as well as an improvement in self-reported quality of life. Plant foods are rich natural sources of antioxidants (quercetin, myristin, and kaempherol) in addition to fiber and other nutrients. A vegan diet often shows highly increased serum levels of beta- and alpha-carotenes, lycopene, lutein, and vitamins C and E.

Rapid-paced discovery is taking place in genetics, patient assessment, new therapeutic targets, and novel methods of treatment delivery (Williams and Clauw, 2009). The best multidisciplinary team includes a rheumatologist, physical therapist, exercise therapist, dietitian, and massage therapist (Lemstra and Olszynski, 2005).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: A multigenic or genome-wide approach may be needed to alter individualized pain therapy according to the patient's genotype. For example, DRD2 polymorphisms decrease the functioning of the dopaminergic reward system; this could cause an individual to require more pain medicine. Research is ongoing to determine whether individuals with FM have the genetic tendency toward lower pain thresholds.

Clinical/History

- Height
- Weight
- BMI
- Tender areas, back pain
- Headache
- Depression, mood disorders
- Morning stiffness
- Fatigue, sleep disturbances
- Fibromyalgia Impact Questionnaire (FIQ)
- FM-pain in shoulders, pelvis, and hips (pain in 11/18 trigger points)
- Carpal tunnel syndrome (in PMR)

Lab Work

CRP or ESR (may be high)
Plasma adrenomedullin (high in PMR)
Trig, Chol
Alb, transthyretin
BUN, Creat
Ca^{++}, Mg^{++}
Na^{+}, K^{+}
Gluc
Alk phos
Vitamin D_3 status (serum 25-OHD)

INTERVENTION

OBJECTIVES

- Relieve pain. Acupuncture, massage, cognitive-behavior therapy (CBT) and varied exercises may be recommended (Assefi et al, 2005; Rosenzweig and Thomas, 2009).
- Lose weight, if obese.
- Correct underlying problems such as hypertension.
- Support lifestyle changes, including stress reduction, relaxation techniques, and exercise.
- Prevent blindness in PMR when there is giant-cell arteritis.

FOOD AND NUTRITION

- Use a balanced diet. The MyPyramid food guidance system is another useful tool for planning a healthy diet. Include phytochemicals; dietary quercetin should be encouraged (Lucas et al, 2006). Table 11-2 is also a useful reference.
- A vegan diet may be beneficial with berries, fruits, vegetables, roots, nuts, germinated seeds, and sprouts.
- A weight loss plan may be needed.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Intake of Bioactive Substances

Assessment Data: Weight and physical activity histories. Medical history, medications and lab values. Much pain; diagnosed FM. Diet hx and 3-day food record shows intake of <2 fruits and vegetables daily. No food allergies.

Nutrition Diagnosis (PES): Inadequate intake of bioactive substances related to low intake of fruits and vegetables as evidenced by diet history and intake records.

Intervention: Food and Nutrient Delivery—Provision of more spices, fruits, vegetables and juices. Education about the role of antioxidants, spices, and phytochemicals in reducing inflammation and the possibility of lessening pain symptoms. Counseling about menus, recipes and cooking tips for including more bioactive ingredients. Encourage intake of fish oils, walnuts, fatty fish such as salmon for omega-3 fatty acids.

Monitoring and Evaluation: Diet Hx showing improved intake of spices (such as turmeric, cumin, cinnamon), cocoa and coffee, fruits including berries and apples and pomegranates, vegetables such as broccoli and cabbage on a daily and weekly basis. Fewer complaints of overt pain.

- Increased intake of omega-3 fatty acids may help to reduce inflammation and relieve pain in some individuals. Increase intake of fatty fish, walnuts, and flaxseed.

Common Drugs Used and Potential Side Effects

- Medications that decrease pain and improve sleep may be prescribed. Low doses of tricyclic antidepressants (amitriptyline, Elavil; cyclobenzaprine, Flexeril) and the serotonin-3 receptor antagonist tropisetron may be helpful (Lucas et al, 2006; Rosenzweig and Thomas, 2009). Opioids, NSAIDs, sedatives, muscle relaxants, and antiepileptics have been used to treat FMS (Mease, 2005).
- Pregabalin (Lyrica) and duloxetine (Cymbalta) are used in FMS. Milnacipran (Savella) is a dual norepinephrine and serotonin reuptake inhibitor that has been shown to be safe (Arnold et al, 2009; Mease et al, 2009).
- Opioids are not recommended for FM (Rosenzweig and Thomas, 2009).
- For PMR, a trial of low-dose corticosteroids is given, usually in the form of 10–15 mg of prednisone (Deltasone, Orasone) per day. Side effects may include sleeplessness, weight gain, loss of nitrogen and calcium, cataracts, thinning of the skin, easy bruising. NSAIDS, such as ibuprofen (Advil, Motrin) and naproxen (Naprosyn, Aleve), are ineffective in the treatment of PMR.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician. CAM is popular for musculoskeletal conditions. Some CAM modalities show significant promise, such as acupuncture (Martin et al, 2006). Excellent resources are available on the Internet from the National Center for Complementary and Alternative Medicine (http://nccam.nih.gov).
- Magnesium; sulfur compounds such as SAMe, dimethylsulfoxide (DMSO), taurine, glucosamine, and chondroitin sulfate; and reduced GSH may have clinical applications in the treatment of FMS; controlled trials are needed.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Aerobic exercise, patient education and CBT are quite effective. Daily exercise will be important for strengthening weak muscles. Exercise adherence can help reduce the need for pain medications (Lemstra and Olszynski, 2005).
- Discuss weight management, as needed.
- Discuss the role of omega-3 fatty acids in reduction of inflammation.

Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

For More Information

- American Fibromyalgia Syndrome Association, Inc. http://www.afsafund.org/
- Fibromyalgia Network http://www.fmnetnews.com/
- Mayo Clinic http://www.mayoclinic.com/health/myofascial-pain-syndrome/DS01042
- Myositis Association http://www.myositis.org/
- National Fibromyalgia Partnership, Inc. http://www.fmpartnership.org/FMPartnership.htm
- National Institute of Arthritis and Musculoskeletal and Skin Diseases http://www.niams.nih.gov/hi/topics/fibromyalgia/fibrofs.htm
- Polymyalgia Rheumatica http://www.rheumatology.org/public/factsheets/diseases_and_conditions/polymyalgiarheumatica.asp
- Polymyalgia Rheumatica and Giant Cell Arteritis http://www.niams.nih.gov/Health_Info/Polymyalgia/default.asp

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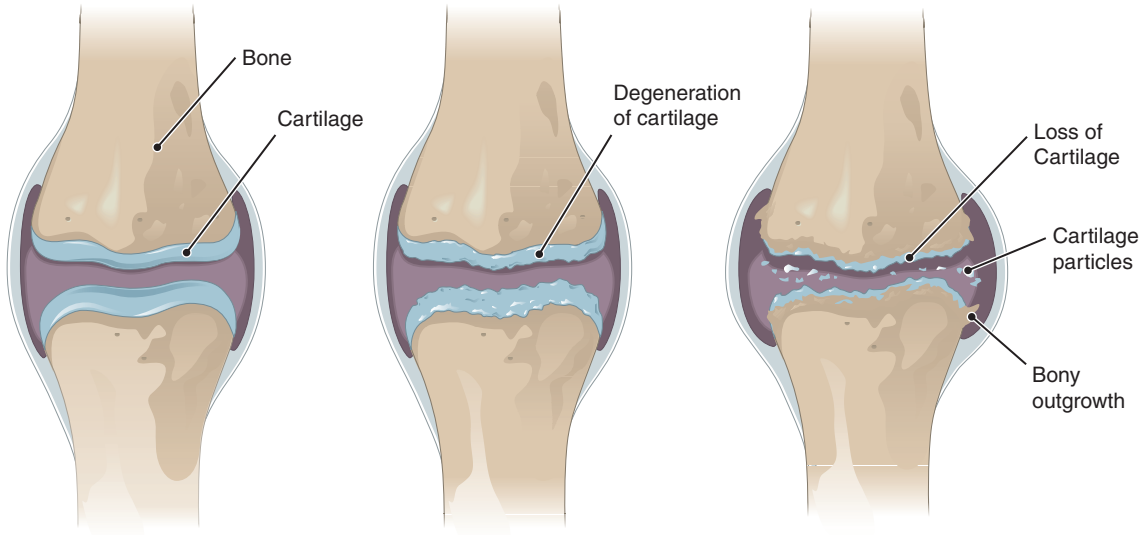
OSTEOARTHRITIS AND DEGENERATIVE JOINT DISEASE

NUTRITIONAL ACUITY RANKING: LEVEL 1–2



Adapted from: Cohen BJ. *Medical Terminology,* 4th ed. Philadelphia. Lippincott Williams & Wilkins 2003.

DEFINITIONS AND BACKGROUND

OA may be primary (in older individuals) or may follow an injury or disease involving the articular surfaces of synovial joints. The joint may lose its normal shape and bone spurs may grow on the edges of the joint. OA is a common health problem in populations over age 40 years, and it is a leading cause of pain and disability. OA mostly affects cartilage where the surface layer breaks down and wears away. The hands, knees, hips, and spine are most commonly affected.

Over 40 million Americans report that they have arthritis and many indicate that it limits their daily activities. High serum concentrations of tumor necrosis factor are associated with lower physical function and more pain, stiffness, and physical disability. Over a third of adults with arthritis experience limitations in their ability to work.

Treatments for OA combine nonpharmacological modalities, pharmacological agents, and surgical procedures. Exercise, weight control, rest, and relief from stress on joints, nondrug pain relief, and various types of complementary

medical techniques may be useful. Continuous passive motion (CPM), massage, and heat treatments may be used. Surgery is reserved for those persons for whom other treatments have been unsuccessful.

Weight loss is a primary treatment for OA for individuals who are obese. Obesity is a significant risk factor for and contributor to increased morbidity and mortality from chronic diseases, including OA (Pi-Sunyer, 2009). Overweight causes strain on joints and should be managed early by health professionals (Gasbarrini and Piscaglia, 2005). An average weight loss of 5% in overweight and obese older patients brings an 18% gain in overall function (Messier et al, 2005), and a 10% weight loss improves function by 28% (Christensen et al, 2005).

While vitamins A, C, and E have major roles in modulating oxidative stress, immune responses, and cell differentiation, controlled trials found that these vitamins do not halt progression of OA (Choi, 2005). Vitamins D and K play a protective role (Bergink et al, 2009; Oka et al, 2009). Diets rich in omega-3 fatty acids may reduce joint stiffness and pain, increase grip strength, and enhance walking pace. Pomegranate fruit extracts can block interleukin-1β (IL-1β) enzymes that contribute to cartilage destruction and OA. Finally, cadgerin-11 is a protein that contributes to joint destruction and a related fabric has been developed for cartilage replacement.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: OA is the breakdown and inflammation of joint cartilage, usually brought on by aging and repetitive joint usage. OA and cardiovascular disease share age and obesity as risk factors, but may also be linked by pathogenic mechanisms involving metabolic abnormalities and systemic inflammation (Puenpatom and Victor, 2009).

Clinical/History

- Height
- Weight
- BMI
- Obesity?
- Pain, swelling of joints (arthralgia)
- Synovial joint stiffness
- Crunching sound of bone against bone

- X-rays; DEXA
- OA Index

Lab Work

- Antistreptolysin titer (ASO)
- Antirheumatoid factor
- BUN, Creat
- Sedimentation rate
- CRP
- Gluc
- Alk phos
- Uric acid
- Ca^{++}, Mg^{++}
- Na^{+}, K^{+}
- Serum folate and B_{12}
- Vitamin D_3 status (serum 25-OHD)

SAMPLE NUTRITION CARE PROCESS STEPS

Overweight

Assessment Data: Weight and physical activity histories; symptoms diary; medical history, medications, and lab values.

Nutrition Diagnosis (PES): Overweight with joint pain related to eating double portions at lunch and dinner meals as evidenced by diet history and BMI of 29.

Intervention: Food and Nutrient Delivery—Offer choices of lower fat and low energy foods and snacks. Educate about the role of excess weight and joint pain. Counsel and offer weight management tips, tips for dining out or snacking. Coordinate care with referral to physical therapy for exercises to reduce pain and stiffness in affected joints.

Monitoring and Evaluation: Weight changes, food intake records, symptoms diary.

INTERVENTION

OBJECTIVES

- Control pain and improve joint function. If joint replacement is necessary, prepare for surgery accordingly.
- Maintain a normal body weight. If needed, weight loss may be beneficial to lessen pressure on weight-bearing joints.
- Maintain an active lifestyle as much as possible.
- Encourage patient (especially if older) to consume adequate amounts of vitamins D and K, protein and calcium from a healthy, nutrient-dense, antioxidant-rich diet.
- Maintain integrity of cartilage in affected joints. Omega-3 fatty acids may reduce the activity of enzymes that destroy cartilage. Include fish oils and certain plant seed oils that impact immune and inflammatory responses as precursors of eicosanoids.

FOOD AND NUTRITION

- Use a calorie-controlled diet if obesity is present. Use of a meal replacement may help to promote weight loss.
- The inflammatory response may be suppressed by an increase in omega-3 fatty acids, as found in fatty fish (mackerel, herring, and salmon) and from walnuts and flaxseed. Use these foods several times a week.
- Calcium is found in dark green, leafy vegetables, such as kale and broccoli; canned sardines and salmon with bones; fortified orange juice; milk and dairy products, such as cheese and yogurt; fortified bread, tofu or soy milk.
- Vitamin C is needed for healthy collagen and cartilage. Good sources include citrus fruits, bell peppers, tomatoes, watermelon, strawberries, and kiwifruit.
- Low dietary vitamin D intake increases the risk of progression of knee OA, particularly in subjects with low baseline BMD (Bergink et al, 2009). Include vitamin D from sardines, herring, fish-liver oil; butter and cream; egg yolks; liver; fortified cow's milk and dairy

products, such as cheese and yogurt; and fortified cereals.
- Vitamin K may be found in leafy greens such as kale, Swiss chard, broccoli, spinach, raw parsley. It is also found in small amounts in olive, soybean or canola oils and in mayonnaise.
- Boron may help OA. Sources include apples, legumes, leafy vegetables, carrots, pears, grapes, and some drinking water.
- Include plenty of phytochemicals. Pomegranates and cranberries are especially protective because of the ellagic acid. See Table 11-2.

Common Drugs Used and Potential Side Effects

- Because of GI risks (including ulcer complications) and cardiovascular risks, including hypertension and thrombotic events associated with NSAIDs, acetaminophen is the first choice anti-inflammatory agent (Berenbaum, 2008). Table 11-6 gives more details on medicines used for OA. Evaluate risks as well as benefits for all drug therapies.

TABLE 11-6 Medications Commonly Used for Osteoarthritis

Medication	Comments	Side Effects
Anti-inflammatory drugs, nonsteroidal (NSAIDs)	Indomethacin (Indocin), aspirin piroxicam (Feldene), naproxen (Naprosyn), nabumetone (Relafen) and Ibuprofen (Advil, Motrin) may be recommended.	Nausea, GI distress, anorexia, flatulence, or vomiting can occur. Take with food. Prolonged use may cause GI bleeding or ulcers. Indomethacin may also cause renal failure, or diarrhea; naprosen may cause heartburn or increased risk of cardiovascular disease.
COX-2 Inhibitors	Cyclooxygenases are needed for the synthesis of prostaglandins. The COX-2 enzyme mediates inflammation and pain. Celecoxib (Celebrex) is the only FDA-approved drug at this time.	These agents may promote increased risk of heart attack and stroke. Rofecoxib (Vioxx) and valdecoxib (Bectra) were removed from the market in 2004 and 2005.
Misoprostol (Cytotec)	Misoprostol reduces stomach acid if NSAIDs are used.	Abdominal cramps may occur.
Frankincense (Boswellia frereana)	Interestingly, this herb may lessen arthritic pain. Epi-lupeol is the principal constituent of B. frereana. B. frereana prevents collagen degradation, and inhibits the production of pro-inflammatory mediators (Blain et al, 2010).	Fewer side effects than glucosamine and chondroitin.
Glucosamine sulfate and chondroitin	Glucosamine reduces cartilage damage and decreases pain associated with osteoarthritis. Taken with chondroitin, it may help relieve symptoms of osteoarthritis. Some pills do not contain sufficient levels to be effective; check brand with www.consumerlab.com to select the best choice.	Glucosamine can increase blood glucose levels andaggravate shellfish allergy because it is made from these shells. Chondroitin may alter blood clotting activity in a manner similar to that of aspirin.
Hyaluronic acid substitutes (viscosupplements)	These injections are designed to replace a normal component of the knee joint involved in joint lubrication and nutrition.	A series of injections are required. When used with methotrexate, the benefits may be greater (Homma et al, 2009).
Omega-3 fatty acids	Supplementation causes a decrease in both degradative and inflammatory aspects of chondrocyte metabolism.	May increase effects of blood-thinning drugs and herbs.
Steroids	Corticosteroids may cause sodium retention; calcium, nitrogen, and potassium depletion; truncal obesity; and hyperglycemia.	Corticosteroids may have a short-term effect in osteoarthritis (Bellamy et al, 2005). Injections are required.
Topical pain relievers (Zostrix, Icy Hot, Therapeutic Mineral Ice, Aspercreme, and Ben Gay)	Creams and rubs stimulate nerve endings to relieve pain; deplete the amount of neurotransmitter (substance P) that sends pain messages to the brain; and block prostaglandins that cause pain and inflammation.	No internal side effects.
Tramadol (Ultram)	Pain reliever that is prescribed when over-the-counter medications do not provide relief.	Potentially addictive.

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National Institite of Arthritis and Musculoskeletal and Skin Disorders. Web site accessed December 13, 2009, at http://www.niams.nih.gov/Health_Info/Osteoarthritis/default.asp#4

TABLE 11-7 Side Effects of Herbs Commonly Used for Arthritis

Bromelain	May increase effects of blood-thinning drugs and tetracycline antibiotics.
Echinacea	Might counteract immunosuppressant drugs, such as glucocorticoids, taken for lupus and rheumatoid arthritis; might increase side effects of methotrexate.
Evening primrose oil	Can counteract the effects of anticonvulsant drugs.
Folic acid	Interferes with methotrexate.
Gamma linoleic acid (GLA)	May increase effects of blood-thinning drugs and herbs.
Garlic	Can increase effects of blood-thinning drugs and herbs
Ginger	Can increase nonsteroidal anti-inflammatory drug (NSAID) side effects and effects of blood-thinning drugs and herbs.
Ginkgo	May increase effects of blood-thinning drugs and herbs.
Ginseng	May increase effects of blood-thinning drugs, estrogens, and glucocorticoids; should not be used by those with diabetes; may interact with monoamine oxidase (MAO) inhibitors.
Kava	Can increase effects of alcohol, sedatives, and tranquilizers.
Magnesium	May interact with blood pressure medications.
***S*-adenosylmethionine (SAMe)**	SAMe may rebuild eroded joint cartilage. Enteric coating is needed because of gastrointestinal (GI) side effects.
Soy and avocado extracts	Antioxidant effects in reducing the symptoms of osteoarthritis; avoid excessive use in patients with hormonal cancers.
St. John's wort	May enhance effects of narcotics, alcohol, and antidepressants; increases risk of sunburn; interferes with iron absorption.
Valerian	May enhance effects of sedatives and tranquilizers.
Vitamin E	Gamma-tocopherol may worsen osteoarthritis; alpha-tocopherol is better.
Zinc	Can interfere with glucocorticoids and other immunosuppressive drugs.

Note: Herbs and botanical supplements should not be used without discussing with physician. Excerpted from The Arthritis Foundation's guide to alternative therapies, Web site accessed December 6, 2009, at http://www.arthritis.org.

Herbs, Botanicals, and Supplements

- Boswellia frereana (frankincense) suppresses cytokine-induced matrix metalloproteinase expression and production of pro-inflammatory molecules; studies are on-going (Blain et al, 2010).
- Glucosamine sulfate combined with omega-3 fatty acids may reduce OA symptoms, including morning stiffness and pain in hips and knees (Gruenwald et al, 2009). Undenatured type II collagen (UC-II) may be more effective in the treatment of OA than glucosamine and chondroitin (Crowley et al, 2009).
- Green tea's anti-inflammatory properties and ginger may aid in pain relief.
- Table 11-7 provides a description of some side effects of products often used for OA.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Physical and occupational therapies, diet, and exercise play an extremely important role. Long-term exercise and dietary weight loss are most effective. Acupuncture may be used to relieve pain.
- Because OA allows muscles around the joints to become weak, exercise and stretching should be suggested to maintain flexibility. Repetitive, high-impact movements are not recommended whereas Tai Chi helps balance and protects bones. Exercises include a series for strengthening, aerobic, agility, and range of motion. Long-term weight training, walking programs, swimming, and flexibility exercises are helpful.
- Encourage patient to avoid fad diets for "arthritis cure." Ensure that the patient's diet is balanced and includes all nutrients. A weight-loss plan may be needed.
- To alleviate stress on the joints, pharmacological and behavioral techniques with self-monitoring, should be included (Berkel et al, 2005).
- Pain initiates and exacerbates sleep disturbance; therefore, improving the sleep of OA patients helps to reduce the pain (Vitello et al, 2009). Transcutaneous electrical nerve stimulation (TENS) directs mild electric pulses to nerve endings that lie beneath the skin in the painful area; it block messages to the brain and by modifies pain perception. CBT is also useful.
- Focus on abilities and strengths rather than on disabilities and weaknesses.

Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

For More Information

- Arthritis Foundation
  http://arthritis.about.com/
- Arthritis Resource Center at Healingwell
  http://www.healingwell.com/arthritis
- NIAMS—Osteoarthritis
  http://www.niams.nih.gov/hi/topics/arthritis/oahandout.htm
- Johns Hopkins Arthritis Center
  http://www.hopkins-arthritis.som.jhmi.edu/

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OSTEOMYELITIS

NUTRITIONAL ACUITY RANKING: LEVEL 1–2

DEFINITIONS AND BACKGROUND

Acute osteomyelitis may be caused by localized infection of the long bones or injury to bone and surrounding soft tissue. *Staphylococcus aureus* is implicated in most patients with acute osteomyelitis; *S. epidermidis, S. aureus, Pseudomonas aeruginosa, Serratia marcescens,* and *Escherichia coli* may be found in the chronic form. When a bone is infected, the bone marrow swells and compresses against the rigid outer wall of bone, and blood vessels may be compressed or die; abscesses may form. Osteomyelitis and inflammatory arthritis affect many children (Pruthi and Thapa, 2009).

Some diseases predispose patients to osteomyelitis, including diabetes mellitus, sickle cell disease, acquired immunodeficiency virus (AIDS), intravenous drug abuse, alcoholism, chronic steroid use, immunosuppression, and chronic joint disease. Use of prosthetic orthopedic devices and recent orthopedic surgery or open fracture may also place a patient at risk for osteomyelitis. Patients with diabetes mellitus with poor glucose control may experience infections of the lower extremities, from superficial cellulitis to deep soft tissue infections and osteomyelitis. Because osteomyelitis is prevalent after diabetic foot ulcers, careful treatment is crucial to avoid amputation (Schinabeck and Johnson, 2005).

Prompt treatment is important. If not treated properly, the condition may become chronic with a poor prognosis. Treatment generally involves evaluation, staging, determination of etiology, antimicrobial therapy, and debridement or stabilization of bone. In children, serious musculoskeletal infections include osteomyelitis, septic arthritis, pyomyositis, and necrotizing fasciitis (Frank et al, 2005).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Deficiency of the interleukin-1-receptor antagonist (DIRA) promotes neonatal osteomyelitis, an autosomal recessive autoinflammatory disease caused by mutations in IL1RN (Aksentijevich et al, 2009).

Clinical/History

- Height
- Weight
- BMI
- Intake and output (I & O)
- BP
- Bone pain
- Sudden, acute pain in joints near the infection
- Fever, chills
- Tachycardia, diaphoresis
- Nausea
- Dehydration, electrolyte imbalance
- Local swelling, redness
- Contractures in affected extremities
- Magnetic resonance imaging (MRI)
- Bone densitometry or x-rays
- Pressure ulcers or lesions with a sinus tract?

Lab Work

- CRP and ESR (increased)
- White blood cell (WBC) count (increased)
- Gluc
- Alb, transthyretin
- BUN, Creat
- Alk phos
- AST, ALT
- Ca^{++}, Mg^{++}
- Na^{+}, K^{+}
- Vitamin D_3 status (serum 25-OHD)

SAMPLE NUTRITION CARE PROCESS STEPS

Underweight

Assessment Data: Weight and physical activity histories. Medical history, medications, and lab values.

Nutrition Diagnosis (PES): Underweight related to poor nutrition quality of life and inadequate oral intake as evidenced by insufficient pain medicine before meals and BMI of 18.

Intervention: Food and nutrient delivery—offer nutrient and energy-dense favorite foods. Educate about the benefits of gaining weight to tolerate medical therapies more effectively, to gain energy, and to improve nutritional quality of life. Coordinate timing of medicines with meals to assure that pain medicine is given 20–30 minutes before meals.

Monitoring and Evaluation: Weight gain in 3–6 months; improved timing of meals with pain medicine to decrease anorexia at mealtime.

INTERVENTION

OBJECTIVES

- Characterize and treat the infection. Prevent further infection, dehydration, and other complications.
- Promote recovery and healing of any skin lesions or pressure ulcers.
- Correct defective blood flow to allow nutrients and oxygen to reach all tissues.
- Control serum glucose and alleviate hyperglycemia with insulin if needed.
- Correct anorexia, poor intake, weight loss, nausea and vomiting where present.

FOOD AND NUTRITION

- Encourage adequate fluid intake.
- Maintain a normal to high intake of calories, protein, zinc, vitamin A, and vitamin C in particular. A multivitamin–mineral supplement may be needed.
- With diabetes, control carbohydrate to promote more effective healing.

Common Drugs Used and Potential Side Effects

- For optimal results, antibiotic therapy must be started early, with antimicrobial agents administered parenterally for at least 4–6 weeks if needed. Vancomycin or amphotericin B may be used; monitor for side effects related to timing and meals.
- Analgesics may be used for pain. GI distress is a common side effect.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- It is reasonable to include phytochemical-rich foods each day. See Table 11-2.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss role of nutrition in wound healing, immunity, and other conditions related to this disorder.
- Discuss signs that may indicate reversal of status or recovery, such as increased fever, elevated glucose levels, additional infections, more redness in affected areas.
- Promote use of nutrient-dense foods that are rich in antioxidants, phytochemicals, protein, zinc and vitamins.

Patient Education—Food Safety

- If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used. Infection control is extremely important to avoid additional microbial contamination.

For More Information

- Cleveland Clinic—Osteomyelitis
  http://my.clevelandclinic.org/disorders/osteomyelitis/hic_osteomyelitis.aspx
- Mayo Clinic—Osteomyelitis
  http://www.mayoclinic.com/health/osteomyelitis/DS00759
- National Institutes of Health–Osteomyelitis
  http://www.nlm.nih.gov/medlineplus/ency/article/000437.htm

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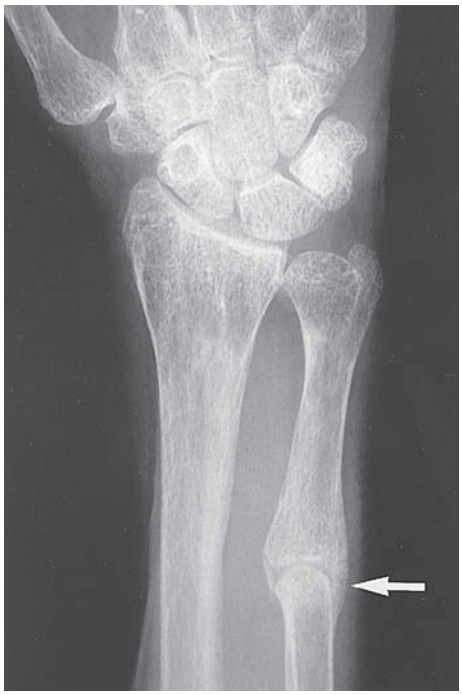
OSTEOMALACIA

NUTRITIONAL ACUITY RANKING: LEVEL 2



Adapted from: Yochum TR and Rowe LJ. *Yochum and Rowe's Essentials of Skeletal Radiology,* 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 2004.

DEFINITIONS AND BACKGROUND

Osteomalacia, adult rickets, causes softening and demineralization of the bone from insufficient vitamin D. Osteomalacia may occur in conjunction with bone loss and hip fractures. It more commonly results from intestinal malabsorption as from Crohn's disease, colon resection, cystic fibrosis, celiac disease, or chronic use of anticonvulsants. It is also seen in kidney failure, liver disease, and some types of cancer. Severe vitamin D deficiency leads to secondary hyperparathyroidism, increased bone turnover and losses.

Derangements in serum phosphate level result in osteomalacia (Saito and Fukumoto, 2009). Matrix extracellular phosphoglycoprotein (MEPE) inhibits mineralization; altered expression is associated with oncogenic osteomalacia and hypophosphatemic rickets (Boskey et al, 2010). In addition, deficient actions of fibroblast growth factor, FGF23, result in hypophosphatemic osteomalacia; FGF23 works as a hormone (Saito and Fukumoto, 2009).

Osteomalacia often occurs in older people, in dark-skinned individuals who live in northern latitudes, and in those who have limited sunlight exposure. Vitamin D is produced in response to sun exposure, so the process works faster in pale individuals. Sun exposure of about 15 minutes without sunscreen a few times a week is needed. Darker skinned individuals may need to take supplements.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: The vitamin D receptor (VDR) is responsible for the expression of over 900 genes, or about 3% of the human genome.

Clinical/History	Lab Work	
Height	Serum P (decreased)	PTH
Weight	Serum Ca^{++} (decreased)	Mg^{++}
BMI	Urinary Ca^{++}	Na^{+}, K^{+}
Bone pain, aches	Vitamin D_3 (serum 25-OHD): normal >30 ng/mL; low between 15 and 30 ng/mL; very low at less than 15 ng/mL	Serum phos
Softened, deformed bones		Alk phos (increased)
Muscular weakness, listlessness		Alb, transthyretin
Numbness of arms, feet		CRP
Easy bone fractures		BUN, Creat
Bone densitometry, DEXA		
Bone biopsy		

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Vitamin D Intake

Assessment Data: Weight, physical activity histories, medical history, medication use, lab values. Limited sunlight exposure; works indoors in a sedentary job. Complains of bone pain. Low serum vitamin D_3 at 17. Diet Hx shows limited to no intake from milk and milk products, dislike of fish, no use of multivitamin supplements.

Nutrition Diagnosis (PES): Inadequate vitamin D intake related to limited sunlight exposure, indoor job, and diet low in vitamin–D rich foods as evidenced by low serum vitamin D levels.

Intervention: Food-nutrient delivery—Encourage intake of more fortified dairy products, fish; use daily supplement containing the active form of vitamin D (cholecalciferol) in dose as prescribed by physician. Educate about the need to get sunlight exposure (20 minutes without sunscreen) several times weekly. Counsel about ways to use vitamin–D rich foods in menu planning and recipes that are acceptable to patient and/or family members.

Monitoring and Evaluation: Serum levels of 25-hydroxyvitamin D (25-OHD) at more desirable level after 2–3 months. Patient statement of acceptance of foods rich in vitamin D, such as cream soups and casseroles made with milk. No further signs of osteomalacia.

INTERVENTION

OBJECTIVES

- Provide correct amount of calcium, phosphorus, and vitamin D_3. Include other nutrients that support bone health; meet DRI levels. See Table 11-8.
- Prevent or reverse, if possible, bone density loss resulting from calcium loss in the bone matrix.
- Prevent heart disease and stroke, which may be consequences of severe vitamin D deficiency.

FOOD AND NUTRITION

- Diets should be high in calcium; adults will need 1200–1500 mg. If patient is lactose intolerant, try Lactaid or other forms of lactose-free milk, broccoli, greens, and other sources of calcium.
- Vitamin D is administered at high levels. Dietary sources include fish (particularly salmon, tuna, and mackerel) and fish liver oils. Small amounts are found in egg yolk and beef liver.
- Potassium, magnesium, vitamins C and K and other potentially important nutrients should be highlighted.

Common Drugs Used and Potential Side Effects

- Monitor treatment with calcium salts to prevent hypercalcemia; use with plenty of liquids. Avoid taking with iron supplements or bulk-forming laxatives. High-calcium diets may reduce zinc absorption and balance and may, therefore, increase zinc intake.

TABLE 11-8 Nutrients and Bone Health

Nutrient	Comments
Alcohol	Moderate drinking (1–2 glasses of wine daily) is associated with increased trochanteric bone mineral density (BMD), but higher intakes may be associated with lower BMD. Heavy alcohol consumption may be linked to tobacco use, poor dietary habits, and poor bone health.
B-complex vitamins	Folic acid and vitamins B_6 and B_{12} help to lower homocysteine when elevated.
Boron	Some role but not clearly defined.
Caffeine	Over 300 mg/d of caffeine can negatively impact the vitamin D receptor gene (*VDR*), and the Site Testing Osteoporosis Prevention and Intervention Trial (STOP-IT) found that greater amounts of caffeine affect BMD negatively (Rapuri et al, 2007). Limit intake to three cups of coffee daily and five servings of caffeinated soft drinks or tea; be sure to include adequate amounts of calcium.
Calcium and vitamin D	Dietary supplementation with calcium (1200 mg or more) and vitamin D (800–1000 IU) supports strong bone matrix, moderately reduces bone loss, and reduces the incidence of fractures. Vitamin D may actually be more important than calcium.
Copper	Copper is integral to the process of cross-linking of collagen and elastin molecules, and may have other roles in bone cells as well. Copper is found in meat, poultry, shellfish, organ meats; chocolate; nuts; cereal grains; dried legumes and dried fruits.
Dietary Fiber	A high intake of dietary fiber may interfere with calcium absorption; this may impact vegans, who consume 50 or more grams of fiber per day.
Iron	Iron is important for collagen maturation, and has other roles in osteoblasts and osteoclasts. Iron is found in organ meats, such as liver, kidney, heart; seafood; lean meat, poultry; dried beans; egg yolks; dried fruits; dark molasses; whole-grain and enriched breads or cereals.
Magnesium	Low intakes of magnesium contribute to bone loss. More than 50% of the total magnesium in the body is found in the bone, mostly in bone fluids. Magnesium is found in seeds, nuts; legumes; milled cereal grains; dark-green leafy vegetables such as spinach, broccoli, turnip greens, dark lettuces; milk.
Manganese	Manganese is necessary for the formation of bone matrix and is found in whole grains, nuts, legumes, tea, instant coffee, fruits, and vegetables.
Phosphorus	Meat, poultry, fish, eggs; cereals, grains; legumes; milk and dairy products, nuts.
Protein	70–100 g/d provides more bone building. Avoid larger doses, which can lead to excessive urinary calcium losses.
Silicon	There may be a role for silicon in stimulation of collagen synthesis and osteoblast differentiation. Intake of biologically active silicon, orthosilicic acid, enhances bone density and may help to preserve bone mass (Devine et al, 2005).
Sodium	Excesses can increase calcium excretion. Avoid using salt at the table, and limit total intake to 2400 mg/d.
Soy	Soy seems to be protective against fractures. Isoflavones increase bone density; use dietary sources.
Vitamin A	Too much retinal (not derived from the carotenoids found in plant sources) may contribute to hip fractures, especially in postmenopausal Caucasian women. Preformed vitamin A is found in liver, milk fat, fortified skim milk, eggs.
Vitamin C	Part of collagen, which supports healthy bone structure. Tissues saturate at 200 mg, therefore large doses are wasted.
Vitamin K	Supports osteocalcin for bone strength, reduces urinary calcium excretion, and modifies bone matrix proteins. A low intake of this fat-soluble vitamin increases the risk for bone fracture. Supplement with 120 μg if needed. Vitamin K is found in dark-green leafy vegetables, dairy products, meat, and eggs.
Zinc	The enzymes in osteoblasts require zinc in order to synthesize collagen. Zinc is found in animal products such as meat, fish, poultry; fortified and whole-grain cereals; milk and milk products; shellfish; liver. Zinc is also found in dry beans; nuts.

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Devine A, et al. Protein consumption is an important predictor of lower limb bone mass in elderly women. *Am J Clin Nutr.* 81:1423, 2005.

- Anticonvulsant therapy, tranquilizers, sedatives, muscle relaxants, and oral diabetic agents may deplete vitamin D. Phosphate binders with aluminum may precipitate osteomalacia; calcium carbonate may be useful, but do not take it with whole grains, bran, high-oxalate foods, or iron tablets.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Many doctors believe that between 1000–2000 IU of vitamin D per day may be needed to maintain adequate serum levels of this hormone. The upper limit for vitamin D is 10,000 IU per day.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Explain which foods are good sources of vitamin D. Encourage use of cholecalciferol (vitamin D_3) and not vitamin D_2.
- Encourage patient to spend time in the sun for skin synthesis of vitamin D; 15–20 minutes may be needed, after which use a sunscreen to avoid sunburn.
- A spoonful of cod liver oil contains about 1300 IU of vitamin D; an 8-oz glass of fortified milk contains about 100 IU.
- Vegetarians who avoid dairy products may be at risk for calcium and vitamin D depletion; discuss alternative sources from diet or from necessary supplementation.

Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

For More Information

- Mayo Clinic—Osteomalacia
  http://www.mayoclinic.com/health/osteomalacia/DS00935
- Medline—Osteomalacia
  http://www.nlm.nih.gov/medlineplus/ency/article/000376.htm#Definition

OSTEOMALACIA—CITED REFERENCES

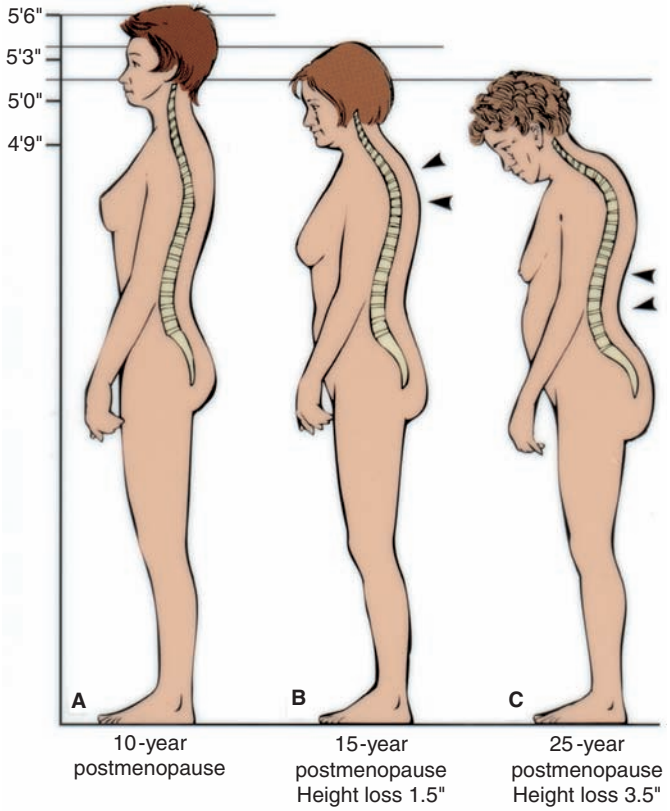
Boskey AL, et al. MEPE's Diverse Effects on Mineralization [published online ahead of print December 9, 2009]. *Calcif Tissue Int.* 86:42, 2010.

Rapuri PB, et al. Caffeine decreases vitamin D receptor protein expression and 1,25(OH)2D3 stimulated alkaline phosphatase activity in human osteoblast cells. *J Steroid Biochem Mol Biol.* 103:368, 2007.

Saito T, Fukumoto S. Fibroblast Growth Factor 23 (FGF23) and Disorders of Phosphate Metabolism. [published online ahead of print October 7, 2009] *Int J Pediatr Endocrinol.* 2009:496514.

OSTEOPENIA AND OSTEOPOROSIS

NUTRITIONAL ACUITY RANKING: LEVEL 2



Adapted from: Smeltzer SC, Bare BG. *Textbook of Medical-Surgical Nursing,* 9th ed. Philadelphia: Lippincott Williams & Wilkins, 2000.

DEFINITIONS AND BACKGROUND

Osteopenia is a decrease in the amount of calcium and phosphorus in the bones. It is identified by a decrease in bone density, which is evident through a DEXA scan. It can occur in premature infants or in adults as a result of long-term inflammatory bowel disease, especially Crohn's disease, or from low BMI. Plasma 25-hydroxyvitamin D (25-OHD) is the most sensitive indicator of BMD and clinical vitamin D_3 status.

Osteoporosis is the most common bone disease in humans; it is characterized by low bone mass, structural deterioration, and decreased bone strength in an estimated 10 million Americans (NOF, 2009).

The aging population is highly affected. Seven percent of non-Hispanic white and Asian men aged 50 and older are estimated to have osteoporosis, and 35% are estimated to have low bone mass. Men are especially vulnerable when they have renal failure, smoke, or take medications on a regular basis, such as anticonvulsants, corticosteroids, or barbiturates.

The World Health Organization (2009) defines osteoporosis as a BMD value that is 2.5 standard deviations or more below the mean of a young adult of the same sex. The lower the BMD, the greater the fracture risk. Osteoporosis can be a silent disease until a fragility fracture occurs at the hip and proximal humerus, when significant physical disability can result.

TABLE 11-9 Risk Factors for Osteoporosis

Factors That Cannot Be Changed

Advanced age	History of fracture in a first-degree relative
Caucasians (e.g., Northern European and Asian)	Low body mass index (BMI) and low muscle mass
Female gender	Personal history of fracture after age 50 years
Family history of osteoporosis	

Factors That Might Be Altered

Anorexia nervosa	Hypogonadism, as from low estrogen levels or anorexia nervosa
Current smoking	Lifetime diet low in calcium (poor diet, excess fiber)
Depression, past or current	Low testosterone levels in men
Diabetes	Low vitamin D intake or sunlight exposure
Estrogen deficiency (premature menopause, amenorrhea)	Sedentary lifestyle or extended bed rest (immobilization)
Excessive use of alcohol	Use of chemotherapy, tamoxifen, glucocorticoids, lithium, and some anticonvulsants
Homocysteine, elevated plasma levels	Total parenteral nutrition, long-term use
Hypertension	

Conditions or Diseases That May Lead to Osteoporosis

AIDS/HIV	Gastrectomy	Liver disease, severe	Primary biliary cirrhosis
Amyloidosis	Gaucher's disease	Lymphoma or leukemia	Rheumatoid arthritis
Ankylosing spondylitis	Hemochromatosis	Malabsorption syndromes	Spinal cord transection
Celiac disease	Hemophilia	Mastocytosis	Stroke
Chronic obstructive pulmonary disease	Hyperparathyroidism	Multiple myeloma	Thalassemia
Congenital porphyria	Hypophosphatasia	Multiple sclerosis	Thyrotoxicosis
Cushing's syndrome	Idiopathic scoliosis	Osteomalacia	Tropical sprue
Diabetes, type 1	**Inflammatory bowel disease**	Pernicious anemia	

Women can lose up to 20% of their bone mass in the 5–7 years following menopause; 50% of will experience an osteoporotic fracture at some point in time. About 20% of postmenopausal white women in the United States have osteoporosis and 1.5 million fractures occur annually, especially of the hip and spine. Falls are associated with a high risk of frailty fractures (Schwartz et al, 2005).

Spinal or vertebral fractures may lead to loss of height, severe back pain, and spinal deformities such as kyphosis or stooped posture. Hip fractures require hospitalization and major surgery; they impair the ability to walk and may cause disability or death. By 2050, the annual number of hip fractures is expected to triple (World Health Organization, 2009).

Awareness and management of risk factors is important for preventing osteoporosis and the related disability. Both genetic and lifestyle factors play a role. A family history of hip fracture carries a twofold increased risk of fracture among descendants; genetic factors play a major role in BMD and in osteoporosis risk (Ferrari, 2008). Yet BMD is just one of many contributors to bone strength and fracture risk reduction. Dairy, fruit, and vegetable intakes have emerged as an important modifiable protective factor for bone health (Tucker, 2009). Women may lose bone during lactation if their diets are low in calcium and other nutrients. Magnesium, potassium, vitamin C, vitamin K, several B vitamins, and carotenoids are important (Tucker, 2009); see Table 11-8.

In the skeleton, interleukin-1 protein causes an increase in the number and activity of osteoclastic cells—the cells that break down bone tissue. Depression and elevated plasma homocysteine levels are also associated with osteoporosis. See Table 11-9 for the full list of risk factors for osteoporosis.

Physical activity has different effects depending on its intensity, frequency, and duration, and the age at which it is started, with greater effects in adolescence and as a result of weight-bearing exercise. In addition, diet contributes significantly. Building strong bones during childhood and adolescence can be the best defense against developing osteoporosis later. By about age of 20, most women have acquired 98% of total bone mass. Acquisition of a high peak bone mass (reaching genetic potential) by 30 years of age helps reduce bone losses later in life.

Markers of bone turnover can be used to predict the rate of bone loss in post-menopausal women and can also be used to assess the risk of fractures (Eastell and Hannon, 2008). Markers of bone formation include serum bone alkaline phosphatase, total osteocalcin and the procollagen type I N-terminal propeptide assay (Eastell and Hannon, 2008).

Serotonin can serve as a marker for low bone mass. Circulating levels of the neurotransmitter serotonin are inversely associated with bone mass in women; this may have implications when using SSRIs. Bone formation is inhibited by serotonin in the gut. Inactivation of the leptin receptor in serotonergic neurons identifies a molecular basis for the common regulation of bone and energy metabolisms (Yadav et al, 2009). A drug that stops the gut from synthesizing serotonin may be able to reverse severe bone loss and prevent osteoporosis.

Measurements of the urinary excretion of N- and C-terminal cross-linked telopeptides and of serum C-terminal cross-linked telopeptides are sensitive and specific for bone resorption (Eastell and Hannon, 2008). These measures are as important as measurement of BMD.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Genes coding for the LDL-receptor related protein 5 (LRP5), estrogen receptor alpha (ESR1), and osteoprotegerin, OPG (TNFRSf11b) are known to pose a risk for osteoporosis (Ferrari, 2008). In a large-scale study, single nucleotide polymorphisms (SNPs) from nine gene loci (ESR1, LRP4, ITGA1, LRP5, SOST, SPP1, TNFRSF11A, TNFRSF11B, and TNFSF11) were associated with BMD, but not always in a predictable manner (Richards et al, 2009).

Clinical/History

- Height
- Weight
- BMI
- Back pain
- BP
- Bone densitometry, DEXA

Lab Work

- Ca^{++}
- Urinary Ca^{++} (24 hours)
- Mg^{++}
- Na^{+}, K^{+}
- Vitamin D_3 status (serum 25-OHD)
- Alb
- CRP
- PTH (useful in some patients)
- Serum P (decreased with hyperparathyroidism)
- TSH
- Total testosterone in men
- Serum homocysteine
- Serum folate and vitamin B_{12}

INTERVENTION

OBJECTIVES

- Preserve height, support independence, and improve functional status. Prevent fractures (Tussing and Chapman-Novakofski, 2005).

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Vitamin Intake

Assessment Data: Food records, DEXA scan showing two deviations below normal. Weight and physical activity histories. Medical history, medications, and lab values.

Nutrition Diagnosis (PES): Inadequate intake of vitamin D (NI5.9.1.3) related to poor diet, no food sources, and no use of supplements as evidenced by very low BMD on DEXA.

Intervention: Food and Nutrient Delivery: ND 1.3—add calcium containing beverages and foods, vitamin–D rich foods, adequate protein. ND 3.2.3.3—Supplement use for Vitamin D.

Education: E 2.2—Educate patient concerning calcium and vitamin D food and beverage sources to meet needs along with supplement, sunlight exposure 10–30 min/d, protein needs, increased physical activity.

Counseling: Good sources of vitamin D from diet and supplements, meal planning and shopping tips, dining out guide, referral to Meals-on-Wheels or other social agencies as appropriate, appropriate role for sunshine exposure.

Monitoring and Evaluation: Improvements in dietary and supplemental intake of vitamin D as shown in food records, lab values. DEXA scan report showing no further decline, and a slight improvement in risk level. No fractures.

- Optimize bone health: choose a balanced diet rich in calcium and vitamin D; use weight-bearing and resistance-training exercises. Follow a healthy lifestyle with no smoking.
- Decrease precipitating factors, such as use of anticonvulsants or corticosteroids, lactase deficiency, low intake of fruits and vegetables and dairy, calcium malabsorption, sedentary lifestyle, and low BMI. Provide adequate time for evaluating improvement (6–9 months at least).
- Assure adequate intake of protein. Rather than having a negative effect on bone, protein intake appears to benefit bone status, particularly in older adults (Tucker, 2009).
- Intake of magnesium, potassium, fruit, and vegetables is positively associated with bone health and total bone mass.

FOOD AND NUTRITION

- Advise all patients to consume adequate amounts of calcium (≥1200 mg/d, including supplements if necessary) and vitamin D. Women after menopause or over age 65 years will need 1500 mg calcium daily. To fulfill the requirement, 1 quart of milk daily can be consumed. If fluid milk is not consumed, dry skim milk powder can be added to many foods. Aged cheeses and yogurt are sources as well.
- Calcium supplements can be used if dairy products are not tolerated; calcium absorption averages approximately 30–40% from most sources See Table 11-10. Space the supplements throughout the day; take no more than 500–600 mg two or more times daily with meals. Use with vitamin D and magnesium.
- For vitamin D, choose fortified milk, cod liver oil, egg yolks, and fatty fish. Supplements may be needed. Do not exceed 10,000 IU/d.

TABLE 11-10 Tips on Calcium Supplements[a]

Product	Source of Calcium (mg)	No. of Tablets/Day to Provide About 900–1000 mg Calcium Per Tablet
Caltrate 600	Carbonate (600 mg)	1.5
Os-Cal 500	Carbonate from oyster shell (500 mg)	2
Os-Cal 500 + Vitamin D	Carbonate from oyster shell (500 mg)	2
Posture (600 mg)	Phosphate (600 mg)	1.5
Posture–Vitamin D	Phosphate (600 mg)	1.5
Citracal	Citrate (200 mg)	5
Citracal + Vitamin D	Citrate (315 mg)	3
Citracal Liquitab	Citrate (500 mg)	2
Tums 500 mg	Carbonate from limestone (500 mg)	2
Tums E-X	Carbonate from limestone (300 mg)	3.5
Tums Ultra	Carbonate from shell (400 mg)	2.5
Calcet + Vitamin D	Carbonate, lactate, gluconate (300 mg)	3.5
Fosfree	Carbonate, gluconate, lactate (175 mg)	6

[a]Excesses of calcium supplements can cause hypercalcemia; monitor intakes carefully and take no more than 500–600 mg two or more times daily with meals. Avoid taking with iron supplements. Use extra water with supplements. Excess vitamin D can cause vitamin D calcinosis. Rates of calcium absorption vary, and dietary sources are the best absorbed; calcium maleate is also well absorbed. Elemental calcium varies in different supplements, as follows:

1. Calcium carbonate (Tums, Roxane, Os-Cal, Calciday, Oyst-Cal, Oystercal, Caltrate) contains 40%. Calcium carbonate temporarily decreases gastric acidity, which is needed for calcium absorption. Tricalcium phosphate provides 39%.
2. Calcium chloride contains 36%.
3. Bone meal or Dolomite contains 33% but should be avoided as it also contains lead.
4. Calcium acetate (Phos-Ex, PhosLo) contains 25%.
5. Calcium citrate (Citracal) contains 21%.
6. Calcium lactate contains 13%.
7. Calcium gluconate contains 9%.

Updated from: Shils M, et al, eds. *Modern nutrition in health and disease.* Baltimore: Lippincott Williams & Wilkins, 1999.

- Extra protein may be needed (Devine et al, 2005; Tucker, 2009).
- For sufficient intake of vitamin B_{12}, include dairy products, meat, poultry, fish, and fortified cereals.
- Isoflavones may also be beneficial; use two to three servings of soy foods daily.
- If patient is obese, use a nutrient-rich, calorie-controlled diet that provides adequate protein, vitamins, calcium, and other minerals. Adequate manganese, vitamins C and K, potassium, and magnesium should be consumed to meet at least the DRI levels. Include fruits and vegetables that contribute to bone health.
- Assure that folic acid and vitamins B_6 and B_{12} are adequate, especially if serum homocysteine levels are elevated.
- Sodium must be controlled. Keep sodium within desired limits while increasing potassium and magnesium.
- Beware of excesses of wheat bran because phytates may increase calcium excretion.
- Caffeine from coffee does not seem to be a problem if calcium (as from milk) is consumed in adequate amounts. However, cola drinks should be limited (Tucker, 2009).
- Moderate alcohol intake shows positive effects on bone, particularly in older women (Tucker, 2009).

Common Drugs Used and Potential Side Effects

- Adequate calcium is crucial; supplementation in bioavailable forms is necessary in individuals who do not achieve recommended intake from dietary sources (see Table 11-11). Side effects may include abdominal pain, anorexia, constipation, vomiting, nausea or dry mouth.
- Oral doses of vitamin D_3 in the range of 1800 to 4000 IU per day may be needed to take serum levels up to 75 to 110 nmol/L (Bischoff-Ferrari et al, 2010).
- Oral alendronic acid is the reference drug for menopausal women with osteopenia. It may be used with parathormone as well, but this has a 2-year limit (Black et al, 2005). See Table 11-12 for more guidance.
- The once monthly injections of risedronate (150 μg) are beneficial for those for which daily or weekly dosing is a challenge (Rackoff, 2009). Bone markers can be used to monitor the efficacy of antiresorptive therapy such as hormone-replacement therapy, raloxifene and bisphosphonates (Eastell and Hannon, 2008).
- SSRIs have been associated with lower BMD and increased rates of bone loss, as well as increased rates of fracture (Haney et al, 2010). Their use should be closely monitored.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Cabbage, pigweed, dandelion, avocado, and parsley have been recommended, but have not shown efficacy.

TABLE 11-11 Medications Commonly Used for Management of Osteoporosis[a]

Medication Comments	Effects	Comments
Bisphosphonates: risedronate (Actonel); alendronate (Fosamax)	Effective agents for reducing vertebral and nonvertebral fracture risk. Alendronate is approved for the treatment of osteoporosis in men. Alendronate and risedronate are approved for use by men and women with glucocorticoid-induced osteoporosis. Zoledronic acid is under study. Bisphosphonates inhibit atherogenesis.	Risedronate may cause dysphagia, esophageal ulcer, and stomach ulcer. Take on an empty stomach 30 minutes before meals; sit upright. Take additional vitamin D and calcium. Headache, gastrointestinal (GI) distress, diarrhea, nausea, constipation, and rash may occur, although rarely. Alendronate may cause metallic taste, nausea, diarrhea, and decreased potassium and magnesium. Avoid in severe renal disease, pregnancy, or breastfeeding. Nausea, heartburn, irritation or pain of the esophagus, anorexia, vomiting, dysphagia, sensation of fullness, and constipation or diarrhea may occur.
Calcitonin-salmon (Miacalcin)	Bone loss is reduced, and bone mass increases, although not in the hip. A modest increase in bone mass occurs.	200 IU/d, the recommended regimen, reduces vertebral fracture risk by 33% in women with low bone mass. Calcitonin makes calcium more available to bones. It is given as an injection or nasal spray; it may cause allergic reactions and flushing of the face and hands, urinary frequency, anorexia, nausea, constipation, or skin rash.
Calcitriol (1,25-dihydroxyvitamin D)	Active form of vitamin D hormone that increases GI absorption of calcium from the gut, kidney reabsorption of calcium, stimulates bone resorption, decreases PTH production, and stimulates skeletal osteoblasts/osteoclasts. Larger doses than the DRI for vitamin D may be needed; 700–800 IU may be beneficial along with 500–1200 mg calcium (Cranney et al, 2007).	Anorexia, abdominal cramping, headache, lethargy, nausea, weight loss, and weakness may result from larger doses.
Ibandronate (Boniva)	Ibandronate is used to treat or prevent osteoporosis in women after menopause; it may increase bone mass by slowing loss of bone.	Should not be taken if hypocalcemia is a problem.
PTH (teriparatide; Forteo)	PTH is the only anabolic osteoporosis agent available for clinical use to lower vertebral fracture incidence by triggering formation of new bone.	Use only in ambulatory patients.
Raloxifene (Evista)	Significantly reduces vertebral fracture risk but not nonvertebral fracture risk.	Protects against thin, weak bones and fractures; also lowers serum cholesterol by 7% and low-density lipoprotein (LDL) by 11%. It may trigger menopausal symptoms, including hot flashes, but is less likely to have an estrogen-like increase in cancer risk.
Sodium fluoride	The slow-release form may increase bone formation and decrease the risk of fractures.	In patients with mild-to-moderate osteoporosis, long-term supplements with fluoride plus calcium result in lower rates of vertebral fracture than supplementation with calcium alone. Intake of fluoride in drinking water at 1 ppm does not appear to be associated with increased risk of hip fracture. Side effects may include abdominal pain, diarrhea, nausea, vomiting.
Statins	Statins, agents that reduce atherogenesis, stimulate bone formation.	Cardiovascular disease and low bone mineral density have some common etiologies.

[a]The FDA approves calcitonin, alendronate, raloxifene, and risedronate for the treatment of postmenopausal osteoporosis; alendronate, risedronate, and raloxifene are approved for the prevention of the disease. Current pharmacological options for osteoporosis prevention and/or treatment are bisphosphonates (alendronate and risedronate), calcitonin, estrogens and/or hormone therapy, parathyroid hormone (PTH 1–34), and raloxifene.
Source: National Osteoporosis Foundation Web site accessed December 14, 2009, at: http://www.nof.org/patientinfo/medications.htm.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Prevention is the best medicine. Encourage patient to stand upright, rather than sit or recline, as often as feasible. Measures to decrease fall frequency and to slow down the rapid life pace of healthy people with low bone mass should prevent some fractures (Kelsey et al, 2005).
- Change a sedentary lifestyle. Regular resistance and high-impact exercise, contributes to development of high peak bone mass and may reduce the risk of falls in older individuals (Moayyeri, 2008). Aerobic and strengthening exercises are helpful as well.
- Walking or running is beneficial. However, excessive weight-bearing exercise can cause amenorrhea in premenopausal women when a low-calorie diet is consumed.

TABLE 11-12 Features of Rheumatic Arthritis

Tender, warm, swollen joints
Symmetrical pattern of affected joints
Joint inflammation *often* affecting the wrist and finger joints closest to the hand
Joint inflammation *sometimes* affecting other joints, including the neck, shoulders, elbows, hips, knees, ankles, and feet
Fatigue, occasional fevers, a general sense of not feeling well
Pain and stiffness lasting for more than 30 minutes in the morning or after a long rest
Symptoms that last for many years
Variability of symptoms among people with the disease

Source: National Institute of Arthritis and Musculoskeletal and Skin Diseases. Available at http://www.niams.nih.gov/Health_Info/Rheumatic_Disease/default.asp#ra_14.

- An educational osteoporosis prevention program with hands-on activities can increase self-efficacy (Tussing and Chapman-Novakofski, 2005). Explain that calcium absorption declines with age and that adequate calcium and vitamin D are important throughout life. The overall benefit of healthful eating must be strongly emphasized. Consider calcium and vitamin D supplementation in the elderly.
- Describe importance of the use of milk, cheeses, yogurt, broccoli, kale and other greens, and soybeans. Provide recipes and shopping tips.
- Decrease the use of tobacco. Use only moderate amounts of alcohol.
- Caffeine poses a minimal risk unless it replaces calcium-containing beverages; BMD is not affected by caffeine if at least 1 glass of milk is consumed daily.
- Encourage adequate exposure to sunlight (10–30 min/d). Avoid sunburn and overexposure, with its risks of skin cancer.
- Remind all teenagers that osteoporosis is "kid stuff;" maintenance of weight-bearing activity is important during the growing years. Intake of carbonated beverages instead of milk is a big concern.
- Some mineral waters are excellent sources of calcium; bioavailability is good.
- Avoid long-term use of high doses of retinol from fortified foods or supplements.
- Persons with previous fractures are at risk and should be monitored carefully for osteoporosis. The National Osteoporosis Foundation supports an Awareness and Prevention Month in May of each year.
- When steroids are used, check on bone density changes; there is a high incidence of osteoporosis.
- Note that improvements in BMD may take up to 3 years to note improvement (Compston, 2009).

Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

For More Information

- Clinical Guidelines–Osteoporosis
  http://www.nof.org/professionals/clinical.htm
- National Osteoporosis Foundation
  http://www.nof.org/
- Osteopenia
  http://www.nlm.nih.gov/medlineplus/ency/article/007231.htm

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PAGET'S DISEASE (OSTEITIS DEFORMANS)

NUTRITIONAL ACUITY RANKING: LEVEL 1–2

DEFINITIONS AND BACKGROUND

Paget's disease of the bone (PDB) is a disorder of skeletal remodeling, where areas on bone grow abnormally, enlarging and becoming soft. It is of unknown etiology, with excessive bone destruction and repairing. Of all persons older than 50 years of age, 3% have an isolated lesion; actual clinical disease is much less common. PDB is the second most common bone disease in the world. A systematic laboratory screening including serum alkaline phosphatase of an older subject complaining of bone pain, articular pain, or back pain is a strategy to improve the diagnosis of PDB (Varenna et al, 2009).

The disease tends to run in families. Genetic analysis indicates that 40% of patients with Paget's disease have an affected first-degree relative. Approximately 3 million Americans have the disease; it rarely occurs before age 40. Juvenile Paget's disease is very debilitating. Osteoclasts are larger than normal and increased in size (Deftos, 2005). Juvenile Paget's disease usually presents in infancy or childhood and results in progressive deformity, growth retardation, and deafness.

The disease is higher in frequency in people who are aged 65 or older. There is a slight male predominance. Prognosis is good in mild cases.

Sarcoma can also be found in this population (Mankin and Hornicek, 2005). There has been a decline in incidence of this complication but where it does occur, prognosis is still poor (Mangham et al, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: There seem to be strong ties to European ancestry in Paget's disease, including Australia and New Zealand. A majority of cases harbor germline mutations in the SQSTM1, Sequestosome1 gene (Merchant et al, 2009).

Clinical/History

- Height
- Weight
- BMI
- Deep "bone pain"
- Joint pain, neck pain
- Skull enlargement
- Hearing loss
- Headaches
- Tickening of long bones
- Bowing of limbs
- Reduced height
- Spontaneous fractures
- X-rays (denser, expanded bones)
- Bone scans

Lab Work

- Alk phos (increased)
- Urinary Ca^{++} (altered)
- Vitamin D_3 status (serum 25-OHD)
- Uric acid (UA), elevated?
- PTH (abnormal)
- Ca^{++}, Mg^{++}
- Na^{+}, K^{+}
- Alb, transthyretin
- CRP
- Transferrin
- Serum P
- H & H
- Serum B_{12}
- Radiolabeled bisphosphonate

INTERVENTION

OBJECTIVES

- Prevent complications, especially related to the nervous system (e.g., fractures, spinal stenosis, paraplegia, cardiac failure, and deafness).
- Prevent side effects of drug therapy.
- Promote full recovery when possible.
- Differentiate from other conditions with bone lesions.
- Alleviate anemia and other complications.

FOOD AND NUTRITION

- Adequate protein is important, with adequate calories to spare protein.
- Adequate levels of calcium and vitamins C and D may be needed.
- To correct anemia, monitor serum levels of iron and vitamin B_{12} to determine need for an altered diet.

Common Drugs Used and Potential Side Effects

- Drugs that inhibit bone resorption—bisphosphonates (etidronate, pamidronate, clodronate, or alendronate)—

SAMPLE NUTRITION CARE PROCESS STEPS

Abnormal Nutritional Labs

Assessment Data: Weight and physical activity histories. Medical history, medications, abnormal lab values for alk phos, uric acid, serum vitamin D_3, and PTH.

Nutrition Diagnosis (PES): Abnormal nutritional labs related to metabolic changes from Paget's disease as evidenced by increased alk phos, altered urinary calcium, abnormal PTH, elevated uric acid levels, diagnosis of anemia, and bone pain.

Intervention: Treatment with bisphosphonates to alter serum and urinary labs; careful monitoring for side effects affecting intake and appetite. Food-nutrient delivery—Correct iron-deficiency anemia from poor intake and disease process.

Monitoring and Evaluation: Improvement in serum lab values; resolution of anemia; decreased bone pain.

may be used to slow the progression. Bisphosphonates are pyrophosphate analogs that bind to bone at active sites of remodeling. The bisphosphonate zoledronic acid (Zometa), given in a single injection, yields a rapid and long-lasting improvement in bone health (Reid et al, 2005). The nitrogen-containing BPs pamidronate (Aredia) and zoledronic acid (Zometa) are capable of causing bisphosphonate-associated osteonecrosis of the jaw (Grewal and Fayans, 2008).

- Risedronate (Actonel) can cause dysphagia, esophageal ulcer, and stomach ulcer. Take on an empty stomach 30 minutes before meals; consume additional vitamin D and calcium. Headache, diarrhea, nausea, constipation, and rash may occur, although they are rare.
- Osteoprotegerin may be used in managing the juvenile form of Paget's disease (Cundy et al, 2005).
- Thyrocalcitonin or synthetic calcitonin may be used to decrease passage of calcium from bones to bloodstream. Methods of administration include a nasal spray. Monitor for nausea or vomiting.
- Analgesics may be needed for pain.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- Unusual bone diseases may be associated with use of Chinese herbs.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss appropriate dietary alterations for patient's condition, individualized for the current condition and status. Include good food sources of calcium, B-complex vitamins, iron, protein, and vitamin D. Monitor carefully, if supplements are used, in addition to dietary guidance.
- Discuss side effects for the specific drugs ordered.

Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

For More Information

- National Association for the Relief of Paget's Disease
  http://www.paget.org.uk/
- National Institute of Arthritis and Musculoskeletal and Skin Diseases
  http://www.niams.nih.gov/bone/hi/paget/diagnosed.htm
- National Institutes of Health Osteoporosis and Related Bones Diseases
  http://www.niams.nih.gov/bone/
- Paget's Disease
  http://www.nlm.nih.gov/medlineplus/ency/article/000414.htm
- Paget Foundation
  http://www.paget.org/

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POLYARTERITIS NODOSA

NUTRITIONAL ACUITY RANKING: LEVEL 1–2

DEFINITIONS AND BACKGROUND

Polyarteritis nodosa (PAN) is characterized by necrotizing inflammation of medium- or small-sized arteries without glomerulonephritis or vasculitis in arterioles, capillaries, or venules (Colmegna and Maldonado-Cocco, 2005). Viral infections such as hepatitis B may trigger it.

In PAN, arteries become inflamed in several organs, causing damage in brain, heart, liver, GI tract, or renal tissues. Renal involvement develops and is accompanied by hypertension in half of patients. PAN also commonly involves the gut (abdominal angina, hemorrhage, perforation), heart (myocarditis, myocardial infarction), or eye (scleritis); rupture of renal or mesenteric microaneurysms can also occur. PAN is two to three times more common in men and usually develops between ages 40 and 50 years. Rarely, it occurs after a Hepatitis-B vaccination.

It is fatal if not treated. Treatment includes use of prednisone, plasmapheresis to remove immune complexes, and antiviral therapy (lamivudine) for the hepatitis B infection.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Medium-sized artery vasculitides that occur in childhood manifest mostly as PAN, with high morbidity and mortality rates (Dillon et al, 2009). PAN is likely to have a genetic connection; MEFV is one gene under study.

Clinical/History

Height
Weight
BMI
Weight loss >4 kg since onset of illness
Hematuria
Edema
Chest pain
Tachycardia
Shortness of breath
Fever?
Abdominal pain
BP (elevated)
Myalgias, weakness
Neuropathy
Hematuria
Fatigue
Rash, nodules or Raynaud's disease
Biopsy of medium vessels

Lab Work

ESR (elevated)
CRP
Glucose
Hepatitis B antigen or antibody in serum
Ca^{++}, Mg^{++}
Na^{+}, K^{+}
Alb, transthyretin
BUN, Creat (elevated but not from dehydration)
Transferrin
H & H
Vitamin D_3 status (serum 25-OHD)

INTERVENTION

OBJECTIVES

- Treat as soon as possible to decrease heart and renal damage.
- Improve appetite and intake.
- Prevent weight loss.
- Increase calorie intake when there is fever.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Oral Food and Beverage Intake

Assessment Data: Weight and physical activity histories. Medical history, medications, and lab values.

Nutrition Diagnosis (PES): Inadequate oral food and beverage intake related to anorexia, fever and abdominal pain as evidenced by weight loss of 3 kg in 8 weeks.

Intervention: Food-Nutrient delivery—Offer preferred foods, enhanced with energy kilocalories from milk powder, fats, etc. Educate about how to manage nausea; suggest small, frequent meals throughout the day and liquids separate from meals. Coordinate care with nursing, medical teams if medications are needed.

Monitoring and Evaluation: Resolution of weight loss; no further losses. Improvement in nausea and abdominal pain.

- Reduce edema, anorexia, hypertension, and other effects of the disorder.

FOOD AND NUTRITION

- A high-energy intake may be beneficial in case of weight loss.
- A normal to high protein intake generally is required.
- Fluid or sodium intake may be limited with hypertension, kidney disease, or edema or with use of steroids.
- Include phytochemicals derived from spices such as turmeric (curcumin); red pepper (capsaicin); cloves (eugenol); ginger (gingerol); cumin, anise, and fennel (anethol); basil and rosemary (ursolic acid); garlic (diallyl sulfide, *S*-allylmercaptocysteine, and ajoene); and pomegranate (ellagic acid) (Aggarwal and Shishodia, 2004).

Common Drugs Used and Potential Side Effects

- Steroids such as prednisone may be used for 2 weeks. Side effects of long-term use include negative nitrogen and potassium balances; decreased calcium and zinc levels; CHO intolerance; and excessive sodium retention. With weight gain, a calorie-controlled diet may be useful.
- Pain relievers may be needed; monitor individually for side effects such as GI distress.
- Immunosuppressive cyclophosphamide may be used; long-term effects can reduce the ability to fight infections. Corticosteroids plus cyclophosphamide is the standard of care, in particular for patients with more severe disease, in whom this combination prolongs survival (Colmegna and Maldonado-Cocco, 2005).
- Infliximab may be used as an alternative agent for the treatment of patients with PAN refractory to conventional therapy (Al-Bishri et al, 2005).

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss alternate dietary guidelines as appropriate for medications and side effects of the disease.
- Discuss sources of nutrients as appropriate for the ordered diet. Provide guidance on enhancing nutrient and energy density from meals and snacks.
- With abdominal pain and GI bleeding, PAN occasionally is mistaken for inflammatory bowel disease. Be certain to see a trained specialist as needed.

Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

For More Information

- Johns Hopkins Vasculitis Center
  http://vasculitis.med.jhu.edu/typesof/polyarteritis.html
- Polyarteritis Nodosa Foundation
  http://www.angelfire.com/pa3/autoimmunedisease/aifeindex.html
- Polyarteritis Nodosa
  http://www.emedicine.com/ped/topic1844.htm
- Vasculitis Foundation
  http://www.vasculitisfoundation.org/polyarteritisnodosa

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RHABDOMYOLYSIS

NURITIONAL ACUITY RANKING: LEVEL 3

DEFINITIONS AND BACKGROUND

Rhabdomyolysis (RML) is a clinical and biochemical syndrome resulting from skeletal muscle injury with release of myoglobin into the plasma and breakdown of muscle fibers with release into the circulation. Some of these changes are toxic to the kidney, often resulting in kidney damage or acute renal failure. A disturbance in myocyte calcium homeostasis takes place.

RML may occur in infants, toddlers, and adolescents who have inherited enzyme deficiencies of carbohydrate or lipid metabolism, DMD, or malignant hyperthermia. RML may also occur from extensive muscle damage as from a crushing injury, major burn, electrical shock, toxins, bacterial infections, excessive exercise (Olpin, 2005), seizures, alcoholism, overdose of cocaine, or use of drugs such as cholesterol-reducing statins. The most common causes of RML in adults include crush injury, overexertion, alcohol abuse, use of certain medicines, and toxic substances. Postoperative RML in bariatric surgery occurs with prolonged muscle compression; potential consequences may lead to death (de Menezes Ettinger et al, 2005).

Muscle pain caused by RML may involve specific symptoms of groups of muscles or may be generalized throughout the body. Muscles in the calves and the lower back are commonly affected but each patient is different. Early complications of RML include severe hyperkalemia with cardiac arrhythmia and arrest. The most serious late complication is acute renal failure.

RML can be defined with CK values exceeding 10–25 times the upper limit of normal irrespective of renal function (Linares et al, 2009). Management of suspected drug-induced myopathy should include immediate discontinuation of the offending agent and supportive care (Mor et al, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: RML may occur in infants, toddlers, and adolescents who have inherited enzyme deficiencies of carbohydrate or lipid metabolism or who have inherited myopathies. Elevated CK levels have been found with a high-density SNP genotype in a p.Trp3X allele; this mutation is associated with a mild Becker phenotype of MD (Flanigan et al, 2009).

Clinical/History

- Height
- Weight
- BMI
- Weight gain (unintentional)
- I & O
- Tea-colored urine
- Temperature
- BP (elevated)
- Exposure to toxic substances or chronic alcohol use
- Use of medications such as statins
- Muscle tenderness
- Weakness of the affected muscles
- Muscle stiffness or aching (myalgia)
- Seizures
- Joint pain
- Fatigue
- Abnormally dark colored urine from excretion of myoglobin

Lab Work

- Creatine phosphokinase (CPK) (very high)
- Serum myoglobin test (positive)
- Urinary casts or hemoglobin
- Ca^{++}, Mg^{++}
- Na^{+}
- K^{+} (may be high from muscle breakdown)
- Alb, transthyretin
- CRP
- BUN
- Creat
- Transferrin
- H & H
- UA (elevated)
- Vitamin D_3 status (serum 25-OHD)

INTERVENTION

OBJECTIVES

- Preserve renal function.
- Eliminate myoglobin out of the kidneys with early and aggressive hydration. Medicines may also be needed to make the urine more alkaline.
- Treat kidney failure or hyperkalemia if needed.

FOOD AND NUTRITION

- Hydration needs with muscle necrosis may approximate the massive fluid volume needs of a severely burned patient.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Physical Activity

Assessment Data: Weight and physical activity histories. Medical history with diagnosis of RML.

Nutrition Diagnosis (PES): Excessive physical activity related to 2-hour workouts twice daily as evidenced by complaints of weakness, muscle stiffness and tenderness, and very high CPK levels.

Intervention: Education about a more desirable level of physical activity to lessen strain on muscles. Counseling about nutrition for athletics and maintenance of other healthy habits, including adequate rest and sleep. Physical activity logs.

Monitoring and Evaluation: Improvement in muscle stuffness and tenderness; CPK levels returning to a normal range. Physical activity logs showing workouts no longer than 30 minutes twice daily.

- Special dietary advice is required if there is renal disease or the need for dialysis.
- It is important to offer advice according to the medical condition that preceded RML. Avoidance of fasting, feeding with a high-carbohydrate and low-fat diet, and intravenous drip infusion soon after every onset of RML may be needed for children (Korematsu et al, 2009).

Common Drugs Used and Potential Side Effects

- Statins block the enzyme in the liver that is responsible for making cholesterol, hydroxy-methylglutaryl-coenzyme A reductase (HMG-CoA reductase).
- Despite the withdrawal of cerivastatin because of fatal RML, the risk of this complication with other statins is extremely low (Waters, 2005). Options for managing statin myopathy include statin switching, particularly to fluvastatin or low-dose rosuvastatin; nondaily dosing regimens; nonstatin alternatives, such as ezetimibe and bile acid-binding resins; and coenzyme Q10 supplementation (Joy and Hegele, 2009).
- Diuretic therapy may be needed if there is hypertension.
- If there is hyperkalemia, calcium chloride or calcium gluconate may be used.

Herbs, Botanicals, and Supplements

- Health care practitioners must take an active role in identifying patients who are using CAM and provide appropriate patient education (Gabardi et al, 2007). Herbs and botanical supplements should not be used without discussing with physician. There are 17 dietary supplements that have been associated with direct renal injury, CAM-induced immune-mediated nephrotoxicity, nephrolithiasis, RML with acute renal injury, and hepatorenal syndrome (Gabardi et al, 2007).
- Even brief exposure to atorvastatin causes a marked decrease in blood coenzyme Q10 concentration, with commonly reported adverse effects of exercise intolerance, myalgia, and myoglobinuria.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss alternate dietary guidelines as appropriate for medications and side effects of the disease.
- Discuss how to use diet and exercise to manage high serum cholesterol if this information has not been given before. Reinforce what the patient has been doing well.
- After damage to any muscles, extra fluid is needed to dilute urine and to eliminate myoglobin. Among soldiers, RML occurs in 25% of those who are injured (Carter et al, 2005).

Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

For More Information

- E-medicine
  http://www.emedicine.com/emerg/topic508.htm
- Rhabdomyolysis
  http://www.nlm.nih.gov/medlineplus/ency/article/000473.htm

RHABDOMYOLYSIS—CITED REFERENCES

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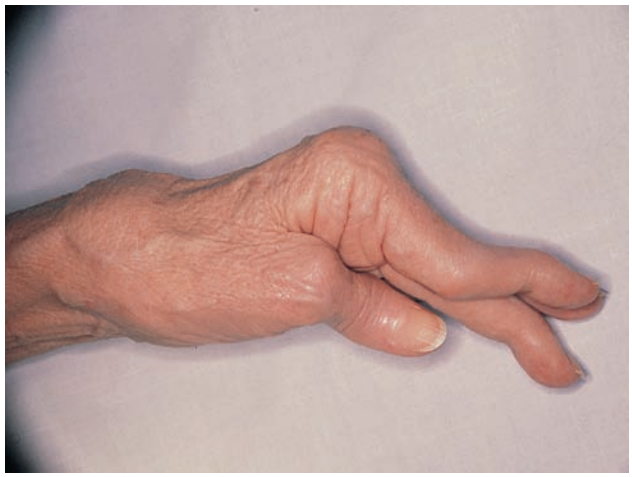
Waters DD. Safety of high-dose atorvastatin therapy. *Am J Cardiol.* 96:69, 2005.

RHEUMATOID ARTHRITIS

NUTRITIONAL ACUITY RANKING: LEVEL 1



Adaprted from: Strickland JW, Graham TJ. *Master Techniques in Orthopeadic Surgery: The Hand*, 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2005.

DEFINITIONS AND BACKGROUND

RA is a chronic polyarthritis mainly affecting the smaller peripheral joints and is accompanied by general ill health. Crippling deformities can occur. Of all cases, 75% are women. Most patients are between ages 20 and 40, and RA affects over 1.3 million Americans. To diagnose RA, symptoms must have been present for at least 6 weeks; see Table 11-12.

The cause of RA is increased inflammatory cytokine production, such as from mast cells, interleukin-6, tumor necrosis factor alpha (TNFα), and acute-phase proteins. Inflammation of synovial tissues is the dominant manifestation. Antibodies against IgG and collagen are noted. Hand involvement and knees or ankles/feet are involved in most. Table 11-13 provides a list of the variant forms of RA.

Some studies show an improvement in RA symptoms over the short term with a diet high in omega-3s or fish oil supplements. Omega-3 fatty acids reduce tenderness in joints, decrease morning stiffness, and reduce the amount

TABLE 11-13 Variant Forms of Rheumatic Arthritis (RA)

Condition	Background	Nutritional Implications
Juvenile RA (JRA)	JRA causes joint inflammation and stiffness for more than 6 weeks in a child 16 years of age or less. It is classified into three types, depending on symptoms, number of joints involved, and presence or absence of antibodies in the blood. Pauciarticular JRA is most common and affects mainly the knees. The polyarticular form affects 30% of children with JRA. Stills disease is the systemic form; it tests negative for the usual antibodies, may affect internal organs, may become chronic in adulthood and affects 20% of children with JRA. Both genetic factors and environmental factors, such as a virus, can trigger JRA. Because JRA often affects knees, limping can occur. Salicylates, gold salts, or glucocorticoids may be used.	Children suffering from JCA may have reduced serum levels of beta-carotene, retinol, and zinc.
Sjögren's syndrome	Dry eyes and dry mouth occur as a result of insufficient production of lacrimal and salivary secretions. Artificial tears and glucocorticoids may be needed. Sjögren's syndrome is relatively common and affects 4 million Americans, mostly women. It is most often related to RA, lupus, scleroderma, or polymyositis. Debilitating pain and fatigue can occur. Sensitivity to sunlight is common; sunscreen is helpful.	Plan meals and use artificial saliva for easier swallowing. Chewing sugar-free gum can stimulate saliva production if any is available. Gel-based saliva substitutes are useful. Sip water often, and avoid caffeinated drinks, which can be dehydrating. Drink water during meals to help with swallowing. Mouth infections are common; use good oral hygiene. With dry mouth or dysphagia, there is a risk for aspiration pneumonia. Weight loss and digestive problems are common.
Felty's syndrome	Felty's syndrome only affects about 1% of RA patients. This is a triad of RA, granulocytopenia, and splenomegaly. Painful, stiff, and swollen joints occur. Infections, leg ulcers, burning eyes, and anemia also can complicate the condition. Sometimes, splenectomy is indicated; drug therapy may be helpful to others.	Fever, weight loss, and brown pigmentation may occur. If immunosuppressive drugs are used, monitor for side effects.
Rheumatoid vasculitis	Rheumatoid vasculitis can be life threatening and usually occurs in patients with severe deforming arthritis and a high titer of rheumatoid factor. A majority have a strong human leukocyte antigen relationship. Vasculitic lesions include rheumatoid nodules, small nail fold infarcts, and purpura. Fatigue, weight loss, fever, organ ischemia, CNS infarctions, myocardial infarction, and peripheral neuropathy can occur.	Corticosteroids are the usual treatment. D-penicillamine and prednisone generally are used.

of medication needed; they also downregulate T-cell proliferation. People with RA who eat 4 oz of fish every day have less morning stiffness, swollen joints, and all-around pain. Fish oil and aspirin are blood thinners, and they should not be taken together for a long time.

Supplements of GLA, as from borage oil, may reduce generation of mediators of inflammation and attenuate symptoms but may cause potentially harmful increases in serum arachidonic acid unless EPA is also used. GLA increases prostaglandin E levels, which increase cyclic adenosine monophosphate (cAMP) levels which, in turn, suppress TNFα synthesis.

Epidemiological studies suggest that the antioxidant potential of dietary carotenoids may protect against the oxidative damage that can result in inflammation (Pattison et al, 2005). Proper antioxidant nutrients provide defense against increased oxidant stress. Supplementation of folate and vitamin B_{12} is needed in patients treated with methotrexate to reduce side effects and to offset elevated plasma homocysteine.

Complications of RA may include osteoporosis and chronic anemia. Calcium and vitamin D reduce the bone loss in patients who take steroids. An iron supplement may prevent anemia, and serum ferritin levels may be low. Patients benefit from a basic dietary supplement.

Higher intakes of meat and total protein and lower intakes of fruit, vegetables, and vitamin C are associated with an increased risk of RA (Choi, 2005). However, dietary factors such as fruit, coffee, long-chain fatty acids, olive oil, vitamins A, E, C, and D, zinc, selenium, and iron need to be studied over a longer time period (Pedersen et al, 2005).

Rheumatoid cachexia, loss of muscle mass and strength and increase in fat mass, is very common in patients with RA and persists even after joint inflammation improves (Roubenoff, 2009). Cardiovascular disease is a concern. Body composition studies are as important as BMI and other traditional assessment measures (Elkan et al, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: B cell, cytokine and inflammation response, and antigen presentation pathways are associated with RA; this confirms the known biological mechanisms for auto-immunity (Ballard et al, 2009).

Clinical/History

- Height
- Weight
- BMI
- Temperature
- Pain and stiffness >30 minutes in the morning or after a long rest
- Food allergies

Lab Work

- RBC
- CRP
- LE prep
- Creat (may be decreased)
- ESR (increases with inflammation)
- ANA
- Rheumatoid factor (RF)
- Antistreptococcal antibody titer
- Immunoglobulins (may be elevated in Sjögren's)
- Ceruloplasmin (may be increased)
- H & H
- Serum ferritin
- Serum B_{12}
- Transferrin
- Serum folate, RBC folate
- Serum copper
- Alb, transthyretin
- Gluc
- BUN
- Ca^{++}, Mg^{++}
- Na^{+}, K^{+}
- Vitamin D_3 status (serum 25-OHD)

INTERVENTION

OBJECTIVES

- Preserve a high level of physical and social functioning to promote good quality of life; reduce the effects of pain and swelling.
- Maintain satisfactory nutritional status; malnutrition and loss of lean body mass are common in this condition. Monitor weight changes.
- Simplify meal preparation.
- Support the immune system. Consume foods rich in antioxidants, such as carotenoids (Pattison et al, 2005), vitamin E, selenium, and vitamin D. A vegetarian diet may have significant benefits.
- Promote adequate growth in children who have RA; stunting can occur from glucocorticoids.
- Promote return of fat-free body mass and improvement in muscle strength.
- Restrict sodium intake, if needed.

SAMPLE NUTRITION CARE PROCESS STEPS

Drug–Nutrient Interaction

Assessment Data: Weight and physical activity histories. Medical history, medications and lab values. DEXA scan results.

Nutrition Diagnosis (PES): Food-Medication interaction (NC-2.3) related to corticosteroid use secondary to diagnosis of RA as evidence by abnormal Ca^{++} level <8.4, DEXA scan at 80% of desirable range for age, perimenopausal status, low calcium and vitamin D intake from diet history.

Intervention: Food-Nutrient Delivery—include extra calcium-rich foods. Education about use of steroid therapy and its impact on nutritional status. Counseling about good sources of calcium and vitamin D from diet and supplements, meal planning and shopping tips, dining out guide, referral to Meals-on-Wheels or other social agencies as appropriate. Coordinate care with nursing and physician to administer calcium and vitamin D supplements at different time than corticosteroids to help increase absorption.

Monitoring and Evaluation: Improvements in dietary and supplemental intake of vitamin D and calcium as shown in food records, lab values, and DEXA scan report.

- Modify patient's diet if hyperlipidemia is present or if there is elevated homocysteine.
- Avoid or correct constipation.

FOOD AND NUTRITION

- Use a high-protein and high-calorie diet if patient is malnourished. Cachexia is common (Marcora et al, 2005).
- A diet that lessens inflammation is useful; olive oil should be used often because it contains oleocanthal, a natural anti-inflammatory agent.
- Eating fatty fish, such as salmon, sardines, mackerel, herring, and tuna, two times per week is suggested. In addition to fatty fish, other good sources of omega-3s include flaxseed, walnuts, soy, canola oils. Try to acquire 3–6 g of omega-3 fatty acids per day for 4 months.
- An uncooked vegan diet may be useful, with berries, fruits, vegetables, roots, nuts, and seeds; see Table 11-2. There is improvement in RA when eating a lactovegetarian, vegan, or Mediterranean diet (Skoldstam et al, 2005).
- Adequate fluid, fiber, vitamins, and minerals are important. Use foods high in beta-carotene, lutein lycopene, selenium, vitamins C and E; choose nutrient-dense foods. Antioxidants such as beta-cryptoxanthin (as from one glass of freshly squeezed orange juice daily) can reduce the risk of developing RA (Pattison et al, 2005).
- Increase vitamin D intakes to decrease the incidence and severity of RA. Provide adequate intake of calcium, magnesium, B-complex vitamins, potassium, and zinc.
- Increase folic acid if methotrexate is used; enhance diet or encourage folic acid supplements.
- Provide meals that are easy to tolerate when the drugs being used cause gastric irritation. Avoid acidic or highly spiced foods if needed.
- With dysphagia, tube feed or use soft/thick, pureed foods as needed.
- Identify and eliminate any food allergens. Individualize the diet accordingly.

Common Drugs Used and Potential Side Effects

- With biologic therapies, such as TNF inhibitors, many patients with RA have seen significant improvement in symptoms, function, and quality of life (Barton et al, 2009). See Table 11-14.

TABLE 11-14 Medications Used in Rheumatoid Arthritis

Medications	Uses/Effects	Side Effects	Monitoring
Analgesics and nonsteroidal anti-inflammatory drugs (NSAIDs)	Analgesics relieve pain; NSAIDs relieve pain and reduce inflammation.	Upset stomach, peptic ulcer, bleeding, renal failure. Use of NSAIDs may increase rate of miscarriage for pregnant women.	For all traditional NSAIDs: avoid drinking alcohol or using blood thinners; avoid if there is sensitivity or allergy to aspirin or similar drugs, kidney or liver disease, heart disease, high blood pressure, asthma, or peptic ulcers.
Acetaminophen		Usually no side effects when taken as directed.	Not to be taken with alcohol or with other products containing acetaminophen. Not to be used for more than 10 days unless directed by a physician.
Aspirin: buffered, plain	Aspirin is used to reduce pain, swelling, and inflammation, allowing patients to move more easily and carry out normal activities. It is generally part of early and ongoing therapy.	Upset stomach; tendency to bruise easily; ulcers, pain, or discomfort; diarrhea; headache; heartburn or indigestion; nausea or vomiting.	Doctor monitoring is needed. Not used for children in whom Reye's syndrome is a risk, but otherwise useful in lessening inflammation.
Traditional NSAIDs: ibuprofen, ketoprofen, naproxen	NSAIDs help relieve pain within hours of administration in dosages available over the counter (available for all three medications). They relieve pain and inflammation in dosages available in prescription form (ibuprofen and ketoprofen). It may take several days to reduce inflammation.	For all traditional NSAIDs: abdominal or stomach cramps, pain, or discomfort; diarrhea; dizziness; drowsiness or light-headedness; headache; heartburn or indigestion; peptic ulcers; nausea or vomiting; possible kidney and liver damage (rare).	For all traditional NSAIDs: avoid drinking alcohol or using blood thinners; avoid if there is sensitivity or allergy to aspirin or similar drugs, kidney or liver disease, heart disease, high blood pressure, asthma, or peptic ulcers.
Cyclo-oxygenase (COX)-2 inhibitor NSAIDs: celecoxib, valdecoxib	COX-2 inhibitors, such as traditional NSAIDs, block COX-2, an enzyme in the body that stimulates an inflammatory response. Unlike traditional NSAIDs, however, they do not block the action of COX-1, an enzyme that protects the stomach lining. Vioxx was withdrawn by FDA.	Stomach irritation, ulceration, and bleeding may occur. Caution is advisable for patients with a history of bleeding or ulcers, decreased renal function, hepatic disease, hypertension, or asthma.	Doctor monitoring for possible allergic responses to valdecoxib and celecoxib is important.

(continued)

TABLE 11-14 Medications Used in Rheumatoid Arthritis *(continued)*

Medications	Uses/Effects	Side Effects	Monitoring
Corticosteroids	These are steroids given by mouth or injection. They are used to relieve inflammation and reduce swelling, redness, itching, and allergic reactions.	Increased appetite, indigestion, nervousness, or restlessness.	For all corticosteroids, advise the doctor if there is presence of the following: fungal infection, history of tuberculosis, underactive thyroid, herpes simplex of the eye, high blood pressure, osteoporosis, or stomach ulcer.
Methylprednisolone, prednisone	These steroids are available in a pill form or as an injection into a joint. Improvements are seen up to 24 hours after administration. There is potential for serious side effects, especially at high doses. They are used for severe flares or when the disease does not respond to NSAIDs and disease-modifying antirheumatic drugs.	Osteoporosis, mood changes, fragile skin, easy bruising, fluid retention, weight gain, muscle weakness, onset or worsening of diabetes, cataracts, increased risk of infection, and hypertension.	Doctor monitoring for continued effectiveness of medication and for side effects is needed.
Disease-modifying antirheumatic drugs (DMARDs)	These are common arthritis medications. They relieve painful, swollen joints and slow joint damage, and several DMARDs may be used over the disease course. They take a few weeks or months to have an effect and may produce significant improvements for many patients. Exactly how they work is still unknown.	Side effects vary with each medicine. DMARDs may increase risk of infection, hair loss, and kidney or liver damage.	Doctor monitoring allows the risk of toxicities to be weighed against the potential benefits of individual medications.
Azathioprine	This drug was first used in higher doses in cancer chemotherapy and organ transplantation. It is used in patients who have not responded to other drugs and in combination therapy.	Cough or hoarseness, fever or chills, loss of appetite, lower back or side pain, nausea or vomiting, painful or difficult urination, unusual tiredness or weakness.	Avoid with allopurinol or kidney or liver disease. May decrease immunity; contact doctor immediately with chills, fever, or a cough. Regular blood and liver function tests are needed.
Cyclosporine	This medication was first used in organ transplantation to prevent rejection. It is used in patients who have not responded to other drugs.	Bleeding, tender, or enlarged gums; high blood pressure; increase in hair growth; kidney problems; trembling and shaking of hands.	Avoid with sensitivity to castor oil (if receiving the drug by injection), liver or kidney disease, active infection, or high blood pressure. Using this drug may make you more susceptible to infection and certain cancers. Do not take live vaccines while on this drug. Avoid St. John's wort and echinacea.
Hydroxychloroquine	It may take several months to notice the benefits of this drug, which include reducing the signs and symptoms of rheumatoid arthritis.	Diarrhea, eye problems (rare), headache, loss of appetite, nausea or vomiting, and stomach cramps or pain.	Doctor monitoring is important, particularly with an allergy to any antimalarial drug or a retinal abnormality.
Gold sodium thiomalate (Ridaura)	This was one of the first DMARDs used to treat rheumatoid arthritis.	Redness or soreness of tongue; swelling or bleeding gums; skin rash or itching; ulcers or sores on lips, mouth, or throat; irritation on tongue. Monitor joint pain 1 or 2 days after injection.	Avoid with lupus, skin rash, kidney disease, or colitis. Periodic urine and blood tests are needed to check for side effects.
Leflunomide	This drug reduces signs and symptoms and slows structural damage to joints caused by arthritis.	Bloody or cloudy urine; congestion in chest; cough; diarrhea; difficult, burning, or painful urination or breathing; fever; hair loss; headache; heartburn; loss of appetite; nausea and/or vomiting; skin rash; stomach pain; sneezing; and sore throat.	Doctor must monitor for the following: active infection, liver disease, known immune deficiency, renal insufficiency, or underlying malignancy. Regular blood tests, including liver function tests, are needed. Leflunomide must not be taken during pregnancy; it may cause birth defects in humans.

(continued)

TABLE 11-14 Medications Used in Rheumatoid Arthritis *(continued)*

Medications	Uses/Effects	Side Effects	Monitoring
Methotrexate (Rheumatrex)	This drug can be taken by mouth or by injection and results in rapid improvement (it usually takes 3–6 weeks to begin working). It is very effective, especially in combination with infliximab or etanercept. It produces more favorable long-term responses compared with DMARDs such as sulfasalazine, gold sodium thiomalate, hydroxychloroquine and may be used in pediatrics.	Abdominal discomfort, chest pain, chills, nausea, mouth sores, painful urination, sore throat, and unusual tiredness or weakness.	Doctor monitoring is important, particularly with an abnormal blood count, liver or lung disease, alcoholism, immune system deficiency, or active infection. Methotrexate must not be taken during pregnancy because it may cause birth defects in humans. Avoid Echinacea. Extra folic acid is needed.
Sulfasalazine	This drug suppresses the immune system.	Abdominal pain, aching joints, diarrhea, headache, sensitivity to sunlight, loss of appetite, nausea or vomiting, and skin rash.	Doctor monitoring is important, particularly with allergy to sulfa drugs or aspirin or with a kidney, liver, or blood disease.
Biological response modifiers	These drugs selectively block cytokines, which play a role in inflammation. Long-term efficacy and safety are uncertain.	Increased risk of infection, especially tuberculosis. Increased risk of pneumonia, and listeriosis (a foodborne illness caused by the bacterium *Listeria monocytogenes*).	Avoid eating undercooked foods (including unpasteurized cheeses, cold cuts, and hot dogs) to reduce listeriosis while taking biological response modifiers.
Tumor necrosis factor inhibitors: etanercept (Enbrel), golimumab (Simponi), infliximab (Remicade), and adalimumab (Humira)	Highly effective for treating patients with an inadequate response to DMARDs. Often prescribed in combination with methotrexate. Etanercept requires subcutaneous injections twice weekly. Infliximab is taken intravenously (IV) during a 2-hour procedure, along with methotrexate. Adalimumab requires injections every 2 weeks.	*Etanercept:* pain or burning in throat, redness, itching, pain, and/or swelling at injection site, runny or stuffy nose. *Infliximab:* abdominal pain, cough, dizziness, fainting, headache, muscle pain, runny nose, shortness of breath, sore throat, vomiting, wheezing. *Adalimumab:* redness, rash, swelling, itching, bruising, sinus infection, headache, nausea. *Golimumab:* respiratory infection, sore throat and nasal congestion.	Doctor monitoring is important, particularly with active infection, exposure to tuberculosis, or a central nervous system disorder. Evaluation for tuberculosis is necessary before treatment begins.
Interleukin-1 inhibitor: anakinra (Kineret)	This medication requires daily injections. Long-term efficacy and safety are uncertain.	Redness, swelling, bruising, or pain at the site of injection; headache; upset stomach; diarrhea; runny nose; and stomach pain.	Doctor monitoring is required.
Selective Costimulation Modulator: Abatacept	Abatacept is given intravenously in a 30-minute infusion. It may be given alone or with DMARDs.	Cough, dizziness, headache, infections, sore throat.	Doctor monitoring is needed.
CD20 Antibody: Rituximab	This medication is for people whose rheumatoid arthritis has not responded to other biologic agents. It is given by two IV infusions 2 weeks apart. It is given with methotrexate.	Abdominal pain, chills/shivering, fever, headache, infection, itching.	Doctor monitoring is needed.
Other medications	Pilocarpine hydrochloride (Salagen) and cevimeline (Evoxac).	Available to treat dry mouth associated with Sjögren's syndrome. They simulate the salivary glands.	

Adapted from: National Institutes of Health. Health topics. Accessed December 19, 2009 at http://www.niams.nih.gov/Health_Info/Rheumatic_Disease/default.asp#ra_16.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician. Some people have tried acupuncture and other alternatives to traditional medicine, but it is important not to neglect regular health care or treatment of serious symptoms. Female patients tend to use alternative treatments for RA more than males; psychosocial intervention may be beneficial.
- With borage oil, concomitant NSAID use may undermine the effects. Borage oil is contraindicated in pregnancy given the teratogenic and labor-inducing effects of prostaglandin E agonists.
- St. John's wort and echinacea should not be used with cyclosporine or methotrexate.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Adoption of a Mediterranean diet confers health benefits in this population because of greater consumption of fruits and vegetables, lower consumption of animal products, and use of olive oil, which modulates immune function (Wahle et al, 2005). Inclusion of omega-3 fatty acids is also important (Berbert et al, 2005); herring, salmon, sardines, tuna, and mackerel are good dietary sources.
- No evidence exists to prove that foods from the nightshade family (potatoes, tomatoes, eggplant, and sweet and hot peppers) should be excluded.
- Encourage nutrient-dense foods. If intake is poor, a vitamin–mineral supplement may be needed. Dietary quinones, phenolics, vitamins, amino acids, isoprenoids, and other compounds in functional foods have become very popular (Losso and Bawadi, 2005).
- Instruct patient about simplified planning and preparation tips. Sandwiches, prepared meals, precut fruits and vegetables are easy to use. Cook double portions and freeze leftovers for another day.
- Discourage quackery and substitute sound health practices.
- Carbohydrate intolerance occurs because of chronic inflammation and use of steroids; planning must reflect individual needs.
- A support group may be helpful for coping.
- Physical therapy and exercise are beneficial for most patients. Strengthening exercises may help improve patient's ability to walk and may decrease joint pain and fatigue. Dynamic exercise is beneficial in RA (Hurkmans et al, 2009).
- Check on bone density; there is a high incidence of osteoporosis when steroids are used.

Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

For More Information

- American Autoimmune Related Diseases Association
  http://www.aarda.org
- American College of Rheumatology
  http://www.rheumatology.org
- Arthritis Foundation
  http://www.arthritis.org
- Felty Syndrome
  http://rarediseases.about.com/od/rarediseasesf/a/121104.htm
- Information on Rheumatoid Arthritis
  http://www.niams.nih.gov/hi/topics/arthritis/rahandout.htm
- Juvenile Rheumatoid Arthritis
  http://www.niams.nih.gov/hi/topics/juvenile_arthritis/juvarthr.htm
- National Institute of Dental and Craniofacial Research–Sjögren's Syndrome
  http://www.nidcr.nih.gov/GrantsAndFunding/See_Funding_Opportunities_Sorted_By/ConceptClearance/CurrentCC/SjogrenSynd.htm
- National Sjögren's Syndrome Association
  http://www.sjogrenssyndrome.org/index.html
- Rheumatoid Vasculitis
  http://vasculitis.med.jhu.edu/typesof/rheumatoid.html
- Sjögren's Syndrome Foundation—Food Tips
  http://www.sjogrens.org/home/about-sjogrens-syndrome/living-with-sjogrens/diet-a-food-tips

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RUPTURED DISC

NUTRITIONAL ACUITY RANKING: LEVEL 1

DEFINITIONS AND BACKGROUND

Determining the cause of back pain is complicated as it is often multifactorial; anatomical abnormalities are common in the spine and may not necessarily translate into clinical symptoms (Sheehan et al, 2010).

A slipped or ruptured disc is called a cervical radiculopathy, herniated intervertebral disc, lumbar radiculopathy, or prolapsed intervertebral disc. In this condition, slipping or prolapse of a cervical or lumbar disc occurs, with neck, shoulder, or low back pain accordingly. Degenerating changes in the disks begin around 30 years of age. Overweight and obesity increase the risk of low back pain and the need for medical attention (Shiri et al, 2010).

With **lumbar radiculopathy**, ambulation may be painful, and limping can occur. Muscular weakness, severe back pain that radiates to buttocks or legs and feet, pain that worsens with coughing or laughing, tingling or numbness in legs or feet, and muscle contractions or spasms may also result. With **cervical radiculopathy**, neck pain in back and sides is deep; pain may radiate to shoulders, upper arms, or forearms and worsens with coughing or laughing. Spasm of neck muscles and pain that worsens at night may occur.

A laminectomy surgically removes the lamina of a vertebra. Percutaneous automated discectomy (PAD) surgery can be performed in some cases; this surgery breaks up the disc and removes fragments. There is no convincing medical evidence to support routine use of lumbar fusion, but it may be useful in patients with associated spinal deformity, instability, or associated chronic low-back pain (Resnick et al, 2005). Surgery for radiculopathy with herniated lumbar disc and symptomatic spinal stenosis is associated with short-term benefits compared to nonsurgical therapy, though benefits diminish with long-term follow-up in some trials (Chou et al, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: This condition is acquired and not genetic.

Clinical/History

Height
Weight
BMI
I & O
BP
Constipation
Edema
MRI or computed tomography (CT) scan
Myelography
Discography
Spinal or neck x-rays
Nerve conduction velocity test

Lab Work

H & H
Ca^{++}, Mg^{++}
Na^{+}, K^{+}
Alb, transthyretin
BUN, Creat
Alk phos
Gluc
Vitamin D_3 status (serum 25-OHD)

SAMPLE NUTRITION CARE PROCESS STEPS

Abnormal GI Function

Assessment Data: Weight and physical activity histories. Medical history and medications.

Nutrition Diagnosis (PES): Abnormal GI function related to constipation and infrequent stooling pattern as evidenced by GI distress and evacuation every 3–4 days.

Intervention: Food-nutrient delivery—Assure intake of adequate fluid and fiber at all meals. Education—Discuss tips for alleviating constipation through use of specific foods rich in fiber (fruits, vegetables, whole grains, beans and legumes). Coordinate care—Work with nursing and physicians to determine if any medications that cause constipation can be changed, or if some type of laxative can be added.

Monitoring and Evaluation: Improvement in bowel habits; alleviation of constipation.

INTERVENTION

OBJECTIVES

- Maintain adequate rest and activity levels, as assigned by physician.
- Prevent weight gain from decreased activity.
- Encourage adequate hydration.
- Prevent constipation and straining.
- Assist with feeding, if patient is in traction.
- Relieve pain and promote healing.

FOOD AND NUTRITION

- A regular diet generally is sufficient. For some, a strict energy-controlled diet may be beneficial to promote weight loss.
- Increased fluid and fiber intake can be helpful to reduce constipation. Fresh fruits and vegetables, dried beans, legumes, whole grains, bran, and other foods may be needed.

Common Drugs Used and Potential Side Effects

- Anti-inflammatory drugs may be used. NSAIDs are used for long-term pain control, but narcotics may be given if the pain does not respond. Nausea, GI distress, and anorexia may result. Follow directions regarding when to take (e.g., before or after meals).
- Analgesics may be helpful to relieve pain. Chronic use of aspirin may cause GI bleeding.
- Muscle relaxants may be ordered. GI distress or nausea can occur.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Instruct patient regarding effective methods of relieving constipation.
- Discuss role of nutrition and exercise in health maintenance. Weight loss may be needed.
- After surgery, the role of nutrition in wound healing should be discussed.

Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

For More Information

- Herniated Disk
  http://www.nlm.nih.gov/medlineplus/ency/article/000442.htm
- Lumbar Radiculopathy
  https://health.google.com/health/ref/Herniated+nucleus+pulposus

RUPTURED INTERVERTEBRAL DISC—CITED REFERENCES

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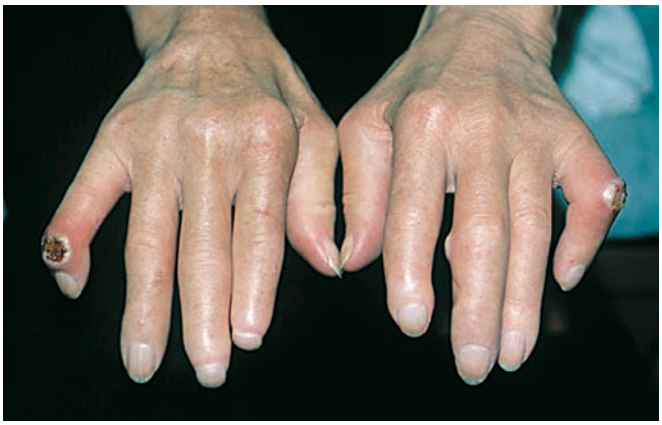
SCLERODERMA (SYSTEMIC SCLEROSIS)

NUTRITIONAL ACUITY RANKING: LEVEL 1–2



Adapted from: Goodheart HP, MD. *Goodheart's Photoguide of Common Skin Disorders,* 2nd ed. Philadelphia: Lippincott Williams & Wilkins, 2003.

DEFINITIONS AND BACKGROUND

Scleroderma is a chronic disease characterized by fibrosis and autoantibodies. Approximately 2% of the population in Europe and North America suffers from disorders such as scleroderma (Chen and von Mikecz, 2005). Genetic, immunological, hormonal, and environmental factors are considered to be triggers (Molina and Shoenfeld, 2005).

The diffuse form affects a large area of the skin and several organs; it is also called **systemic sclerosis** (SSc). In SSc, pathological deposition of fibrous connective tissue in the skin and visceral organs occurs. Fibrosis involves an increase of hydroxylysine aldehyde collagen cross-linkages as well as an increase in inflammatory cytokines (Brinckmann et al, 2005). The GI tract is affected, and Raynaud's syndrome (ischemia of fingers) is common.

The limited form of scleroderma affects the skin and sometimes the lungs. The CREST syndrome (**limited cutaneous sclerosis**) is less severe than SSc and causes less internal organ damage. Calcium deposits, Raynaud's phenomenon, esophageal dysfunction, skin damage on fingers, and telangiectasia form the acronym for CREST.

As the disease progresses, large areas of the skin or just the fingers (sclerodactyly) may be affected. Skin on the face tightens and causes a mask-like appearance. Spider veins (telangiectasia) occur on the fingers, chest, face, lips, or tongue. Calcium deposits can occur on the fingers or other bony areas; sores or contractures may result from the scarring. Scarring of the esophagus may be especially detrimental, causing blockage or even cancer. Lungs can be affected, leading to shortness of breath with exercise.

Neurological involvement consists of epilepsy, central nervous system vasculitis, peripheral neuropathy, vascular malformations, headache, and neuroimaging abnormalities; ocular manifestations include uveitis, xerophthalmia, glaucoma, and papilledema (Zulian et al, 2005). SSc is characterized by vasculopathy, inflammation, vasospasm, microvascular involvement is common; an increased prevalence of distal peripheral artery disease in the digits has been found (Hetterna et al, 2008).

Scleroderma renal crisis (SRC) occurs in 5–10% of SSc patients, who may present with an abrupt onset of hypertension, acute renal failure, headaches, fevers, malaise, hypertensive retinopathy, encephalopathy, and pulmonary edema (Denton et al, 2009). Multiple organ system dysfunction may occur. Pulmonary hypertension, heart failure, and respiratory failure cause serious morbidity and mortality. There is no known cure, and SSc can be fatal.

There seems to be an increased prevalence of celiac disease in patients with scleroderma (Rosato et al, 2009). Both disorders require careful management. Current therapies for scleroderma target the immune system, with the goal of reducing inflammation, ischemic injury to the involved organs, and secondary tissue injury and fibrosis (Henness and Wigley, 2007).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Genetic factors contribute to disease susceptibility; transforming growth factor-ss is a cytokine that contributes to fibroblast activation, collagen overproduction, and pathological tissue fibrosis (Varga, 2008). T-cell polarization is implicated in the lung disease of SSc (Boin et al, 2008).

Clinical/History

- Height
- Weight
- BMI
- I & O
- Weight loss
- Fever?
- BP
- Thickening, swelling of the ends of the fingers
- Dysphagia
- Heartburn
- Fibrosis of salivary and lacrimal glands
- Abdominal pain, flatulence
- Nausea, vomiting
- Diarrhea, constipation
- Skinfold measurements

Lab Work

- ANA (high)
- RF (high)
- LDL Cholesterol (elevated)
- Trig (may be low)
- Anti-tTG antibody
- Serum folate
- H & H
- Serum B_{12}
- Gluc
- Prothrombin time (PT)
- Alb, transthyretin
- CRP
- GFR
- BUN, Creat
- Homocysteine
- Ca^{++}, Mg^{++}
- Na^{+}, K^{+}
- Alk phos
- Vitamin D_3 status (serum 25-OHD)
- Fecal fat test, hydrogen breath test for malabsorption

SAMPLE NUTRITION CARE PROCESS STEPS

Difficulty Swallowing

Assessment Data: Weight, medical history, medications. Low salivation and difficulty swallowing.

Nutrition Diagnosis (PES): Difficulty swallowing related to low saliva production as evidenced by fibrosis, inability to swallow solids.

Intervention: Food-Nutrient Delivery—Alteration in food choices to liquefy meals and make them easier to swallow. Educate about the use of saliva substitutes, more fluids, altered food choices as needed. Counseling about when to request changes, such as tube feeding.

Monitoring and Evaluation: Improvement in swallowing and tolerance for meals. No weight loss.

INTERVENTION

OBJECTIVES

- Prevent or correct protein-energy malnutrition and nutrient deficiencies.
- Correct xerostomia where present; decreased saliva, dysphagia, and difficulty in chewing will result.
- Monitor dysphagia with esophageal involvement; alter method of feeding as needed.
- Counteract vitamin B_{12} and fat maldigestion and absorption, which may be common.
- Monitor hypomotility and gastroparesis; alter fiber intake as appropriate. For many patients, nutritional support and relief of symptoms remain the primary management goals.
- Improve quality of life and reduce fatigue; allow return to work or maintenance of energy levels.

FOOD AND NUTRITION

- Diets high in energy (30–40 kcal/kg) and adequate to high in protein are often necessary. A soft diet with moistened foods and extra fluids is useful. Add fiber if constipation is a problem (such as adding crushed bran to hot cereal).
- Small, frequent feedings may be needed. Tube feed if patient is dysphagic or has obstruction.
- Use parenteral nutrition if GI tract is highly affected, with intractable diarrhea and severe malabsorption.
- If there is celiac sensitivity, omit gluten from the diet.
- Reduce lactose if intolerance occurs. Extra calcium may be needed if lactose is not tolerated orally.
- Give supplements of fat- and water-soluble vitamins.
- With hypertension and multiple organ system dysfunction, reduced sodium or fluid restriction may be needed.

Common Drugs Used and Potential Side Effects

- Topical or systemic corticosteroids, vitamin D analogs (calcitriol and calcipotriol), photochemotherapy, laser therapy, antimalarials, phenytoin, D-penicillamine, and colchicine all have varying degrees of success. Topical tacrolimus cream is an immunosuppressive antibiotic.
- Interstitial lung disease can be treated with cyclophosphamide, vascular disease of the lungs and digits with endothelin receptor antagonists, and general symptoms with phosphodiesterase inhibitor sildenafil or prostacyclins (Henness and Wigley, 2007).
- Early, aggressive treatment with angiotensin-converting enzyme inhibitors helps with a renal crisis (Denton et al, 2009).
- Anti-inflammatory agents, such as steroids, are often used in SSc. Monitor for nitrogen and calcium losses, altered electrolyte levels, and elevated glucose levels. Correct diet accordingly.
- Antihypertensives usually are needed; monitor BP results. Potassium supplements may or may not be required; determine need according to medication selected.
- Trental (pentoxifylline) is used for Raynaud's syndrome to improve circulation. Anorexia or GI distress may result.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with physician.
- For Raynaud's disease, evening primrose, gingko, mustard, garlic, borage, and red pepper have been suggested, but there are no clinical trials that prove effectiveness.
- CAM is frequently used to treat stress-related disorders such as scleroderma; some merit can be noted (Hui et al, 2009).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Artificial saliva (Xero-Lube) or lemon glycerine may be useful.
- Chew sugarless gum.
- If eating orally, adequate chewing time will be required.
- Consume adequate fluids. Choose moist foods or foods with sauces/gravies.
- For heartburn, keep head elevated after meals; decrease or limit intake of chocolate, caffeine, fatty foods, alcohol, citrus, and tomatoes.
- Physical therapy and exercise may help maintain muscle strength but cannot totally prevent joints from locking into stiffened positions.

Patient Education—Food Safety

If enteral or parenteral nutrition is used, careful sanitation and handling techniques must be taught and used.

For More Information

- Scleroderma Foundation
  http://www.scleroderma.org/
- Scleroderma Research Foundation
  http://www.srfcure.org

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SECTION 12

Hematology: Anemias and Blood Disorders

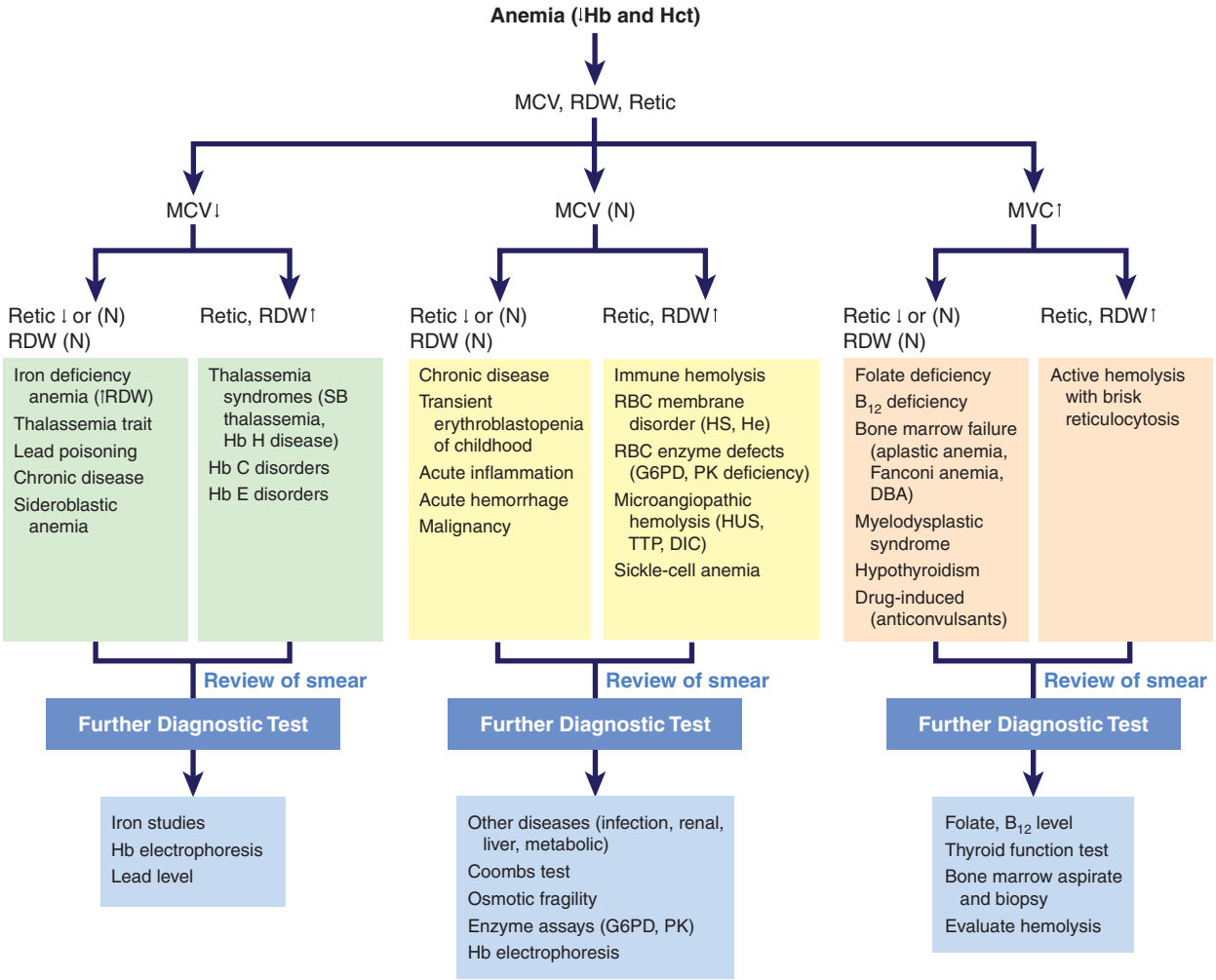
CHIEF ASSESSMENT FACTORS

- Anorexia
- Beefy, Red Tongue or Magenta Tongue; Other Signs of Nutrient Deficiencies
- Blood Type
- Bruising
- Concurrent Asthma, Cancer, Cerebrovascular Disease, Hemorrhage, Myocardial Infarction, Renal Disease
- Dietary Habits: Use of Heme and Nonheme Iron, Vitamin and Mineral Deficiencies, Protein Intake, Vegan Lifestyle
- Exposure to Lead Paint, Other Toxins
- Family History of Allergies, Anemias, Cancer, Immune Disorders, and Leukemias
- Fatigue
- History of Alcohol and Nicotine Use
- Infections, Sepsis
- Lymphadenopathy
- Medication Use (Prescriptions, Over-the-Counter) and Use of Herbal or Botanical Medications
- Occupational or Environmental Exposure to Toxic Substances
- Previous Blood Disorder, Bleeding Tendencies, Blood Transfusion, or Exposure to Radiation
- Surgery, Especially Gastric, Hepatic, or Renal

GENERAL INFORMATION ABOUT ANEMIAS

Blood contains plasma and cells. Plasma is clear and yellow and makes up 55% of blood. It contains proteins, nutrients, hormones, and electrolytes. White cells, red blood cells (RBCs), and platelets make up the remaining 45% of blood. The white cells fight infection, platelets are necessary for blood clotting, and RBCs carry oxygen throughout the body. Hepcidin, the main iron regulatory hormone, is made primarily in hepatocytes in response to liver iron levels, inflammation, hypoxia, and anemia (Munoz et al, 2009). Erythropoietin is the hormone that stimulates RBC production. The erythrocyte life span is 120 days, after which the cells are destroyed by the spleen. Anemias are a set of hematological disorders with a reduced number of RBCs, reduced amount of hemoglobin (Hgb), or reduced number of volume-packed RBCs (hematocrit [Hct]). Excessive bleeding, decreased RBC production, and increased RBC destruction may lead to anemias. The main consequences of these disorders include hypoxia and decreased oxygen-carrying capacity. Overall, anemias affect over 3.4 million people in the United States. Chronic disease and iron deficiency are the most common causes. Other causes of anemias include peptic ulcers, inflammation, infection, cancers, gastritis, liver disease, renal disease, hypothyroidism, history of blood transfusions, blood coagulation disorders, and poor diet. Generally, Hgb, serum iron, TIBC or UIBC, and serum ferritin will establish iron status. In conditions due to blood cell production or cancers, other tests or procedures are needed to determine the cause for abnormal iron levels. These may include a complete blood count (CBC) with differential, zinc protoporphyrin (ZPP) immunological tests, hormone tests, reticulocyte count, C-reactive protein (CRP), sedimentation rate (SED rate), B_{12} or folate levels, genetic testing, tissue biopsy, MRI, ultrasound, bone marrow aspiration, blood smears, urine or fecal sampling, scopes (endoscope or colonoscopy), and tests associated with specific diseases or conditions that can have anemia or iron overload.

Anemias can be encountered with generalized or specific nutritional deficiencies (Table 12-1). The nutritional anemias are caused by deficits, but not all anemias require nutritional intervention. Use caution when evaluating single laboratory results; most anemias have a specific profile. For example, iron and copper participate in one-electron exchange reactions; the same property that makes them essential also generates free radicals that can be seriously deleterious to cells (Arredondo and Nunez, 2005). Table 12-2 provides some key definitions that are used to describe anemias.



Red cell distribution width (RDW), a measure of heterogeneity in the size of circulating erythrocytes, is associated with some chronic diseases and predicts mortality.

TABLE 12-1 Nutritional Factors in Blood Formation

Protein
Iron
Vitamin C
Vitamin E
Folic acid
Vitamin B_6
Vitamin B_{12}
Vitamin K
Copper
Riboflavin (minute amounts)

Inadequate intakes of many nutrients are now known to contribute to several chronic diseases. Folic acid and vitamin B_{12} are among the key nutrients involved. Vitamin B_{12} deficiency, iron or folate deficiency, chronic gastrointestinal (GI) bleeding, and myelodysplastic syndrome are causes of anemia in the elderly. Anemias are more common in the hospitalized elderly than among those who live independently.

Iron is an essential micronutrient as it is required for adequate erythropoietic function, oxidative metabolism, and cellular immune responses (Munoz et al, 2009). Yet it is one of the most frequently lacking nutrients in both developing and developed countries. Iron-deficiency anemia (IDA) affects about 25% of infants worldwide. Adults, especially menstruating women, are also susceptible. Laboratory tests provide evidence of iron depletion in the body, or reflect iron-deficient red cell production; the appropriate combination of these laboratory tests help establish a correct diagnosis of ID status and anemia (Munoz et al, 2009). Table 12-3 lists some relevant tests and Table 12-4 lists common signs and symptoms of anemias.

Up to 10% of young women in developed countries are iron deficient. The problem is not easily resolved by adopting

TABLE 12-2 Definitions

Acute anemia	Precipitous drop in the RBC population due to hemolysis or acute hemorrhage
Anemia	Reduction in the number of circulating RBCs, the amount of hemoglobin, or the volume of packed RBCs (Hct)
Chronic anemia	Anemia that lasts 2 months or longer
Hypochromia	Blood condition in which there is a low level of hemoglobin and color
Hyperchromia	Blood that is excessively pigmented
Microcytic anemia	Usually caused by or resulting in iron deficiency; RBCs are small in size
Macrocytic anemia	Folic acid or vitamin B_{12} insufficiency; RBCs are larger than usual
Megaloblastic anemia	Anemias in which there are large, nucleated abnormal RBCs that are irregular in shape, from pernicious anemia or use of certain immunosuppressive or antitumor drugs
Normocytic anemias	Inhibition of marrow by infection or chronic disease; RBCs are of usual size
Normochromia	Blood with a normal color and level of hemoglobin

TABLE 12-3 Iron Tests

Hemoglobin	Reflects the level of functional iron. Low levels can indicate iron-deficiency anemia or ACD. Hemoglobin values help determine if anemia is present and if a blood donation can be done
Serum ferritin	Measures the amount of iron in storage. One ferritin molecule can hold as many as 4500 iron atoms. Ferritin can be elevated when a person has an infection or inflammatory condition
Serum iron (Fe)	Free or unbound iron in serum. Ideal range is 40–180 μg/dL. Measurement is best done fasting because serum iron is sensitive to foods or supplements recently consumed, time of day, and menstruation
Transferrin	Iron-binding and transport protein that can bind to and transport two molecules of iron. Transferrin carries iron through the bloodstream to the bone marrow, the liver, and ferritin. Transferrin is no longer measured directly by most physicians, instead TIBC is used
TIBC	Demonstrates the iron-binding ability of transferrin. Serum iron divided by TIBC × 100% provides the transferrin-iron saturation percentage (Tsat%), which is also called iron saturation. Normally, Tsat% is 25–35%. Higher numbers are suggestive of iron loading. Lower numbers are suggestive of iron-deficiency anemia

From: Iron Overload Disease Association, http://www.irondisorders.org/Forms/irontests.pdf, accessed December 21, 2009.

TABLE 12-4 General Signs and Symptoms of Anemia

- Anorexia
- Ascites
- Bowel irregularity
- Chest pain, palpitations
- Coldness of extremities
- Dizziness, especially postural
- Dyspnea, especially exercise intolerance
- Decreased libido or impotence
- Decreased urine output
- Difficulty sleeping or concentrating
- Fatigue, weakness, irritability
- Headache
- Mental status changes
- Pale conjunctiva
- Tachycardia
- Thirst
- Tinnitus
- Vertigo, syncope

Comparing Disorders of Iron

IRON PANEL TESTS	Serum Iron	Serum Ferritin	Transferrin Iron Saturation Percentage	Total Iron Binding Capacity (TIBC)	Transferrin	Hemoglobin
Hemochromatosis	↑	↑	↑	↓	↓	NORMAL
Iron Deficiency Anemia	↓	↓	↓	↑	↑	↓
Sideroblastic Anemia	↑	↑	↑	↓	↓	↓
Thalassemia	↑	↑	↑	↓	↓	↓
Porphyria Cutanea Tarda (PCT)	↑	↑	↑	↓	↓	NORMAL
Anemia of Chronic Disease (ACD)	↓	↑ OR NORMAL	↓	↓	↓	↓
African Siderosis (AS)	↑	↑	↑	↓	↓	NORMAL
Vitamin B_{12} Deficiency (pernicious anemia)	↑ OR NORMAL	↑ OR NORMAL	↑ OR NORMAL	↓ OR NORMAL	↓ OR NORMAL	↓

an iron-rich diet because absorption varies greatly. Although the absorption of dietary iron (1–2 mg/d) is regulated tightly, it is balanced with losses (Munoz et al, 2009). Dietary heme iron is important and more readily absorbed than nonheme iron derived from vegetables and grains. Most heme is absorbed in the proximal intestine.

Inherited Hgb disorders, such as sickle cell anemia and thalassemia, can be attributed to the effects of natural selection. In environments in which malaria was common, carriers were protected and survived to have more children.

For More Information

- National Anemia Action Center
  http://www.anemia.org/
- National Heart, Lung, and Blood Institute Information Center
  http://www.nhlbi.nih.gov/about/dbdr/

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ANEMIAS

ANEMIA OF CHRONIC DISEASE

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Anemia of chronic disease (ACD) is the condition of impaired iron utilization where functional iron (Hgb) is low, but tissue iron (such as in storage) is normal or high. ACD is known as anemia of inflammation (AI). Low Hgb, low total iron-binding capacity (TIBC), and low transferrin with elevated ferritin are identified. ACD is the second most common type of anemia after anemia of iron deficiency; it results in increased morbidity and mortality of the underlying disease (Agarwal and Prchl, 2009). ACD is seen in a wide range of chronic autoimmune, cancerous or leukemic,

inflammatory, and infectious disease conditions. In rheumatoid arthritis, ACD and iron-deficiency anemia coexist, resulting from GI bleeding due to the use of many drugs. ACD is also found in approximately 50% of patients with lupus (Giannouli et al, 2006). In aging and heart failure, chronic anemia is common.

Hepcidin is the iron regulatory peptide that is synthesized in the liver to suppress iron absorption and utilization. Synthesis is suppressed by anemia, hypoxia, and erythropoiesis, and induced by inflammatory cytokines such as interleukin-6 (Matsumoto et al, 2009). ACD is characterized by macrophage iron retention induced by cytokines and hepcidin. Excess hepcidin causes proteolysis of the cellular iron exporter, ferroportin, trapping iron in macrophages, and iron-absorbing enterocytes (Ganz and Nemeth, 2009). Because circulating hepcidin levels affect iron traffic, its determination may aid to differentiate between ACD and iron-deficiency anemia to select an appropriate therapy (Theurl et al, 2009).

Hgb improvement is an independent predictor of quality of life improvement in anemic patients, yet supplementation with iron for those with ACD can be harmful and even result in death. Levels of erythropoietin are reduced in ACD. The genetically engineered form (epoetin) can correct anemia caused by cancer in about 50–60% of patients and may improve survival in HIV infection.

Epoetin can eliminate the need for transfusions but is very expensive.

Successful treatment of the underlying disease improves ACD, but if not possible, treatment with erythropoietic agents (ESAs), supplemented with iron if necessary, is helpful in many cases (Agarwal and Prchl, 2009). ESAs are safe and may forestall some of the target-organ damage (Nurko, 2006).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Modifications of hepcidin gene expression suggest a key role for hepcidin in iron homeostasis. HAMP is the gene that encodes hepcidin.

Clinical/History

- Height
- Weight
- Body mass index (BMI)
- Diet history
- Intake and output (I & O)
- Blood pressure (BP)
- Fatigue and weakness
- Headache, irritability

Lab Work

- Complete blood count (CBC)
- RBC count
- Serum hepcidin level
- Total iron-binding capacity (TIBC) (low)
- Hgb and Hct (H & H) (high)
- Serum ferritin (high)
- Serum Fe
- Glucose (Gluc)
- Transferrin (low)
- Albumin (Alb)
- C-reactive protein (CRP)

SAMPLE NUTRITION CARE PROCESS STEPS

Abnormal Nutritional Laboratory Values

Assessment Data: Weight, BMI normal. Hgb is low at 10; ferritin is normal. GI bleeding and pain.

Nutrition Diagnoses (PES): Abnormal nutritional laboratory values related to chronic anemia and high doses of medications for lupus as evidenced by low Hgb, normal serum ferritin, and GI bleeding.

Interventions: Food-nutrient delivery—encourage nutrient-dense foods and frequent snacks; avoid fasting. Education about low-calorie, nutrient-dense foods and timing with medications. Counseling about timing of medications with food to reduce GI bleeding. Coordinate care with medical and nursing teams to review medications and determine which, if any, could be changed to reduce impact on GI tract.

Monitoring and Evaluation: No additional GI bleeding or distress. Resolution or improvement of anemia. Hgb closer to normal.

INTERVENTION

OBJECTIVES

- Prevent infections or sepsis.
- Reduce fever and excessive inflammation.
- Lessen bleeding tendencies and hemorrhages.
- Ensure adequate periods of rest. Simplify meal planning if needed.
- Prepare for bone marrow transplantation, if needed.
- Prevent further complications and decline in organ functioning.

FOOD AND NUTRITION

- Provide a balanced diet that is easily prepared, with six small feedings.
- Provide extra fluid unless contraindicated.
- If steroids are used, limiting sodium intake may be needed.
- Correct iron overload where present.

Common Drugs Used and Potential Side Effects

- Genetically engineered erythropoietin (epoetin) is often used; given weekly, it can improve quality of life and levels of energy.
- Avoid iron supplements in this condition; they can be harmful and even result in death.
- Corticosteroids may be used. Watch side effects of chronic use such as elevated serum sodium levels, decreased potassium and calcium levels, and negative nitrogen balance. Hyperglycemia may occur; alter diet accordingly.
- Antibiotics may be required when infections are present. Monitor for GI distress and other effects.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Alpha tocopherol and *N*-acetylcysteine have been recommended, but more controlled studies are needed.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss needs of the patient, which are specific for signs, symptoms, and side effects of any medications.
- Discuss nutritious meal planning. If patient has diabetes, heart failure, or cirrhosis, counsel specifically for those issues.
- Correcting anemia in heart failure patients improves quality of life and exercise capacity in both men and women (Fox and Jorde, 2005). Once improvement is noted, activity levels can be increased.
- Counsel about reduction of iron overload if present. For example, iron-fortified cereals and oral supplements containing iron should be avoided. Increase grains, fruits, vegetables, cheese, and dairy foods; use fewer heme iron sources.
- Being female is often independently associated with lower Hgb, so assess using sex-specific laboratory values (Fox and Jorde, 2005).

Patient Education—Food Safety

If tube feeding or central parenteral nutrition (CPN) is needed, careful handwashing procedures should be followed.

For More Information

- Anemia of Chronic Disease
  http://www.emedicine.com/emerg/topic734.htm

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ANEMIAS IN NEONATES

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Anemia of prematurity (AOP) is a normocytic, normochromic anemia that presents with very low Hgb and low erythropoietin level. Inadequate RBC production may occur, and the average life span of these cells is about 35–50 days (compared with 120 days for adults). Three causes of AOP include inadequate RBC production, shortened RBC life span, or blood loss. AOP is very common among those born prematurely, where prevalence may be as high as 50% in those born before 32 weeks of gestation. It is also especially common in those born with weight below 1500 g (Haiden et al, 2006).

Hemolytic disease of the newborn (erythroblastosis fetalis) is a condition in which RBCs are broken down or destroyed more rapidly than normal, causing hyperbilirubinemia, anemia, or death; hemolytic disease of the newborn may occur in Rh-positive babies born to Rh-negative mothers (Merck Manual, 2009). Critically ill, extremely premature infants develop anemia because of intensive laboratory blood testing and undergo multiple RBC transfusions in the early weeks of life (Widness et al, 2005). Poor weight gain, apnea and tachypnea, lethargy, tachycardia, and pallor are symptoms.

Reducing anemia in infants may be a preventive measure to lower disease burden from infectious disease in this vulnerable population (Levy et al, 2005). Nutritional deficiencies of vitamin E, vitamin B_{12}, and folate exaggerate the degree of anemia. Vitamin E supplementation, however, when given to preterm infants, does not reduce the severity of this anemia. Administration of vitamin B_{12} and folate with erythropoietin and iron may enhance erythropoietin-induced erythropoiesis more than erythropoietin alone (Haiden et al, 2006).

When detected early in pregnancy, iron-deficiency anemia is associated with a greater risk of preterm delivery (Scholl, 2005). However, it is important not to overdo iron intake. High levels of Hgb, Hct, and ferritin are associated with an increased risk of fetal growth restriction, preterm delivery, and preeclampsia (Scholl, 2005).

Diamond-Blackfan anemia (DBA) (erythrogenesis imperfecta or congenital hypoplastic anemia) is a rare blood disorder characterized by deficiency of RBCs at birth. Other symptoms including slow growth, abnormal weakness, fatigue, pallor, characteristic facial abnormalities, protruding shoulder blades, abnormal shortening of the neck due to fusion of cervical vertebrae, hand deformities, and congenital heart defects. DBA may be inherited as either an autosomal dominant or recessive genetic trait, where the body's bone marrow produces little or no RBCs. A genetic error on chromosome

19 is associated with about 25% of cases, and there is a family history of the disorder in 10–20% of cases.

DBA affects approximately 600–700 million people worldwide but can be difficult to identify. The symptoms may also vary greatly, from very mild to severe and life threatening. DBA is usually diagnosed within the first 2 years of life, sometimes even at birth, on the basis of symptoms. The diagnosis of this anemia might not be recognized right away, however, because it is rare.

The first line of treatment for DBA is prednisone. About 70% of children with DBA will respond to this lifelong treatment, where the medication stimulates the production of more RBCs. If steroids do not work, the next treatment is blood transfusions. Regular blood transfusions will provide RBCs but can lead to iron overloading. Normally, the body uses iron when making new RBCs, but since the person with DBA is not making many cells, the iron builds up. The person then needs to take medication that takes the excess iron out of the body. The only cure available for DBA is bone marrow transplantation. Stem-cell transplantation with human leukocyte antigen (HLA)-matched stem cells has been used for DBA (Kuliev et al, 2005).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Anemia in a newborn may be caused by a genetic condition such as congenital hypoplastic anemia. In DBA, a genetic alteration on chromosome 19 has been noted.

Clinical/History

- Height
- Weight
- BMI
- Diet history
- Slow growth in child (low height–weight percentiles)
- BP
- Weakness
- Fatigue and pallor
- I & O

Lab Work

- CBC
- RBC count
- H & H (>2 standard deviations below mean for age)
- Serum Fe and ferritin
- Gluc
- Alb
- CRP
- Serum folic acid and B_{12}
- K^+, Na^+
- Calcium

INTERVENTION

OBJECTIVES

- Provide improved oxygenation for tissues.
- Prevent infections or sepsis. Reduce fevers or excessive inflammation.
- Control hyperglycemia or other side effects of treatments.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Vitamin-Mineral Intake

Assessment Data: Low birth weight (1450 g), poor suck. Lab work shows low Hgb and serum B_{12}; diagnosis of anemia. Pallor, listlessness, and tachycardia.

Nutrition Diagnoses (PES): Inadequate vitamin and mineral intake related to poor suck and low birth weight as evidenced by lab work with low Hgb and serum B_{12}. Inadequate weight gain.

Interventions: Nutrient delivery—specialized infant formula with added calories and administration of vitamin B_{12}, folate, erythropoietin and iron.

Monitoring and Evaluation: Improvement in heart rate, growth, and pallor. Resolution of anemia.

- Prevent further complications.
- Support growth.

FOOD AND NUTRITION

- Provide an appropriate formula that is easily prepared, with small feedings given frequently.
- Provide extra fluid unless contraindicated.

Common Drugs Used and Potential Side Effects

- If corticosteroids are used, watch side effects of chronic use such as elevated serum sodium levels, decreased potassium and calcium levels, and negative nitrogen balance. Hyperglycemia may occur; alter diet accordingly. Besides diabetes, glaucoma, bone weakening, and high blood pressure can occur, and the medication may suddenly stop working for that person at any point in time.
- Antibiotics may be required when infections are present. Monitor for gastrointestinal distress and other side effects.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss needs of the patient, which are specific for signs and symptoms and for side effects of any medications.
- Discuss nutritious meal planning.
- If patient has diabetes, counsel specifically for nutritional management.
- Activity levels must be restricted to avoid accidents or falls that could promote bleeding.
- Referral to the Women, Infants, and Children (WIC) Program can be beneficial. WIC programs are helpful in

improving Hgb concentration among young children (Altucher et al, 2005). Age-specific values should be used to assess progress:

Age-Specific Values for Hemoglobin and Hematocrit

Age	Hb (g/dL)	Hct (%)
28-week gestation	14.5	45
32-week gestation	15	47
Term	16.5	51
1–3 days	18.5	56
2 weeks	16.6	53

Source: Merck Manual, http://www.merck.com/mmpe/print/sec19/ch273/ch273b.html, accessed December 22, 2009.

Patient Education—Food Safety

If tube feeding or CPN is needed, careful handwashing procedures should be followed.

For More Information

- Anemia of Prematurity
  http://www.emedicine.com/ped/topic2629.htm
- Diamond Blackfan Anemia
  http://www.diamondblackfan.org.uk/
- Perinatal Anemia
  http://www.merck.com/mmpe/sec19/ch273/ch273b.html

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ANEMIA OF RENAL DISEASES

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Anemia of renal disease occurs in both acute and chronic renal disease. This type of anemia is often normochromic, normocytic, and sometimes microcytic. When the kidneys become diseased, scar tissue forms and prevents the cells that make erythropoietin from functioning. The buildup of uremic toxins and decreased erythropoietin production adversely affects erythropoiesis. The accumulation of toxic metabolites, which are normally excreted by the kidneys, shortens the life span of circulating RBCs. Management is complicated by a vicious circle of cardiorenal anemia syndrome in which CKD, heart failure, and anemia exacerbate each other (Besarab et al, 2009).

There is an inverse relationship between blood urea nitrogen (BUN) levels and RBC life span, but there is also diminished renal production of erythropoietin. If no cause for anemia other than chronic kidney disease is detected on the basis of the workup and the serum creatinine is ≥2 mg/dL, anemia is most likely due to erythropoietin deficiency; measurement of serum erythropoietin levels is not needed.

Anemia usually starts during the third stage of renal disease, when glomerular filtration rate (GFR) is below 60 cc/minute but before dialysis has started. Short daily hemodialysis and daily home nocturnal hemodialysis can control blood pressure and manage anemia in this population (Pierratos, 2005). Correction of anemia appears to improve cardiac function and quality of life without a greater risk for adverse events (Besarab et al, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: In disorders such as type-2 diabetes, chronic kidney disease is common. Anemia may be present and cause fatigue and difficulty with daily activities such as climbing stairs.

Clinical/History	Lab Work	
Height	CBC and RBC count	[a]Transferrin saturation (serum iron × 100 ÷ TIBC) <20%?
Weight	Serum Fe	[b]Reticulocyte hemoglobin content (CHr)
BMI	Hgb (may be <12 g/dL)	Serum soluble transferrin receptor (sTfR)—elevated?
Diet history	Hct (often <33%)	
BP	Serum ferritin: (100 absolute deficiency; overload >800 ng/dL	
I & O	TIBC	
Weakness		
Fatigue and pallor		
Dizziness		
Difficulty concentrating		
Shortness of breath		

Gluc	Test for occult blood	Creatinine (Creat)
Alb	Blood urea nitrogen (BUN)	
CRP		
Serum folic acid and B_{12}		

[a]The best indicator for iron availability for erythropoiesis.
[b]Half-life of reticulocytes is 1 day; it represents immediate availability of bone marrow iron.

INTERVENTION

OBJECTIVES

- Prevent infections or sepsis. Reduce fever and excessive inflammation.
- Prevent further complications such as heart failure. Fluid may accumulate and build up in the lungs and liver.
- Support growth in children.
- Improve energy level and decrease fatigue, irritability, and infections.

FOOD AND NUTRITION

- Provide a balanced diet that is easily prepared, with small feedings given frequently.
- Provide extra fluid unless contraindicated.
- Provide sufficient foods rich in iron and B-vitamins, as appropriate (depending on laboratory values, current status, predialysis, or dialysis).

Common Drugs Used and Potential Side Effects

- Iron therapy is effective in 30–50% of patients with CKD. A serum ferritin concentration of 100–500 ng/mL is the target during oral and IV iron therapy for predialysis and peritoneal dialysis patients. IV administration and a target serum ferritin concentration of 200–500 ng/mL is recommended for hemodialysis patients (Besarab et al, 2009; Grabe, 2007).
- Erythropoietin Stimulating Agents (ESAs) are given when Hgb falls below 10 g/dL. Epoetin (EPO) is used when oral iron therapy fails (Nurko, 2006). This can be given every week, or every 2 weeks, or monthly. The two formulations of EPO, epoetin alpha (ProCrit) and darbepoetin (Aranesp, DPO), are effective. Longer half-life of darbepoetin alpha permits administration on a once-monthly basis in patients with CKD and anemia (Grabe, 2007). A recent addition is methoxypolyethylene glycol-epoetin beta (Mircera) that has a longer half-life and can be given every 2 weeks.
- Iron deficiency and inflammation are possible causes of inadequate response to ESAs (Grabe, 2007). In the iron-replete patient with an inadequate response to epoetin, the following conditions should be evaluated and treated, if reversible: infection or inflammation (AIDS, lupus); chronic blood loss; aluminum toxicity; hemoglobinopathies (thalassemias, sickle cell anemia); folate or vitamin B_{12} deficiency; multiple myeloma; malnutrition; or hemolysis.
- Ferric gluconate maintains Hgb and allows lower epoetin doses in anemic hemodialysis patients with low TSAT and ferritin levels up to 1200 ng/mL (Kapoian et al, 2008).
- Parenteral iron is reserved for dialysis patients or those who are intolerant of oral iron. Iron dextran (In-FeD, Dexferrum), sodium ferric gluconate (Ferrlecit), and iron sucrose (Venofer) are available. Ferumoxytol is a new IV iron preparation for CKD (Schwenk, 2010). Iron dextran may cause serious allergic reactions.
- Vitamin C helps increase absorption. Dairy and antacids decrease absorption.
- Docusate helps alleviate constipation. Iron supplements can darken stools.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Vitamin Intake

Assessment Data: CKD with low erythropoietin production; Hgb >12 and Hct >33% with EPO. Serum folic acid and vitamin B_{12} levels remain low. Physical signs of vitamin deficiency.

Nutrition Diagnoses (PES): Inadequate vitamin intake related to poor oral intake as evidenced by diet history, recent anorexia, low serum levels of B_{12} and folate, and minimal use of prescribed water-soluble vitamins.

Interventions: Food-Nutrient Delivery—In addition to EPO and iron, use vitamin B_{12} and folic acid supplements. Educate about the need to use the supplements daily and to retest lab work every 3–6 months. Counseling about good food sources of folic acid and vitamin B_{12}.

Monitoring and Evaluation: Lab work showing normal B_{12} and folic acid levels. Fewer complaints of fatigue; no physical signs of vitamin deficiency.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician. Because the use of CAM is increasing among children and adults with chronic illnesses, efforts should be made to identify those therapies that are beneficial, harmless, and cheap for possible integration with conventional therapy (Oshikoya et al, 2008). Adverse side effects are possible.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss needs of the patient that are specific for signs and symptoms and side effects of any medications.
- Discuss simplified, but nutritious, meal planning.
- If patient has diabetes, heart failure, or cirrhosis, counsel specifically for nutritional management.
- Activity levels must be restricted to avoid accidents or falls that could promote bleeding.

- People who take EPO shots should have regular tests to monitor their Hgb. If it climbs above 12 g/dL, their doctor should prescribe a lower dose of EPO.

Patient Education—Food Safety

If tube feeding or CPN is needed, careful handwashing procedures should be followed.

For More Information

- American Association of Kidney Patients
  http://www.aakp.org/aakp-library/Anemia-in-Chronic-Kidney-Disease/
- National Institute of Diabetes and Digestive and Kidney Diseases–Anemia
  http://kidney.niddk.nih.gov/kudiseases/pubs/anemia/
- National Kidney Foundation – Anemia
  http://www.kidney.org/Atoz/pdf/anemia.pdf

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APLASTIC ANEMIA AND FANCONI'S ANEMIA

NUTRITIONAL ACUITY RANKING: LEVEL 1

DEFINITIONS AND BACKGROUND

Aplastic anemia is a rare bone marrow disorder with normocytic, normochromic anemia in which normal marrow is replaced with fat. Aplastic anemia, myelodysplastic syndromes, and paroxysmal nocturnal hemoglobinuria (PNH) occur when the bone marrow stops making healthy blood-forming stem cells that produce RBCs, white blood cells, and platelets. Telomeres, repeat sequences at the ends of chromosomes, are protective chromosomal structures that shorten with every cell cycle; aplastic anemia is associated with inherited mutations in telomere repair or protection genes (Calado, 2009).

In about 50% of cases, the cause may be inherited or due to autoimmunity. In other cases, exposure to toxic agents (e.g., radiation, heavy metals, inorganic arsenic) or use of drugs (e.g., phenylbutazone, chloramphenicol, anticonvulsants) may be the cause. Use of interferon-gamma (IFN-γ) may be responsible for certain aspects of the pathology seen in bone marrow failure syndromes, including aplastic anemia (Zeng et al, 2006). Signs and symptoms are listed in Table 12-5.

Treatment includes blood transfusion, preventive antibiotics, careful handwashing, hormone therapy, immunosuppressive therapy, and medications to enhance bone marrow cell production. Severe aplastic anemia (SAA) is life threatening and can be treated with bone marrow transplantation, immunosuppressive therapy, and high-dose cyclophosphamide (Brodsky et al, 2009). Resolution of iron overload (such as serum ferritin >1000 ng/mL) should be addressed before transplant because it may lead to lethal infections (Storey et al, 2009).

Fanconi's anemia (FA) is a rare, genetic disorder characterized by multiple congenital anomalies, progressive bone marrow failure, and an increased prevalence of leukemia or liver cancer (Fagerlie and Bagby, 2006). FA is characterized by delayed bone marrow failure with progression to aplastic anemia. It may be apparent at birth or between ages 2 and 15 and is characterized by deficiency of all bone marrow elements including RBCs, white blood cells, and platelets (pancytopenia). FA is associated with cardiac, kidney, or skeletal abnormalities as well as vitiligo or patchy, brown discolorations (pigmentation changes) of the skin. There are

TABLE 12-5 Signs and Symptoms of Aplastic or Fanconi's Anemias

Blood in stool
Bronzing of skin (café au lait spots)
Dizziness
Headache, irritability
Hemorrhagic diathesis (gums, nose, GI tract, urinary tract, vagina)
Hemosiderosis with resulting cirrhosis, diabetes, heart failure
Increasing fatigue and weakness
Increasing or persistent infections
Irritability
Missing or horseshoe kidney (FA)
Missing or misshapen thumbs (FA)
Nausea
Oral thrush or lesions
Petechiae, ecchymosis
Scoliosis
Skeletal anomalies of spine, hips, ribs (FA)
Slow thought processes, headache
Small head, low birth weight (FA)
Tachycardia, tachypnea, dyspnea
Waxlike pallor

several different subtypes, each of which results from abnormal mutations of different genes. Prognosis is poor among those individuals with low blood counts. Treatment of FA involves transfusions, bone marrow transplantation, or gene therapy. FA patients do not tolerate radiation well and are prone to cancers, even after transplantation. Currently, life span is not long; many children do not survive to adulthood.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: In Caucasians, genetic variations in IFNG may be found. Mutations TERC and TERT genes are also seen in aplastic anemia. The FANCM or FNACJ gene mutations are responsible for some forms of Fanconi's anemia.

Clinical/History	Lab Work	
Height	RBC count (decreased)	White blood cell (WBC) count (<1500)
Weight	Prothombin time (PT)	Alb, transthyretin
BMI	Serum Fe	CRP
Diet history	Gluc	Alanine aminotransferase (ALT)
BP	Granulocytes (decreased)	Aspartate aminotransferase (AST)
GI problems	Transferrin	Bilirubin
See Table 12-5 also	H & H	
Bone marrow biopsy	Platelets (decreased)	
Ultrasound		
Hand x-ray or CT scan		

SAMPLE NUTRITION CARE PROCESS STEPS

Self-Feeding Difficulty

Assessment Data: Low BMI, medical hx with diagnosis of Fanconi's anemia, misshapen thumbs with difficulty holding utensils.

Nutrition Diagnoses (PES): Self-feeding difficulty (NB-2.6) related to hand deformity as evidenced by low BMI and difficulty consuming enough at mealtimes.

Interventions: Food-nutrient delivery—add extra kilocalories to foods and recipes, such as extra fats and carbohydrates. Include extra protein-rich foods as tolerated between meals. Serve finger foods and beverages that can be taken through a straw (milkshake, eggnog). Educate parents about changes in menus and foods for greater intake of nutrient and energy-dense foods. Counsel for ways to enhance self-feeding with use of adaptive feeding equipment.

Monitoring and Evaluation: Improvement in BMI over time and better intake from nutrient and energy-dense foods. Enhanced skills using adaptive feeding tools.

INTERVENTION

OBJECTIVES

- Prevent infections or sepsis. Reduce fevers.
- Reduce bleeding tendencies and hemorrhages.
- Ensure adequate periods of rest.
- Prepare for splenectomy or bone marrow transplantation.
- Prevent further complications, where possible, and decline in cardiovascular and hepatic functions.

FOOD AND NUTRITION

- Replenish nutrient stores.
- Provide a balanced diet that is easily prepared, with six small feedings.
- Provide extra fluid unless contraindicated (35 cc/kg or more).
- If patient has mouth lesions, avoid excesses of hot or cold foods, spicy or acidic foods, or foods with rough textures.
- If steroids are used, limit sodium intake.

Common Drugs Used and Potential Side Effects

- Growth factors (erythropoietin, G-CSF, and GM-CSF) may help to improve blood counts.
  - Corticosteroids may be used. Side effects of chronic use include elevated serum glucose and sodium levels, decreased potassium and calcium levels, and negative nitrogen balance.
  - High-dose cyclophosphamide is highly effective therapy for SAA, but large randomized controlled trials are necessary to compare with either bone marrow transplantation or use of antithymocyte globulin and cyclosporine (Brodsky et al, 2009).
  - Antibiotics may be required for infections; monitor for GI distress and other side effects.
  - Aspirin should be avoided because it may aggravate blood losses. Other drugs that may aggravate the condition include chloramphenicol, phenylbutazone, sulfa drugs, and ibuprofen. Each of these has specific GI side effects that should be monitored (see index for more information).
  - A list of drugs that can cause acquired aplastic anemia is found at http://www.wrongdiagnosis.com/a/aplastic_anemia/medic.htm#medication_causes_list

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss needs of the patient, which are specific for signs and symptoms and for side effects of any medications.
- Discuss simplified, but nutritious, meal planning.

- If patient has diabetes, heart failure, or cirrhosis, counsel specifically to those issues for nutritional management.
- Activity levels must be restricted to avoid accidents or falls that could promote bleeding.
- Genetic counseling is advised for parents who wish to have more children.

Patient Education—Food Safety

If tube feeding or CPN is needed, careful handwashing procedures should be followed.

For More Information

- America's Blood Centers
  http://www.americasblood.org/
- Aplastic Anemia and MDS International Foundation, Inc.
  http://www.aplastic.org/
- Bloodline
  http://www.bloodline.net/
- Fanconi's Anemia
  http://www.fanconi.org/aboutfa/diagnosis.htm
- Medline—Fanconi's Syndrome
  http://www.nlm.nih.gov/medlineplus/ency/article/000334.htm
- Fanconi Canada
  http://www.fanconicanada.org

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COPPER DEFICIENCY ANEMIA

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Copper has a role in the production of Hgb (the main component of RBCs), myelin (the substance that surrounds nerve fibers), elastin, collagen (a key component of bones and connective tissue), and melanin (a dark pigment that colors the hair and skin). It is required for the function of over 30 proteins, including superoxide dismutase, ceruloplasmin, lysyl oxidase, cytochrome c oxidase, tyrosinase, and dopamine beta-hydroxylase (Arredondo and Nunez, 2005).

One third of the total body pool of copper is found in skeletal muscle; one third is found in brain and liver; the final third is found in bone and other tissues. Copper is also found in trace amounts in all tissues in the body and is excreted primarily in the bile.

Copper is needed in minute amounts for the formation of Hgb. The metabolism of copper and iron are closely related. Systemic copper deficiency generates cellular iron deficiency, which results in diminished work capacity, reduced intellectual capacity, diminished growth, alterations in bone mineralization, and diminished immune response (Arredondo and Nunez, 2005). Copper deficiency also results in reduced activity of white blood cells and reduced thymus hormone production, thus resulting in increased infection rates.

Marginal deficits of this element can contribute to the development and progression of a number of disease states including cardiovascular disease and diabetes (Li et al, 2005; Urui-Adams and Keen, 2005). Homocysteine thiolactone accumulates when homocysteine is high; it inhibits lysyl oxidase, which depends on copper to catalyze cross-linking of collagen and elastin in arteries and bone. A copper deficiency should, therefore, be avoided. Betaine, copper, folate, pyridoxine, and vitamin B_{12} have proven to be beneficial in lowering serum homocysteine levels. Overall, supplementation with 3–6 mg of copper per day can improve copper status in otherwise healthy individuals; increased intake could reduce the risk of atherosclerosis by promoting improved fibrinolytic capacity (Bugel et al, 2005).

Copper, along with zinc and iron, is an essential metal for normal central nervous system development and function. Imbalances can result in neuronal death (apoptosis), which may contribute to Alzheimer's disease, Parkinson's disease, amyotrophic lateral sclerosis (ALS), and Huntington's disease. Imbalances can also result in neuron deaths in traumatic brain and spinal cord injury, stroke, and seizures (Levenson, 2005).

People with poor intake of protein or whose diets are very high in milk may become deficient in copper or iron. Infants fed on all cow's milk diet without copper supplements may develop copper deficiency. Acquired copper deficiency may be a delayed complication of gastric surgery.

Zinc supplementation (150 mg/d) or vitamin C (1500 mg or more) will reduce copper absorption and increase the potential for anemia. Other conditions that can lead to copper deficiency include burns, pancreatic or liver disease, kidney disease, diarrhea, and prematurity.

Bicytopenia (anemia and neutropenia) with normal platelet count is a feature of hematological disorders caused by copper deficiency; abnormalities improve within a few months after copper supplementation therapy (Nagano et al, 2005). Ceruloplasmin (Cp) is a copper-containing plasma protein with an important role in iron homeostasis; levels are low when copper intake is deficient.

Hospitalized patients should be evaluated carefully. Although enteral feedings contain adequate concentrations

of trace elements, problems with bioavailability may occur, and patients receiving long-term enteral feeding should be monitored to avoid anemia and leukopenia (Ito et al, 2005; Oliver et al, 2005). Copper supplementation is essential in parenteral nutrition to prevent an adverse effect of deficiency; requirements in CPN amount to 0.3 mg/d in adults and 20 μg/kg body wt/d in infants or children (Shike, 2009). Nutritional deficiencies that can occur in otherwise asymptomatic normally growing children are often overlooked (Suskind, 2009). If present, they have a significant impact on the health of the child.

Another area for careful review is bariatric surgery. Serum copper levels should be monitored in patients with a neurologic syndrome, who have undergone gastric bypass surgery (Naismith et al, 2009; Griffith et al, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Copper anemias are acquired.

Clinical/History

Height
Weight
BMI
Diet history
BP
See Table 12-6

Lab Work

CBC
Serum copper (low)
Ceruloplasmin (low)
H & H
Serum Fe
Serum ferritin (increased)
Macrocytic, hypochromic anemia
Platelets (normal)
Erythropoietin levels (elevated)
Serum zinc (Zn) (often elevated)
Homocysteine
Alb, transthyretin
CRP
Retinol-binding protein (RBP)

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Mineral Intake

Assessment Data: Nutritional laboratories showing low-serum copper and Cp with high-serum zinc. Previous bariatric surgery (>1 year) with recent medical visit. Other laboratories are normal. Diet hx reveals low intake of copper from foods.

Nutrition Diagnoses (PES): Inadequate mineral intake of copper related to significant decrease in total food consumed each day for past year post-gastric bypass surgery as evidenced by low-serum Cu and Cp levels and regular use of Zn supplements.

Interventions: Education about ways to enhance copper from meals and snacks. Decrease use of single-Zn supplements and use a multivitamin-mineral supplement that contains copper.

Monitoring and Evaluation: Improved serum Cu and Cp levels after 3 months; better intake of copper-rich foods. Serum Zn within normal range.

TABLE 12-6 Symptoms of Copper Insufficiency and Anemia

Anorexia
Bone fractures
Cardiovascular disease, increased serum cholesterol levels
Diarrhea
Dermatitis or loss of pigmentation of skin, pallor
Edema
Fatigue
Growth retardation
Hair loss
Irregular heart rhythms
Labored respiration, decreased oxygen delivery
Nerve conduction problems
Neurological and immunological abnormalities in newborns if mothers are deficient (Urui-Adams and Keen, 2005).
Myeloneuropathy and myelopathy
Poor collagen formation, decreased wound healing
Reduced red blood cell function
Reduced thyroid function
Shortened red cell life span
Skeletal defects from bone demineralization
Skin sores
Weakness

INTERVENTION

OBJECTIVES

- Correct copper deficiency and documented anemias.
- Instruct patient regarding good sources of protein, iron, and copper to prevent recurrences.
- Monitor use of zinc in supplements, diet, and enteral or parenteral sources to avoid overdosing and related copper depletion.

FOOD AND NUTRITION

- Good sources of copper include oysters, liver, nuts, dried legumes, and raisins. A typical diet provides about 2- to 3-mg copper/d, about half of which is absorbed. A supplement of 3- to 6-mg copper may be useful in adults.
- Protein should be at least 1 g/kg for adults; iron intake should be adequate for age and sex.
- Monitor use of multivitamin-mineral supplements to avoid large doses of zinc. Ascorbic acid can act as a pro-oxidant in the presence of metals such as iron or copper; large doses are not recommended.
- Monitor tube-fed patients to ensure that they are receiving sufficient amounts of copper (Ito et al, 2005; Oliver et al, 2005).

TABLE 12-7 Food Sources of Copper

Blackstrap molasses
Black pepper
Chicken
Chocolate (unsweetened or semisweet baker's chocolate and cocoa)
Enriched cereals (bran flakes, raisin bran, shredded wheat)
Fruits (such as cherries, dried fruits, bananas, grapes)
Legumes (such as soybeans, lentils, navy beans, and peanuts)
Nuts and nut butters (cashews, filberts, macadamia nuts, pecans, almonds, and pistachios)
Organ meats (beef liver, kidneys, and heart)
Potatoes
Seafood (oysters, squid, lobster, mussels, crab, and clams)
Seeds (pumpkin)
Tea
Vegetables (avocado, mushrooms, potatoes, sweet potatoes, tomatoes)
Whole grains

Common Drugs Used and Potential Side Effects

- Cupric sulfate is one brand of injectable copper supplement. Copper gluconate is given orally. Beware of excesses, which can be indicated by black or bloody vomit, bloody urine, diarrhea, heartburn, loss of appetite, lower back pain, metallic taste, nausea (severe or continuing), pain or burning while urinating, vomiting, yellow eyes or skin, dizziness or fainting, severe headache, or even coma.
- Do not refrigerate this supplement. Discard when outdated.
- Avoid taking copper supplements with nonsteroidal anti-inflammatory drugs (NSAIDs), birth control pills, allopurinol, estrogen hormones, or cimetidine.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Have patient avoid fad diets. Monitor vegetarian (non-heme iron) diets carefully.
- Zinc in large doses may deplete copper levels; discuss use of mineral supplements. (Rowan and Lewis, 2005).
- Patients at risk for copper deficiency should be counseled on how to avoid this condition. For example, patients with muscular dystrophy may not consume adequate amounts, and muscle strength can diminish as a result (Motlagh et al, 2005).
- Discuss foods that are good sources of protein, iron, and copper; see Table 12-7.
- Be aware that some denture creams contain a high amount of zinc that could be a concern (Spain et al, 2009).

Patient Education—Food Safety

If tube feeding or CPN is needed, careful handwashing procedures should be followed.

For More Information

- Merck Manual
  http://www.merck.com/mrkshared/mmanual/section1/chapter4/4j.jsp
- Northwestern University – Copper Fact Sheet
  http://www.feinberg.northwestern.edu/nutrition/factsheets/copper.html

COPPER DEFICIENCY ANEMIA—CITED REFERENCES

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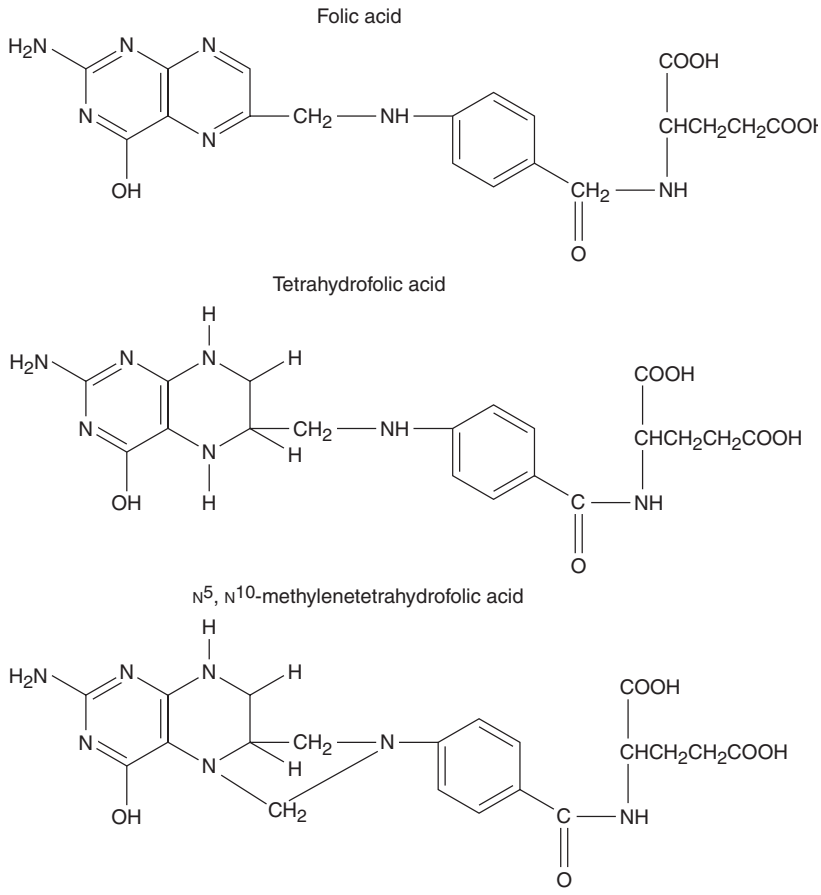
FOLIC ACID DEFICIENCY ANEMIA

NUTRITIONAL ACUITY RANKING: LEVEL 2



Three steps for metabolism of dietary folic acid to the bioavailable form.

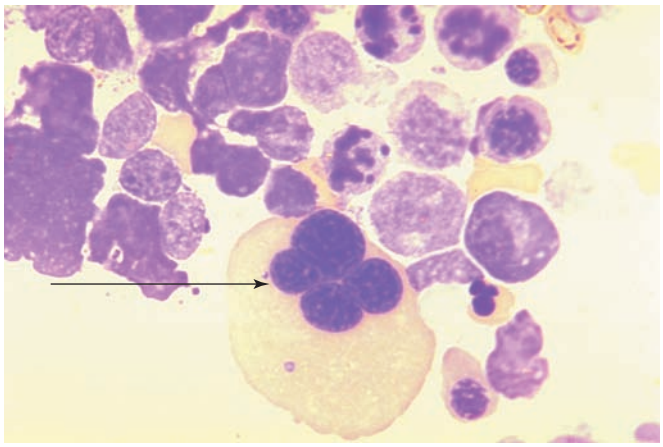
DEFINITIONS AND BACKGROUND

Folic acid is composed of a pterin ring connected to *p*-aminobenzoic acid (PABA). Humans do not generate folate because they cannot synthesize PABA. The amino acid histidine is metabolized to glutamic acid. Formiminoglutamic acid (FIGLU) is an intermediary in this reaction, and tetrahydrofolic acid is the coenzyme that converts it to glutamic acid. Under normal conditions, sufficient intake of dietary histidine can prevent anemia. When dietary intake of histidine is diminished or urinary excretion is greatly increased, anemia results. Folate deficiency depletes histidine through increased urinary excretion.

Folic acid is needed for the synthesis of DNA and maturation of RBCs. Deficiency of folate can lead to many clinical abnormalities, including macrocytic anemia, cardiovascular diseases, birth defects, and carcinogenesis (including colorectal cancer). Folic acid-deficiency anemia generally is caused by inadequate diet, intestinal malabsorption, alcoholism, or pregnancy (Table 12-8).

Folic acid deficiency yields a hyperchromic, macrocytic, megaloblastic anemia. Because similar hematological changes occur with vitamin B_{12} deficiency, it is important to check the serum levels of vitamin B_{12} along with folate tests. Folate is best measured by RBC folate because serum levels are misleading and reflect more recent intake.

Homocysteine elevation is a risk factor for vascular and thrombotic disease. Genetic and acquired influences have been evaluated. While neural tube defects result from maternal



Folic acid anemic cells are hypochromic and macrocytic. Adapted from: *Anderson's Atlas of Hematology;* Anderson, Shauna C., PhD. Copyright 2003, Wolters Kluwer Health/Lippincott Williams & Wilkins.

TABLE 12-8 Conditions and Medications That Deplete Folic Acid

Aging
Alcoholism
Blind-loop syndrome
Burns
Cancers
Celiac disease
Crohn's disease
Dialysis
Elevated homocysteine levels
Hemolytic anemias
Hepatitis
Infection
Inflammatory diseases
Malabsorption
Megacolon
Pregnancy and lactation
Smoking
Stress
Surgery
Medications that interact with folic acid:

- Antiepileptic drugs (AED): phenytoin, carbamazepine, primidone, valproic acid, phenobarbital, and lamotrigine impair folate absorption and increase the metabolism of circulating folate
- Capecitabine: Folinic acid (5-formyltetrahydrofolate) may increase the toxicity of Capecitabine
- Dihydrofolate reductase inhibitors (DHFRI): DHFIs block the conversion of folic acid to its active forms, and lower plasma and red blood cell folate levels. DHFIs include aminopterin, methotrexate, sulfasalazine, pyrimethamine, triamterene, and trimethoprim. Administer leucovorin at the same time
- Nonsteroidal anti-inflammatory drugs (NSAIDs): NSAIDs inhibit some folate dependent enzymes. NSAIDs include ibuprofen, naproxen, indomethacin, and sulindac
- Cholestyramine, Colestipol, Cycloserine, Isotretinoin, oral contraceptives, Methylprednisolone, pancreatic enzymes, Pentamidine, Sulfasalazine either decrease folic acid absorption or increase excretion
- Smoking and alcohol: reduced serum folate levels may occur

folate insufficiency in the periconceptual period, there are also inborn errors of folate metabolism that aggravate the problem.

DNA methylation occurs by transfer of a methyl group from *S*-adenosylmethionine (SAM) to cytosine (Abdolmaleky et al, 2004). SAM serves as a methyl group donor in important functions such as changing norepinephrine to epinephrine, chromatin remodeling, RNA inhibition and modification, and DNA rearrangement (Abdolmaleky et al, 2004). Prenatal intakes of folic acid, vitamin B_{12}, choline, and betaine influence the degree of DNA methylation (Waterland and Jirtle, 2004). Methylation affects adult susceptibility to asthma, cancer, autism, bipolar disease, Alzheimer's disease, stroke, and schizophrenia.

Folate deficiency can result in congenital neural tube defects and megaloblastic anemia; inadequacy is associated with high blood levels of the amino acid homocysteine, which has been linked with the risk of arterial disease, dementia, and Alzheimer's disease (Malouf et al, 2008). Decreasing or low levels of folic acid may also be associated with depression in older adults.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

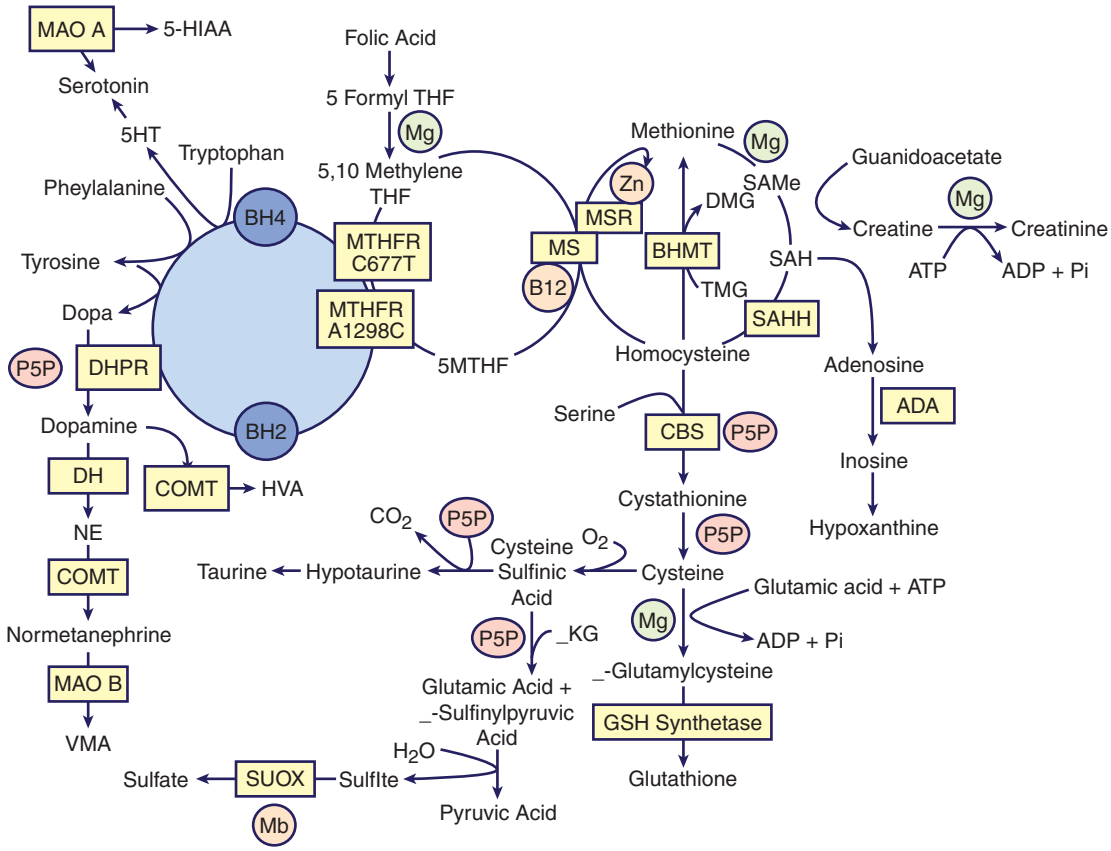
Genetic Markers: Congenital folate malabsorption, methylenetetrahydrofolate reductase (MTHFR) deficiency, and formiminotransferase deficiency are genetic defects. MTHFR deficiency causes neurological problems even without megaloblastic anemia. MTHFR polymorphisms on chromosome 1 affect 10% of the world's population, especially Caucasians. Varied DNA methylation patterns influence the biological response to food components and vice versa (Milner, 2006).

Clinical/History	Lab Work	
Height	RBC folate (<140 ng/mL) – best	Serum Fe (increased)
Weight	Serum folate (<3 ng/mL)	Mean cell volume (MCV)
BMI	MTHFR alleles?	Leukopenia, WBC
Diet history	Mild hyperhomocystinemia (15–25 mmol/L)	Urinary formiminoglutamic acid (FIGLU) after histidine load
BP	Moderate hyperhomocystinemia (26–50 mmol/L)	Serologic testing for parietal cell and intrinsic factor (IF) antibodies (vs. Schilling test)
I & O	Low RBC	
Weight loss	H & H	
Anorexia, malnutrition	CBC (macrocytic cells)	
Smooth and sore red tongue	Transferrin	
Diarrhea	Serum B_{12}	
Fatigue, lethargy		
Poor wound healing		
Coldness of extremities		
History of alcohol abuse?		

INTERVENTION

OBJECTIVES

- Increase folate in diet and supplemental folic acid to alleviate anemia.
- Improve diet to provide nutrients needed to make RBCs: folate and other B-complex vitamins, iron, and protein. Instruct patient to correct faulty diet habits if relevant.
- Check for malabsorption syndromes (celiac disease, blind-loop syndrome, congenital or acquired megacolon, Crohn's disease) and correct these as far as possible through use of medications and other treatments.
- Monitor serum folic acid status regularly.



Folic acid-B_{12} pathway, showing the many substances that could be impacted by deficiency or genetic alterations.

FOOD AND NUTRITION

- Provide a diet that is high in folate, protein, copper, iron, and vitamins C and B_{12}.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Vitamin Intake and Food- and Nutrition–Related Knowledge Deficit

Assessment Data: Hx of MTHFR (C>T) allele and spina bifida in two infants. Diet Hx shows poor intake of folate from diet and no use of supplemental sources. Dislikes fruits and vegetables.

Nutrition Diagnoses (PES): Inadequate vitamin intake of folate (NI 5.9.1.9) related to lack of folic acid food sources in daily diet as evidenced by headache, fatigue, decreased serum and RBC folate, and giving birth to two infants with spina bifida in the past 5 years. Food and nutrition-related knowledge deficit (NB 1.1) related to lack of prior exposure and interest in accurate nutrition-related information as evidenced by daily intake lacking sources of folate, especially from fruits and vegetables.

Interventions: ND 3.2.1, 3.2.3.9—multivitamin/mineral supplement, folic acid supplement of additional 400 mcg/d. E 1.2—priority modifications to diet including addition of food sources rich in folic acid, basic information concerning food pyramid, and balanced meal planning.

Monitoring and Evaluation: One month follow-up visit acceptance of supplements, dietary recall for daily use of folic acid sources, and efficacy of personal meal planning for meeting basic nutrition needs/food pyramid servings.

- Folic acid is distributed widely in green leafy vegetables, citrus fruits, and animal products. Ingestion of one fresh fruit or vegetable provides sufficient folic acid for most people. Other sources include fish, legumes (dried beans and peas), whole grains, leafy dark green vegetables, broccoli, citrus juices, berries, and meats.
- Food manufacturers in the United States have fortified grains with folic acid since 1998. These fortified foods include most enriched breads, flours, corn meal, rice, noodles, macaroni, and other grain products.
- Diets that provide bland, liquid, or soft foods may be needed for patients with a sore mouth; six to eight small meals may be helpful.

Common Drugs Used and Potential Side Effects

- Supplements of folic acid (Folvite) are better than diet alone to alleviate the anemia. Folate deficiency is generally treated with 800–1000 μg of folic acid daily. Leucovorin is an active reduced form.
- Be aware of the interdependence between vitamin B_{12} and folic acid, especially in older individuals. Vitamin B_{12} helps methylate folic acid so that it can travel into cells. The transport form is 5-methyltetrahydrofolate. Because B_{12} deficiency can produce anemia similar to folate deficiency, there is a risk that folate supplementation can delay the diagnosis of B_{12} deficiency, which can lead to irreversible neurological damage (Malouf, 2009).
- 5-methyltetrahydrofolate is an option for supplementation and fortification without masking the anemia of vitamin B_{12} deficiency (Pentieva et al, 2004). Metafolin is a

registered trademark of Merck; Deplin is one brand, containing 7.5 mg of L-methylfolate.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Vitamin C promotes absorption of folate from foods. See Table 12-9 for a list of folate sources.
- Pregnant women should receive appropriate counseling; 30% may have a folate deficiency. Daily needs increase by approximately 200 μg over the adult requirements of 400 μg. Folate protects against neural tube defects in the first trimester.

TABLE 12-9 Folic Acid Sources

Source	Folic Acid (μg)
Breakfast cereals fortified with 100% of the DV	400
Beef liver, cooked, braised, 3 ounces	185
Black-eyed peas, immature, cooked, boiled, half cup	105
Spinach, frozen, cooked, boiled, half cup	100
Great Northern beans, boiled, half cup	90
Asparagus, cooked, four spears	85
Broccoli, cooked, half cup	84
Rice, white, long-grain, parboiled, enriched, cooked, half cup	65
Vegetarian baked beans, canned, one cup	60
Spinach, raw, one cup	60
Green peas, frozen, boiled, half cup	50
Broccoli, chopped, frozen, cooked, half cup	50
Egg noodles, cooked, enriched, half cup	50
Avocado, raw, all varieties, sliced, half cup sliced	45
Peanuts, all types, dry roasted, 1 ounce	40
Lettuce, Romaine, shredded, half cup	40
Wheat germ, crude, two tablespoons	40
Tomato Juice, canned, 6 ounces	35
Orange juice, chilled, includes concentrate, three-fourth cup	35

Derived from: NIH Fact Sheet, http://ods.od.nih.gov/factsheets/folate.asp, accessed December 27, 2009.

- Women who have a folic acid allele in the MTHFR gene may need to use a special brand of supplement, such as Neevo (pamlabs.com) during pregnancy.
- Large intakes of folic acid (>1 mg/d) can cure the anemia but may mask a correlated vitamin B_{12} anemia; monitor carefully. 5-methyl THFA enters cells via a diverse range of folate transporters where it may be demethylated to THFA, the active form. Because vitamin B_{12} is required in this conversion, its absence traps folic acid in its inactive form as 5-methyl THFA.
- In seniors with low vitamin B_{12} status, high-serum folate is associated with anemia and cognitive impairment but when not vitamin B_{12} status was normal; however, high-serum folate was associated with protection against cognitive impairment (Morris et al, 2009).
- Attractive meals may help appetite. Fad and restrictive diets should be avoided.
- Alcoholic beverages interfere with folate metabolism and absorption.
- Food folates are oxidized easily and destroyed by lengthy cooking; advise patients accordingly.

Patient Education—Food Safety

If tube feeding or CPN is needed, careful handwashing procedures should be followed.

For More Information

- E-medicine
  http://www.emedicine.com/med/topic802.htm
- Folic Acid Supplements
  http://ods.od.nih.gov/factsheets/folate.asp
- March of Dimes–Folic Acid Deficiency
  http://www.marchofdimes.com/professionals/19695_1151.asp

FOLIC ACID DEFICIENCY ANEMIA—CITED REFERENCES

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Waterland RA, Jirtle RL. Early nutrition, epigenetic changes at transposons and imprinted genes, and enhanced susceptibility to adult chronic diseases. *Nutrition.* 20:63, 2004.

HEMOLYTIC ANEMIAS

NUTRITIONAL ACUITY RANKING: LEVELS 2–3

DEFINITIONS AND BACKGROUND

In **hemolytic anemia,** RBCs have an abnormal membrane, which results in hemolysis. RBCs are destroyed faster than they can be produced in bone marrow. In severe cases in infancy, encephalomalacia can result. The incidence of all types of hemolytic anemias is 4 in 100,000 persons in the United States. Treatment may involve splenectomy or steroid use. Most are not affected specifically by vitamin E.

Types of hemolytic anemias include Hgb-SC disease, hemolytic anemia due to glucose-6-phosphate dehydrogenase (G6PD) deficiency, hereditary elliptocytosis, hereditary spherocytosis, idiopathic autoimmune hemolytic anemia, nonimmune hemolytic anemia caused by chemical agents, and secondary immune hemolytic anemia. This text covers aplastic anemia, sickle cell anemia, and thalassemia. Table 12-10 describes some of these types of anemias.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Some types of hemolytic anemia have genetic links.

Clinical/History

- Dizziness
- Edema
- Pallor
- Nosebleeds, bleeding gums
- Dark urine
- Jaundice, splenomegaly
- Puffy eyelids
- Weakness, confusion
- Chills
- Fatigue, intolerance for exercise
- Heart murmur
- Height
- Weight
- BMI
- Diet history
- Growth percentile
- BP
- Tachycardia
- Shortness of breath

Lab Work

- RBC (low)
- Hgb (low)
- Reticulocyte count (increased)
- Serum alpha-tocopherol levels
- Hgb in urine
- Hemosiderin in urine
- TIBC
- Bilirubin (elevated)
- Transferrin
- Gluc
- AST (increased)
- Blood test for G6PD
- CRP

INTERVENTION

OBJECTIVES

- Prevent further complications.
- Correct anemia or deficits of nutrients, such as vitamin E.

FOOD AND NUTRITION

- Provide diet as usual for age and sex.
- Avoid excesses of iron.
- Ensure adequate intake of vitamin E and zinc, which may become deficient. Good sources of vitamin E include wheat germ, almonds, sunflower seeds, sunflower or safflower oil, peanut butter, peanuts, corn oil, spinach, broccoli, and

TABLE 12-10 Types of Hemolytic Anemia

Type	Description
Acquired autoimmune hemolytic anemia	A rare autoimmune disorder characterized by the premature destruction of RBCs. Normally, RBCs have a life span of 120 days before the spleen removes them, but in this condition, RBCs are destroyed prematurely. Bone marrow production of new cells can no longer compensate. This anemia occurs in individuals who previously had a normal RBC system. Patients with autoimmune hemolytic anemia usually are associated with thrombosis
Familial hemolytic jaundice (spherocytic anemia)	A hereditary anemia in which RBCs are shaped like spheres rather than their normal, donut-like shape. Jaundice and anemia occur from destruction of the abnormal cells by the spleen. Surgical removal of the spleen usually is indicated. There is no permanent cure
Glucose-6-phosphate dehydrogenase (G6PD) deficiency anemia	This anemia is seen in about 10% of African–American males in the United States and is also common in persons from the Mediterranean area or Asia. The severity differs among different populations. In the most common form in the African–American population, the deficiency is mild, and the hemolysis affects primarily older RBCs. In Caucasians, G6PD deficiency tends to be more serious because even young red blood cells are affected. It affects millions of people worldwide, especially in malaria-prone areas
Hereditary nonspherocytic hemolytic anemia	A group of rare genetic blood disorders characterized by defective RBCs (erythrocytes) that are not abnormally "sphere shaped" (spherocytes). Membranes of RBCs, abnormal metabolism of a chemical contained in hemoglobin (porphyrin), and deficiencies in certain enzymes such as G6PD or pyruvate kinase are thought to be the cause of these disorders
Vitamin E–sensitive hemolytic anemia	This condition may occur in infants who receive polyunsaturated fatty acids (PUFAs) without adequate vitamin E. Children with cystic fibrosis should be screened for vitamin E–deficient hemolytic anemia

SAMPLE NUTRITION CARE PROCESS STEPS

Unintentional Weight Loss

Assessment Data: BMI 20, recent weight loss 10#. GI distress and pallor noted. Diagnosis of hemolytic anemia with splenectomy planned.

Nutrition Diagnoses (PES): Unintentional weight loss related to GI distress and loss of appetite as evidenced by recent weight loss of 10# in 6 weeks.

Interventions: Prepare for splenectomy; use nutrient-dense and energy-rich foods as tolerated, such as milkshakes or eggnogs with or between meals. Educate patient about ways to enhance food intake while not feeling well.

Monitoring and Evaluation: Postoperative evaluations; return of appetite and improved intake. No further weight loss; eventual weight regained.

soybean oil. Good sources of zinc include oysters, beef shank, crab, pork, chicken, lobster, baked beans, cashews, and yogurt.

Common Drugs Used and Potential Side Effects

- For hemolytic anemia that is sensitive to vitamin E deficiency, water-soluble vitamin E (alpha-tocopherol) is likely to be given daily. Avoid taking with an iron supplement, which could interfere with utilization.
- Persons with G6PD deficiency need to avoid exposing themselves to certain medicines such as aspirin (acetylsalicylic acid), certain antibiotics used to treat infections, fava beans, and mothballs.
- Medicines can improve autoimmune hemolytic anemia (AIHA). Where prednisone is used, monitor for side effects. Monoclonal antibody therapy such as rituximab is used in difficult cases; it appears to be a safe and effective option (Hoffman, 2009).

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Flavonoid preparations, marketed for a variety of effects and generally safe, should be evaluated carefully because there have been reports of toxic flavonoid–drug interactions, hemolytic anemia, and other problems (Galati and O'Brien, 2004).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- For hemolytic anemia that is sensitive to vitamin E deficiency, discuss, in layman's terms, the role of vitamin E in lipid oxidation and utilization. Discuss sources of polyunsaturated fatty acids (PUFAs) and why excesses should be controlled. Discuss sources of vitamin E in the diet; natural sources are more bioavailable than synthetic sources.
- Discuss exercise tolerance and ability to eat sufficient amounts of food as related to fatigue.

Patient Education—Food Safety

If tube feeding or CPN is needed, careful handwashing procedures should be followed.

For More Information

- American Autoimmune Related Diseases Association, Inc. http://www.aarda.org
- Medline http://www.nlm.nih.gov/medlineplus/ency/article/000571.htm
- NIH – Hemolytic Anemias http://www.nhlbi.nih.gov/health/dci/Diseases/ha/ha_whatis.html

HEMOLYTIC ANEMIAS—CITED REFERENCES

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IRON-DEFICIENCY ANEMIA

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Hgb transports oxygen to the tissues and carbon dioxide back to the lungs where it is exhaled. Hgb levels are influenced by sex, age, altitude, and smoking. In the adult male and the elderly, iron deficiency is usually caused by chronic blood loss. In children and women, low intake of iron may be a problem. The nutrient most commonly deficient in the world is iron. Iron deficiency affects two billion people, mostly in developing countries (Lynch, 2005).

IDA results from inadequate intake, impaired erythropoiesis or absorption of iron, blood loss, or demands from closely repeated pregnancies (Table 12-11). Serious systemic consequences include impaired cognitive function, koilonychia, and impaired exercise tolerance. Hct is the measure of RBCs in a given volume of blood, packed by centrifuge. Transferrin is the carrier protein that picks up iron from the intestines.

Absorption of iron occurs in the ferrous form; storage is in the liver, spleen, and bone marrow. See Table 12-12 for

TABLE 12-11 Stages of Iron Deficiency

Stages of Iron Deficiency	Indicator	Diagnostic Range
Stage 1 Depletion of iron stores	Stainable bone marrow iron Total iron binding capacity Serum ferritin concentration	Absent >400 μg/dL <12 μg/L <20 μg/L + low Hb or Hct indicates iron deficiency
Stage 2 Early functional iron deficiency	Transferrin saturation Free erythrocyte protoporphyrin Serum transferrin receptor	<16% >70 μg/dL erythrocyte >8.5 mg/L
Stage 3 Iron deficiency anemia	Hemoglobin concentration Mean cell volume	<12 g/dL <80 fL

Adapted from: Dietary Reference Intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc (2002), Iron, p. 302 Food and Nutrition Board (FNB), Institute of Medicine (IOM).

content of iron in various body sources. Approximately 90% of the body's store of iron is reused. Diet replaces iron lost through sweat, feces, and urine. The duodenum (upper small intestine) is where iron is best absorbed. Damage or surgery of the duodenum can greatly inhibit total iron absorption, thus leading to greater risk of deficiency. Table 12-13 describes factors that can modify iron absorption.

IDA is the final stage of a long period of deprivation. Serum ferritin (storage form) is the most useful test to differentiate IDA from ACD. Iron deficiency is relatively common in toddlers, adolescent girls, and women of childbearing age. Ingestion of cow's milk causes occult intestinal blood loss in young infants. The Hgb content of reticulocytes (young RBCs) is a good indicator of iron deficiency and IDA in children. Risk of iron deficiency may be underestimated in high-risk populations.

Postpartum anemia is associated with breathlessness, tiredness, palpitations, and maternal infections, and blood transfusions and iron supplementation have been used in the treatment of IDA. Erythropoietin may be useful.

TABLE 12-12 Normal Iron Distribution in the Body

Forms	Men (mg iron/kg BW)	Women (mg iron/kg BW)
Storage—ferritin	9	4
Storage—hemosiderin	4	1
Transport protein: transferrin	<1	<1
Functional hemoglobin	31	31
Functional myoglobin	4	4
Enzymes	2	2
TOTAL	50	42

Based on data from: Insel P, Turner R, Ross D. *Nutrition*. Sudbury, MA: Jones & Bartlett Publishers, 2001.

TABLE 12-13 Factors That Modify Iron Absorption

Factor	Description
Physical state (bioavailability)	Heme > Fe^{2+} > Fe^{3+}
High gastric Ph	Hemigastrectomy, vagotomy, pernicious anemia, histamine H_2-receptor blockers, calcium-based antacids
Disruption of intestinal structure	Crohn's disease, celiac disease (nontropical sprue)
Inhibitors	Phylates, tannins, soil clay, laundry starch, iron overload
Competitors	Cobalt, lead, strontium
Facilitators	Ascorbate, citrate, amino acids, iron deficiency

From: Information Center for Sickle Cell and Thalassemic Disorders. Iron deficiency. Available at http://sickle.bwh.harvard.edu/fe-def.html.

Celiac disease may be present in children and is associated with IDA (Goel et al, 2005). In persistent IDA, screening for celiac disease (anti-tissue transglutaminase antibodies), autoimmune gastritis (gastric, anti-parietal, or anti-IF antibodies), and *Helicobacter pylori* (IgG antibodies and urease breath test) is recommended (Hershko and Ronson, 2009).

Because menstruation increases iron losses each month, women of childbearing age tend to become iron deficient. When there is not enough Hgb, free erythrocyte protoporphyrin (FEP) accumulates. Athletes are also at risk for iron deficiency. Recreational athletes should be screened for iron deficiency using serum ferritin, serum transferrin receptor, and Hgb (Sinclair and Hinton, 2005).

As many as 25% of children and 20% of those seen in mental health clinics have pica, which is characterized by persistent and compulsive cravings to eat nonfood items. Pica can occur in pregnant women, in autism, and in persons with brain injuries. Pica is seen in about half of patients with iron deficiency; it is a consequence of iron deficiency and is relieved by iron supplementation.

Exposure to lead also has a significant effect on Hgb and Hct levels. Serum levels above 50 mcg/dL are a problem. Lead poisoning reduces Hgb production, causes iron deficiency, and elevates FEP as the precursor.

Poor intake of vitamins A, B_{12}, C, and E, folic acid, and riboflavin is also linked to the development and control of IDA. Multiple micronutrient (MMN) supplementation during pregnancy reduces the risk of low birth weight, small-for-gestational age, and anemia; MMN supplementation improves CD4 counts and HIV-related morbidity and mortality in adults (Allen et al, 2009).

When Hgb levels are seriously low, the heart is particularly vulnerable. Anemia in heart failure patients is associated with reduced exercise tolerance, increased heart failure hospitalizations, and increased all-cause mortality (Stamos and Silver, 2009). Whole-blood transfusion or IV iron may be needed. Iron fortification of food is also a cost-effective method for reducing the prevalence of nutritional iron deficiency. In populations where young children are routinely fed cooked rice daily, fortifying it with iron helps improve iron status (Beinner et al, 2010).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Mutations in a type-II serine protease, matriptase-2/TMPRSS6, are associated with severe iron deficiency caused by inappropriately high levels of hepcidin expression; hemojuvelin is a cell surface protein that regulates hepcidin expression (Lee, 2009).

Clinical/History

- Weight
- BMI
- Diet history
- BP
- I & O
- Pallor
- Brittle, spoon-shaped fingernails (koilonychia)
- Stool examination for occult blood
- Impaired cognitive function
- Blue sclerae
- Impaired exercise tolerance
- Weakness, fatigue
- Vertigo
- Headache, irritability
- Heartburn
- Dysphagia
- Flatulence, vague abdominal pains
- Anorexia
- Diarrhea
- Glossitis, stomatitis
- Ankle edema
- Tingling in extremities
- Palpitations
- Alopecia

Lab Work

- Ferritin (decreased stores in liver, spleen, bone marrow; levels are <20 g/L)
- Serum iron (low)
- H & H (Hgb is more sensitive)
- Mean cell Hgb (MCH) (decreased)
- Mean cell Hct (MCHC) (decreased)
- CBC (every 3–6 months after initial)
- Transferrin (increased)
- MCV (<80)
- RBC (small, microcytic, hypochromic)
- WBC/differential (increased)
- TIBC (increased >350 μg/dL)
- Reticulocyte count
- Serum copper
- Cholesterol (Chol)
- Test for *H. pylori*

SAMPLE NUTRITION CARE PROCESS STEPS

Harmful Beliefs About Food

Assessment Data: Diet hx indicates pica during this pregnancy. Mom states that "the starch is good for my baby." Eats starch after each meal. Twenty-weeks pregnant; age 19. Low H & H.

Nutrition Diagnoses (PES): Harmful beliefs/attitudes (NB-1.2) about food or nutrition-related topics.

Interventions: Initial/Brief nutrition education (N.I.2.2) to provide basic nutrition-related educational about food and foods rich in iron; reasons to discontinue eating starch and to begin taking a prenatal vitamin. Counseling—work with client to set goals for a healthier pregnancy.

Monitoring and Evaluation: Improvement in H & H. Discontinuation of pica. Positive pregnancy outcome.

INTERVENTION

OBJECTIVES

- Alleviate cause of the anemia and associated anorexia.
- Provide adequate oral iron to replace losses or deficits, especially heme sources of protein (liver, beef, oysters, lamb, pork, ham, tuna, shellfish, fish, and poultry).
- Provide an acid medium to favor better absorption. Enhancers include gastric juice and ascorbic acid. Food sources of vitamin C should be included daily.
- Monitor and correct pica, including geophagia (clay eating), amylophagia (starch eating), ice eating, or any lead exposure.
- Avoid or correct constipation.
- Screen for IDA or sports anemia in athletes (Sinclair and Hinton, 2005).
- Reduce iron inhibitors, such as excessive fiber (as in whole grains), phytic acid (as in spinach, bran, legumes, and soy products), tannins in tea, and polyphenols in coffee or red wine. In many developing countries, cereal and legume-based diets contain low amounts of bioavailable iron, which may increase the risk of iron deficiency (Zimmermann et al, 2005).

FOOD AND NUTRITION

- If IDA is related to inadequate iron in diet, usually adding three portions of lean red meat (heme iron sources) per week, along with all other essential vitamins and minerals, will correct the anemia. The average mixed diet contains approximately 6 mg of iron per 1000 kcal. Iron absorption increases as stores become depleted. Good sources of iron include liver, dried beans, egg yolks, kidney, lean beef, dark meat of chicken, salmon, tuna, dried fruits, enriched whole-grain cereals, molasses, and oysters.
- Heme iron is found readily in beef, pork, and lamb; consume with fruit or fruit juice. Heme iron is absorbed well, regardless of other foods in the diet.
- Nonheme iron absorption is greatly affected by other foods. Absorption of nonheme iron is best in the presence of foods rich in vitamin C or with heme-containing sources. Increase intake of vitamin C (oranges, grapefruit, tomatoes, broccoli, cabbage, baked potatoes, strawberries, cantaloupe, and green peppers), especially with an iron supplement.
- Detect pica and discuss with patient. Pica substance may displace other important foods, leading to nutrient malnutrition. The ingested substance may also be toxic.
- Tea, coffee, wheat brans, and soy products tend to inhibit absorption of nonheme iron. Monitor use carefully; avoid excesses.

Common Drugs Used and Potential Side Effects (Table 12-14)

- If anemia is caused by an increased demand for iron such as a growth spurt (toddlers, adolescents) or pregnancy, oral supplementation may be necessary: inorganic

TABLE 12-14 Medications to Correct Iron-Deficiency Anemia

Medication	Description
Ferrous salts (Feosol, Fer-In-Sol, Mol-Iron) or tablets (Feostat, Fergon, Feosol)	Prolonged-release ferrous sulfate (Slow Fe) improves iron absorption with fewer side effects than standard ferrous sulfate pills. Other forms include ferrous fumarate (Femiron, Feostat, Fumerin, Hemocyte, Ircon) and ferrous gluconate (Fergon, Ferralet, Simron). These may cause gastric irritation and constipation.
Enteric-coated or sustained-release iron	More expensive and often carry the iron past maximal absorption site in the upper intestine.
Heme iron (Proferrin Forte)	This is a medical food that contains heme iron plus folic acid. It is absorbed regardless of achlorhydria, and has fewer GI side effects than IV or ferrous iron sources. It can be taken with or without meals.
Parenteral or IV iron	Can be administered by injection or infusion. This therapy is reserved for cases of trauma where blood loss is life threatening and is not used for insufficiency due to inadequate dietary iron intake. Imferon can be given intramuscularly, if oral iron is not tolerated; pain and skin discoloration may result.

iron in ferrous form (50–200 mg/d for adults; 6 mg/kg for children) combined with increased consumption of heme-rich sources of iron. This is best absorbed on an empty stomach, but with food if there are GI side effects.

- Iron pills should be taken 2 hours before or after other medications. Iron can inhibit the effectiveness of thyroid medications, antibiotics, and some antidepressant drugs. Once ingested, it is imperative that the stomach contains acid to dissolve the iron salt; if taking antacids or H2 blockers such as cimetidine (Tagamet), the iron salt will not dissolve.
- The amount of elemental iron contained in iron pills will vary. A 325-mg supplement is probably made of ferrous fumarate or gluconate, with only 100 mg of elemental iron per pill.
- Heme iron supplements (such as Proferrin) can be taken with meals, unlike ionic iron preparations, which must be taken on an empty stomach between meals. However, individuals with allergies to beef, milk, or other dairy products should not be given Proferrin.
- It takes 4–30 days to note improvements after iron therapy, especially in Hgb levels. Hgb should rise 0.1–0.2 g/dL/day after the fifth day of treatment; then should rise 2.0 g/dL/week for 3 weeks. Iron therapy should be continued for at least 2 months after the Hgb has returned to normal to replenish the iron stores.
- Iron stores are replaced after 1–3 months of treatment. Increased supplementation in normal individuals can cause additional, unnecessary iron to go into storage, reflected by ferritin elevation.
- Aspirin or corticosteroids can cause GI bleeding or peptic ulceration. Vitamin C and nutrient levels may be decreased.
- Some medications, including antacids, can reduce iron absorption. Iron tablets may also reduce the effectiveness of other drugs, including the antibiotics tetracycline, penicillamine, and ciprofloxacin and the anti-Parkinson's drugs methyldopa, levodopa, and carbidopa. Wait 2 hours between doses of these drugs and iron supplements.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Hgb is made from protein, iron, and copper. RBCs are made from vitamin B_{12}, folacin, and amino acids. Explain which foods are good sources of iron, protein, vitamin C, and related nutrients.
- Temporary changes in stool color (green or tarry and black) are common with supplements; this is not cause for alarm. To avoid side effects of supplements, take them with meals or milk; food iron has fewer side effects.
- Foods and substances that can interfere with the absorption of iron include calcium, tannins, which are found in coffee, tea, grapes, red wine, purple or red rice, and bran fiber or chocolate. Avoid excesses of oxalates, alkalis, and antacids; discuss sources. Iron supplementation is best taken 2 hours after consuming these substances.
- The average American diet contains 10–20 mg of iron daily, roughly 10% of which is absorbed. Avoid overdosing with iron supplements. The body can only synthesize 5–10 mg of Hgb per day, and excesses may work against the immune system.
- Local or systemic infections interfere with iron absorption and transport.
- In children under age 2, limit milk intake to no more than 500 mL/d for better iron status.
- Explain nonfood pica—clay, starch, plaster, paint chips—and the relationship with nutrition. In food pica in which singular foods are eaten instead of balanced meals, the foods chosen are often crunchy or brittle. Excessive consumption of lettuce, ice, celery, snack chips, and chocolate has been noted; after iron supplementation, cravings often subside.
- Iron deficiency may be partly induced by plant-based diets containing low levels of poorly bioavailable iron (Kesa and Oldewage-Theron, 2005). Young people who follow a vegan diet should have their iron status monitored closely.
- Use culturally appropriate nutrition counseling. In some cultures, boys may be fed iron-rich foods preferentially over girls; counseling should be designed to improve intake by girls (Shell-Duncan and McDade, 2005).

Patient Education—Food Safety

If tube feeding or CPN is needed, careful handwashing procedures should be followed.

For More Information

- E-medicine
  http://emedicine.medscape.com/article/202333-overview
- Iron Deficiency Anemia for Kids
  http://www.kidshealth.org/parent/medical/heart/ida.html
- Mayo Clinic
  http://www.mayoclinic.com/health/iron-deficiency-anemia/DS00323
- National Institutes of Health–Iron Deficiency Anemia
  http://www.nlm.nih.gov/medlineplus/ency/article/000584.htm
- University of Maryland
  http://www.umm.edu/blood/aneiron.htm

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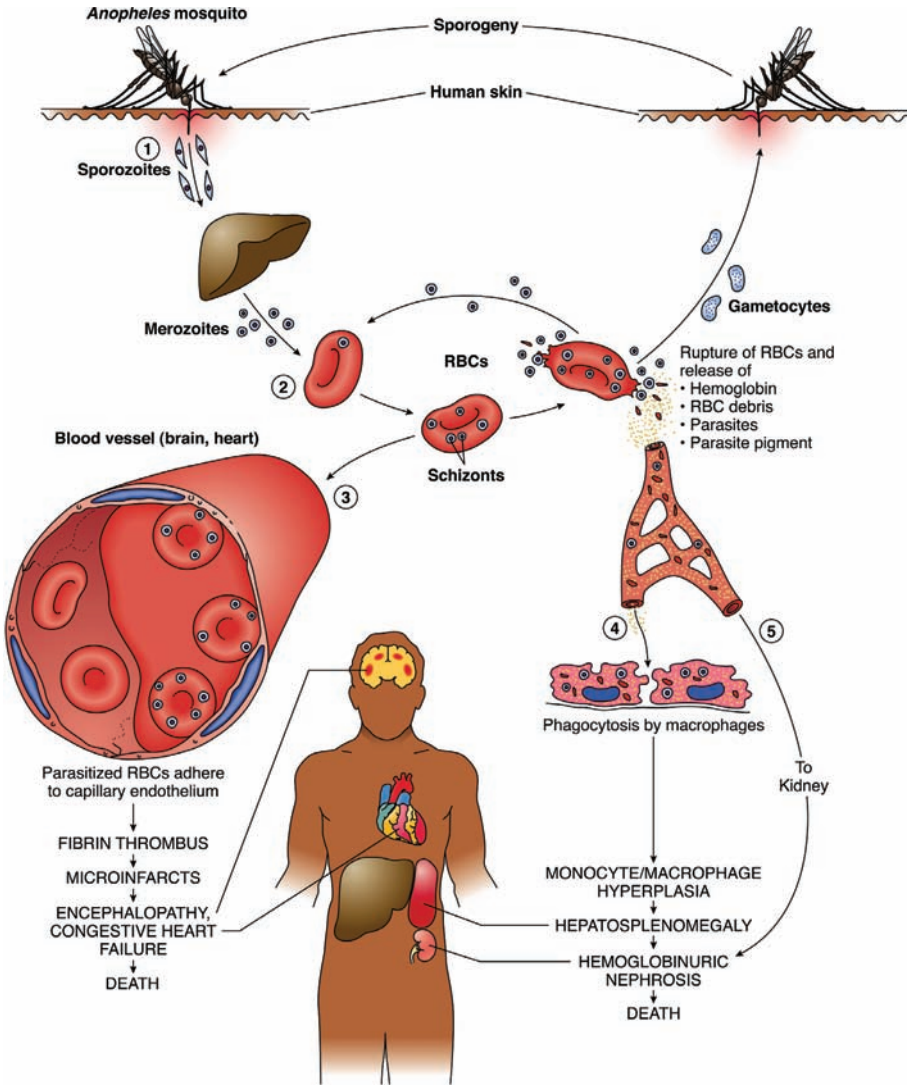
MALARIA AND PARASITIC ANEMIAS

NUTRITIONAL ACUITY RANKING: LEVEL 1



Adapted from: Rubin E MD and Farber JL MD. *Pathology,* 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 1999.

DEFINITIONS AND BACKGROUND

Gastrointestinal infestation by parasitic worms that feed on blood (hookworm) or on nutrients (tapeworm) may occur, especially in tropical or subtropical areas. Intestinal parasites that affect nutrition in particular include soil-transmitted helminths, *Giardia duodenalis*, *Entamoeba histolytica*, other parasites such as the coccidia, *Schistosoma* sp., and malarial parasites. Iron deficiency is found to be correlated with these parasites in tropical countries (Shell-Duncan and McDade, 2005).

The neglected infections of poverty are parasitic, bacterial, and viral infections that disproportionately affect impoverished populations in the United States; these include Chagas disease, cysticercosis, congenital cytomegalovirus (CMV), toxocariasis, toxoplasmosis, and trichomoniasis (CDC, 2009). Toxoplasmosis is considered to be the third leading cause of death attributed to foodborne illness in the United States (CDC, 2009).

Malaria is a bloodborne, parasitic infection transmitted by mosquitoes. It kills more than 1 million people annually, especially in Africa. Pregnant women, children, and immunocompromised individuals have the highest rates of morbidity and mortality (Schantz-Dunn and Nour, 2009). More than 1000 cases of malaria are reported to the Centers for Disease Control and Prevention each year in the United States. Travelers or immigrants present with fever, chills, nausea, vomiting, headache, abdominal pain, severe anemia, and acute renal failure (Vicas et al, 2005).

Risks for miscarriage, intrauterine demise, premature delivery, low birth-weight neonates, neonatal death, severe anemia, and maternal death are high among pregnant women with malaria (Schantz-Dunn and Nour, 2009). Between 150 and 300 children die each hour from malaria (Breman, 2009). For those who survive, neurologic impairment, anemia, hypoglycemia, and low birth weight imperil normal development and survival (Breman, 2009).

Malaria can be prevented with appropriate drugs, bednets treated with insecticide, and effective educational outreach. Resistance of *Plasmodium falciparum* to drugs and Anopheles mosquitoes to insecticides has stimulated discovery and development of artemisinin-based combination treatments (ACTs) and other drugs, long-lasting insecticide-treated bednets (with synthetic pyrethroids) and a search for nontoxic, long-lasting, affordable insecticides for indoor residual spraying (Breman, 2009). A malaria vaccine is under development (Greenwood, 2008).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Congenital CMV is passed from mother to infant during pregnancy.

Clinical/History

- Height
- Weight
- BMI
- Diet history
- BP
- I & O
- Fatigue
- Abdominal discomfort
- Nausea, vomiting
- Fever
- Irritability
- Pica?

Lab Work

- Hgb (often <5 g/dL)
- Hct
- Serum Fe
- Ferritin
- Serum B_{12}
- TIBC
- Alb
- Serum folic acid
- CRP
- Transferrin
- Gluc
- Serum Cu
- Serum Zn

INTERVENTION

OBJECTIVES

- Correct anemia from blood losses; eliminate parasitic infestation.
- Prevent GI tract perforation or obstruction, when likely to exist.
- Improve nutritional status and appetite. Parasitic infections affect the intake of food, subsequent digestion and absorption, metabolism, and nutrient storage and cause subtle micronutrient deficiency, such as vitamin A deficiency.
- Prevent low birth weight and other adverse effects in pregnant or postpartum women and their infants.

FOOD AND NUTRITION

- A diet high in protein, B-complex vitamins, and iron may be appropriate. Provide adequate energy to meet individual's needs for anabolism where needed.
- Foods rich in heme iron and vitamins C and A should be included in meals served or planned. Iron inhibitors should be excluded from diet as far as possible until recovery is complete.
- Include plenty of other nutrient-dense foods, such as good sources of zinc and other micronutrients (Table 12-15).

SAMPLE NUTRITION CARE PROCESS STEPS

Abnormal GI Function

Assessment Data: Weight loss, GI distress, Hgb <5, temperature averaging 101 degrees F. Dx of parasitic infection *Giardia* from drinking water at a campsite.

Nutrition Diagnoses (PES): Abnormal GI function related to parasitic infection of *Giardia* as evidenced by fever, low Hgb <5, GI distress, and weight loss.

Interventions: Food–Nutrient delivery—offer tolerated foods and beverages. Suggest use of a multivitamin-mineral supplement. Educate about risks of drinking potentially contaminated water from various sources.

Monitoring and Evaluation: Improvement in Hgb level; return of lost weight. Resolution of *Giardia*.

TABLE 12-15 Micronutrient Deficiencies in Parasitic Anemias such as Malaria

Micronutrient	Deficiency Effects
Vitamin A	Increased susceptibility to malarial anemia, altered iron metabolism, deficit of retinol for synthesis of acute phase reactants
Vitamin C	Impaired T-lymphocyte response, delayed cutaneous hypersensitivity, impaired complement function, reduced phagocytic function
Vitamin E	Impaired T-lymphocyte response, altered B-cell function and impaired humoral response, delayed cutaneous hypersensitivity, impaired cytokine function or production, reduced phagocytic function; deficiency can contribute to oxidant damage to erythrocytes, leading to hemolysis, but deficiency can make the parasite more vulnerable to oxidation generated with some antimalarial drugs
Riboflavin	Decreased iron absorption, increased erythrocyte fragility, depressed erythropoiesis; deficiency may protect against malaria by diminished parasite multiplication and growth
Folate	Impaired erythropoiesis; deficiency may protect against malaria through impaired parasite metabolism
Copper	Involvement in acute phase response to infection
Iron	Impaired erythropoiesis, decreased T-lymphocyte response, altered B-cell function and impaired humoral response, delayed cutaneous hypersensitivity, impaired cytokine function or production, reduced phagocytic function; deficiency and associated microcytosis may reduce malaria parasite multiplication. Avoid excesses.
Selenium	Unknown role
Zinc	Impaired immune function including decreased T-lymphocyte response, altered B-cell function and impaired humoral response, delayed cutaneous hypersensitivity, impaired cytokine function or production, reduced phagocytic function; can contribute to increased parasitemia

From: Nussenblatt V, Semba, RD. Micronutrient malnutrition and the pathogenesis of malarial anemia. *Acta Trop.* 82:321, 2002; and Scrimshaw NS, Sangiovanni JP. Synergism of nutrition, infection, and immunity: an overview. *Am J Clin Nutr.* 66:464S, 1997.

Common Drugs Used and Potential Side Effects

- Artemisinin combination therapy replaces ineffective chloroquine and sulphadoxine pyrimethamine for first-line treatment of malaria and for the provision of long-lasting, insecticide treated bednets (Greenwood, 2008).
- If needed, oral or parenteral iron may be given to correct anemia more rapidly. Beware of excessive use of oral supplements because of their potential side effects with iron overloading; monitor all sources (including iron-enriched foods).

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss ways to prevent further parasitic infestations, as with small children playing in soil. Pregnant women must be particularly careful, especially in developing countries where malaria, hookworm, and other parasites are common.
- Discuss ways to prepare foods high in necessary nutrients and methods to increase bioavailability (e.g., combining orange juice at breakfast with an iron-fortified cereal, etc.). In vulnerable populations, use fortified beverages to correct micronutrient deficiency.
- In areas of malaria transmission, anemia is apparent from the first few months of life, and there is a great need to target interventions at pregnant women and infants, which are the groups at highest risk. Food-fortification programs can be very beneficial.

Patient Education—Food Safety

- Avoid eating raw fish or meats.
- Washing vegetables thoroughly before eating them and cooking meat to recommended temperatures to avoid toxoplasmosis (CDC, 2009).
- If tube feeding or CPN is needed, careful handwashing procedures should be followed.
- To prevent the spread of CMV, do not share food, drinks, or eating utensils with young children. Wash hands with soap and water after touching diapers or saliva.

For More Information

- Parasites of the Intestinal Tract
  http://www.dpd.cdc.gov/dpdx/HTML/Para_Health.htm
- Parasitic Disorders
  http://www.oas.org/osde/publications/Unit/oea37e/ch10.htm
- World Health Organization–Malaria
  http://www.who.int/tdr/diseases/malaria/mim.htm

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Megaloblastic Anemias: Pernicious or Vitamin B_{12} Deficiency

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

Megaloblastic anemias affect the nervous system if left untreated. Deficiency of folate and vitamin B_{12} changes in the concentrations of metabolites, such as methylmalonic acid and homocysteine.

Pernicious anemia (PA) is a macrocytic anemia caused by a vitamin B_{12} deficiency and intrinsic factor (IF). PA is thought to be an autoimmune disorder and is often found with other disorders such as thyroid or diabetes. The antiparietal cell antibodies test measures the presence of antibodies against gastric parietal cells. Long-standing *Helicobacter pylori* infection probably plays a role with the irreversible destruction of the gastric body mucosa (Lahner and Annibale, 2009).

The three forms of PA are congenital PA, juvenile PA, and adult-onset PA. The forms are based on the age at onset and the precise nature of the defect causing impaired vitamin B_{12} utilization (e.g., absence of IF). PA affects 1–2% of older individuals. Defective RBC production occurs, caused by a lack of IF of the stomach. IF helps vitamin B_{12} produce RBCs. IF is present in gastric juice and binds to B_{12}. Once bound, IF changes and becomes less susceptible to digestion; this protects B_{12} and allows its absorption from gastric juice. When IF is not available, B_{12} cannot be properly absorbed. Vitamin B_{12} deficiency and achlorhydria have a detrimental effect on bone strength. Having PA increases the risk for hip fracture, even after treatment (Merryman et al, 2009).

Vitamin B_{12}-deficiency anemia may take 5–6 years to appear; this megaloblastic anemia is a reversible form of ineffective hematopoiesis. There may be almost 800,000 older adults in the United States who have undiagnosed and untreated vitamin B_{12} deficiency. It is often masked by high folate intakes. Hidden blood loss, gastric atrophy, and poor dietary intake should be addressed (Table 12-16).

The Schilling test is no longer used. Serologic testing for parietal cell and IF antibodies are used instead. With normal absorption, the ileum absorbs more vitamin B_{12} than the body needs and excretes excess into the urine. With impaired absorption, however, little or no vitamin B_{12} is excreted into the urine. Low serum levels cannot identify all cases of vitamin B_{12} deficiency; serum methylmalonic acid level may also be needed. Holotranscobalamin (holoTC), when compared with the other markers of vitamin B_{12} deficiency, shows promise for diagnosing early vitamin B_{12} deficiency (Hvas and Nexo, 2005).

Adequate selenium intake plays an important role in maintaining vitamin B_{12} adequacy. Glutathione forms a complex called glutathionylcobalamin, which could protect against diseases related to vitamin B_{12} depletion.

Areas of research include intermittent vitamin B_{12} supplement dosing and better measurements of the bioavailability of vitamin B_{12} from fermented vegetarian foods and algae. Vegetarians are at risk for vitamin B_{12} deficiency (Allen, 2008). Vitamin B_{12} anemia is corrected by the use of oral cyanocobalamin.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Human leukocyte antigen-DR genotypes suggest a role for genetic susceptibility in PA (Lahner and Annibale, 2009). In congenital PA, IF is missing. Nearly all people with PA test positive for antiparietal cell antibodies.

Clinical/History		
Height	Postural hypotension?	Diarrhea
Weight	Fatigue	Constipation
BMI	Flatulence, nausea, and vomiting	Anorexia
Diet history		Tachycardia, cardiomegaly
Weight loss?		Achlorhydria

TABLE 12-16 Risks and Causes of Pernicious Anemia or Vitamin B_{12}–Deficiency Anemia

Pernicious Anemia	Vitamin B_{12}–Deficiency Anemia
High risk:	High risk:
Family history of pernicious anemia; African, Scandinavian, or Northern European descent; autoimmune endocrine disorders	Vegans; elderly; persons with intestinal malabsorption or gastric atrophy or hypochlorhydria; stomach removal surgery; drug use (colchicine, neomycin); metabolic disorders (homocystinuria methylmalonic aciduria); breastfed infants of vitamin B_1–deficient mothers; poor diet in infancy or pregnancy
Causes:	Causes:
Autoimmune endocrine diseases such as type 1 diabetes, hypoparathyroidism, Addison's disease, hypopituitarism, testicular dysfunction, Graves' disease, chronic thyroiditis, myasthenia gravis, secondary amenorrhea, vitiligo, gastric surgery, anorexia nervosa, or bulimia nervosa	Poor intake of extrinsic factor (vitamin B_{12}). Chronic alcoholism. *Helicobacter pylori* infection with diminished production of intrinsic factor. Hidden blood loss. Fish tapeworm. Reduced intestinal absorption, as with celiac disease or Crohn's disease

- Glossitis with beefy, red tongue
- Lemon yellow or waxy skin
- Numbness or tingling in hands and feet
- Impaired sense of smell

- MCV, MCHC, MCH (increased)
- Macrocytic/nucleated cells
- Reticulocyte count (low)
- Hct (low)
- Lactate dehydrogenase (LDH) (increased)

- TIBC
- Urinary methylmalonic acid
- Serum folate
- Serum homocysteine (elevated?)
- Holotranscobalamin (holoTC)
- Bilirubin
- Transferrin

Lab Work

- Parietal cell and IF antibodies
- RBCs
- Serum B_{12}
- CBC (altered platelets and WBC count)
- Gastrin (increased)

INTERVENTION

OBJECTIVES

- Alleviate the etiology of anemia, where possible.
- Provide foods that will not hurt a sore mouth. Glossitis decreases the desire to eat.
- Correct patient's anorexia.
- Prevent neurological defects if treatment is delayed or insufficient; depression, psychosis, and mania can appear.
- Where present, correct PA; prevent progression to gastric cancer. PA requires vitamin B_{12} injections.
- Prevent or correct hyperhomocysteinemia.

FOOD AND NUTRITION

- Diet should make liberal use of high biological value (HBV) proteins. Good sources of vitamin B_{12} include liver, other meats, fish, poultry, eggs, milk, cheese, yogurt, fortified products such as cereals, and soy milk. The daily average intake is 2–30 mg.
- Supplement diet with iron, vitamin C, folic acid and other B vitamins, copper, and selenium.
- If patient has a sore mouth, use a soft or liquid diet with fewer spicy or acidic foods.

SAMPLE NUTRITION CARE PROCESS STEPS

Abnormal Nutrition Laboratories

Assessment Data: PA with low serum vitamin B_{12}, Hgb, and Hct; elderly resident in long-term care facility.

Nutrition Diagnoses (PES): Altered nutrition-related laboratories related to resident's body inability to produce IF and to absorb cobalamin as evidence by abnormal vitamin B_{12} <200 pg/mL, Hgb <12 gm/dL, and Hct <37%.

Interventions: Food and nutrient delivery—provide resident with foods/beverages with vitamin C such as orange juice, citrus fruits, and melon to be consumed at every meal to aid with iron absorption. Resident will eat two scrambled eggs at breakfast to increase vitamin B_{12}. Coordinate care with nursing and medical staff to administer vitamin B_{12} shots and support with adequate dietary intake.

Monitoring and Evaluation: Monitor monthly laboratories; goal vitamin B_{12} >200 pg/mL, Hgb >12, and Hct >38%.

Common Drugs Used and Potential Side Effects

- Vitamin B_{12} deficiency is effectively treated with oral vitamin B_{12} supplementation. Crystamine or Rubramin PC is cyanocobalamin in drug form for vitamin B_{12} deficiency.
- For PA, vitamin B_{12} injections are given weekly until remission, after which six to eight injections yearly will suffice. For some, vitamin B_{12} supplements may be as effective as injections; high doses, such as 1000 μg, are needed daily for about 18 months.
- Trinsicon contains vitamin B_{12}, ferrous fumarate, vitamin C, folacin, and IF. It is less effective when taken with dairy products.
- Some medications that conflict with vitamin B_{12} absorption include chloramphenicol, proton pump inhibitors, histamine 2 inhibitors, and metformin.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- True vegan diets do not contain vitamin B_{12}; it is found only in animal foods. Include eggs, meat, fish, shellfish, cheese, milk, milk products, or use B_{12} fortified soy products. Some fad diets may also be low in vitamins and protein; monitor intake carefully.
- PA develops after total gastrectomy unless vitamin B_{12} is administered. The problem may also occur in patients with partial gastrectomy or gastric bypass. Lifelong vitamin B_{12} replacement or injections are necessary.
- Avoid fatigue; plan simple meals and snacks.
- Megaloblastic B_{12} anemia may occur in elderly; careful food choices are essential.
- Breastfed infants of vitamin B_{12}–deficient mothers are at risk for severe developmental abnormalities, growth failure, and anemia. Counseling for lactating women is important.

Patient Education—Food Safety

If tube feeding or CPN is needed, careful handwashing procedures should be followed.

For More Information

- Medline–Pernicious Anemia
  http://www.nlm.nih.gov/medlineplus/ency/article/000569.htm
- Pernicious Anemia
  http://www.med.unc.edu/medicine/web/perniciousanemia.htm

- Vitamin B_{12} Deficiency Anemia
  http://www.nlm.nih.gov/medlineplus/ency/article/000574.htm

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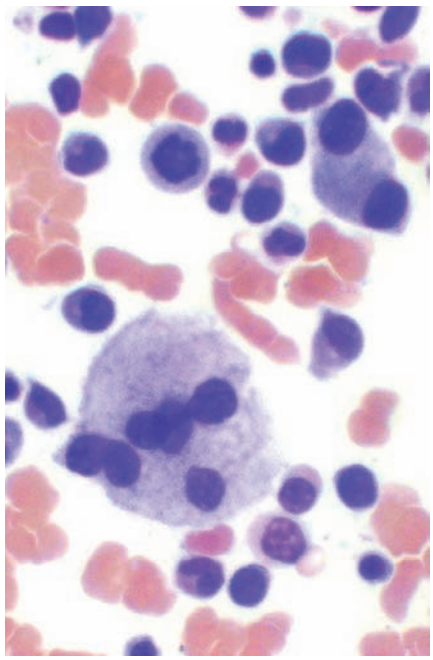
SIDEROBLASTIC ANEMIA

NUTRITIONAL ACUITY RANKING: LEVEL 1



Adapted from: Raphael Rubin, David S. Strayer, *Rubin's Pathology: Clinicopathologic Foundations of Medicine,* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2008.

DEFINITIONS AND BACKGROUND

Sideroblastic anemias are a group of blood disorders characterized by an impaired ability of the bone marrow to produce normal RBCs. Inherited sideroblastic anemia comprises several rare anemias due to heterogeneous genetic lesions, all characterized by the presence of ringed sideroblasts in the bone marrow (Camaschella, 2009).

Abnormal RBCs called sideroblasts are found in the blood of people with these anemias. This anemia is a microcytic, hypochromic anemia similar to that caused by iron deficiency, except that serum iron is normal or elevated. The iron inside RBCs is inadequately used to make Hgb, despite adequate or increased amounts of iron. Therapy comprises application of antioxidants, vitamins, iron, bone marrow-stimulating factors, or substitution of cells (Finsterer, 2007).

The disease X-linked sideroblastic anemia with ataxia is due to a mutation in the protein transporter that is thought to transfer iron clusters from the mitochondrion to the cytoplasm (Napier et al, 2005). Another name for the congenital type of anemia is hereditary iron-loading anemia.

X-linked sideroblastic anemia (delta-aminolevulinate synthase deficiency) is vitamin B_6 responsive that responds to high pyridoxine doses (Clayton, 2006). Pyridoxal phosphate is the cofactor for over 100 enzyme-catalyzed reactions in the body. Vitamin B_6 is the main vitamin for processing amino acids and is also needed to make melatonin, serotonin, and dopamine.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Mutations in the mitochondrial carrier family gene SLC25A38 cause nonsyndromic autosomal recessive congenital sideroblastic anemia (Petkau, 2009). The enzyme directly relevant to sideroblastic anemia is ALAS-2 or ALAS-e on the X chromosome. However, most sideroblastic anemias are acquired.

Clinical/History

- Height
- Weight
- BMI
- Diet history
- BP
- I & O
- Alcohol or drug toxicity?
- Fatigue
- Dizziness
- Decreased tolerance for exercise

Lab Work

- Serum B_6 levels
- Hgb (low at 4–10 g/dL)
- RBC
- Transferrin saturation (often elevated)
- Serum folic acid
- Serum homocysteine
- WBC
- Serum Fe, ferritin
- Serum Cu

INTERVENTION

OBJECTIVES

- Identify causes and solutions.
- Remove any precipitating factors, such as specific drugs or alcohol use.
- Correct problems, such as suppression of bone marrow, iron loading, and anemia.

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Vitamin Intake

Assessment Data: Diagnosis of sideroblastic anemia, pyridoxine-responsive. BMI normal. Complaints of fatigue and poor exercise tolerance. Diet indicates intake of B_6-rich foods to be infrequent. Lab work with high transferrin saturation, Hgb of 6, and low serum B_6 levels.

Nutrition Diagnoses (PES): Inadequate vitamin intake (B_6) related to X-linked genetic defect and low dietary intake as evidenced by diet and medical histories.

Interventions: Trial of 100- to 200-mg pyridoxine by mouth. Counseling about foods rich in vitamin B_6.

Monitoring and Evaluation: Improvement in symptoms (less fatigue and better exercise tolerance). Lab work showing improvement in serum B_6 and Hgb, and lower transferrin saturation levels.

FOOD AND NUTRITION

- A diet high in vitamin B_6 may be beneficial with medication. Potatoes, bananas, raisin bran cereal, lentils, liver, turkey, and tuna are good sources of vitamin B_6.
- Protein and carbohydrate (CHO) intake should be adequate, and energy should also be adequate to spare protein. Folic acid and copper may also be needed.
- Alcohol intake should be severely limited.
- Balanced meals and snacks, as necessary, may be helpful.

Common Drugs Used and Potential Side Effects

- Vitamin B_6 may be ordered; age-dependent doses are specified. The National Academy of Sciences performed an analysis of vitamin B_6 studies. It is usually safe at intakes of up to 100 mg/d in adults, but neurological side effects can sometimes occur at or above that level. Vitamin B_6 toxicity damages sensory nerves, leading to numbness in the hands and feet as well as difficulty walking.
- Chloramphenicol may cause drug-induced bone marrow suppression, resulting in sideroblastic anemia. Isoniazid, busulfan, penicillamine, and cycloserine can cause abnormal vitamin B_6 metabolism. Monitor for gastrointestinal side effects.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss adequate sources of all needed nutrients such as vitamin B_6, especially if deficiency caused the anemia.
- Discuss attractive menu planning and balancing of meals because appetite and intake may be poor chronically. Discuss snacks and frequency.

Patient Education—Food Safety

If tube feeding or CPN is needed, careful handwashing procedures should be followed.

For More Information

- Genetics Home Reference
  http://ghr.nlm.nih.gov/ghr/resource/health
- Harvard
  http://sickle.bwh.harvard.edu/sideroblastic.html
- Medline Plus: Anemia
  http://www.nlm.nih.gov/medlineplus/anemia.html

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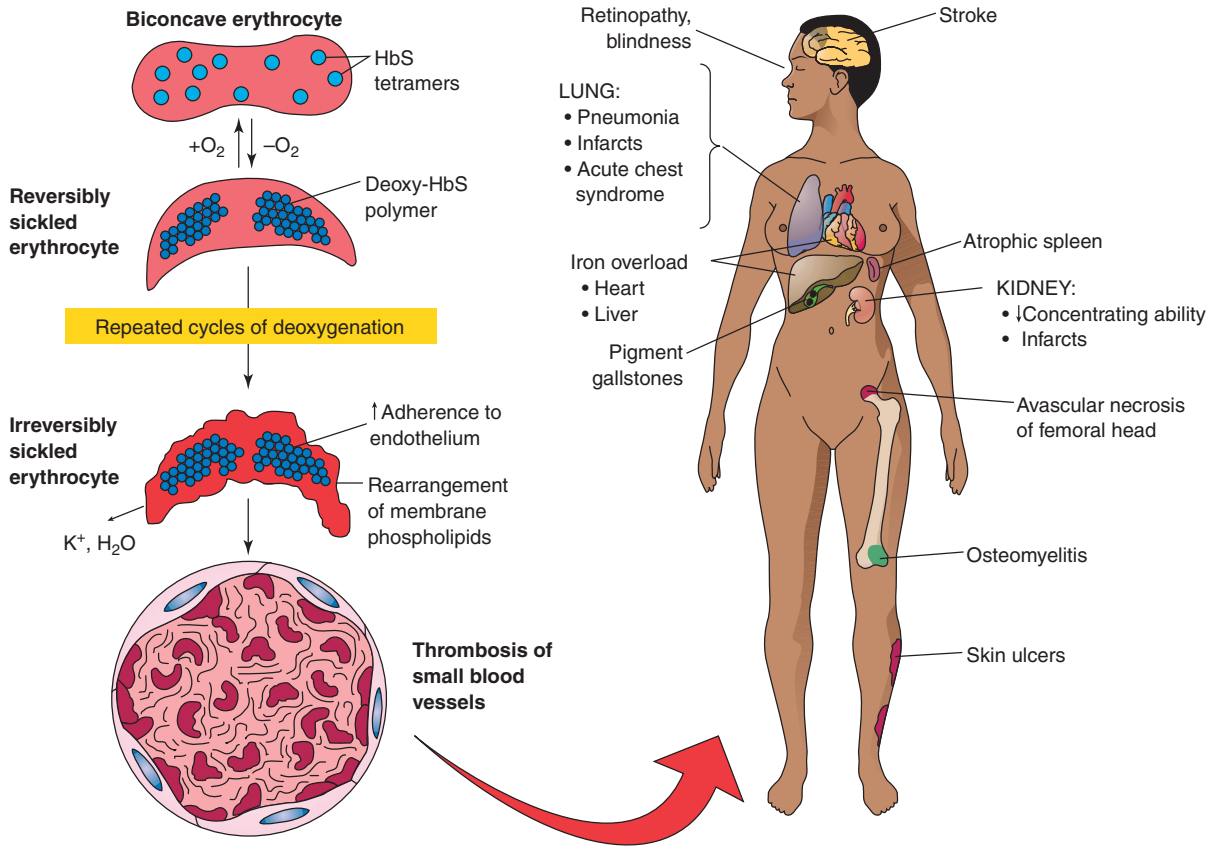
HEMOGLOBINOPATHIES

SICKLE CELL ANEMIA

NUTRITIONAL ACUITY RANKING: LEVEL 1



Adapted from: Raphael Rubin, David S. Strayer, *Rubin's Pathology: Clinicopathologic Foundations of Medicine,* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2008.

DEFINITIONS AND BACKGROUND

Sickle cell disease (SCD) is the most common genetic disorder of the blood. SCD involves anemia that is hereditary and hemolytic. Cells in SCD are crescent shaped and become rigid; they lodge themselves in the capillaries of the peripheral-blood system outside the heart. The sickling of RBCs occurs when partially or totally deoxygenated Hgb molecules distort their normal disk shape, producing stiff, sticky, sickle-shaped cells that obstruct small blood vessels; this causes vaso-occlusion as well as deprivation of oxygen to body tissues (Edwards et al, 2005). Everyone with SCD has chronic hemolytic anemia, vasculopathy, vaso-occlusive disease, acute and chronic organ damage, and shortened life span (Steinberg, 2008).

SCD has several forms including sickle cell anemia, sickle cell Hgb C disease, and sickle cell thalassemia disease. It is usually detected within the first year of life. Routine use of daily antibiotics until 5 years of age, immunization of children with pneumococcal vaccine, annual influenza vaccination after 6 months of age, and meningococcal vaccination after 2 years of age are important preventive measures (Mehta et al, 2006).

The largest population in the world with sickle cell anemia is in Africa. While this condition most commonly affects blacks of African descent, it is also found in people of Middle Eastern, East Indian, and Mediterranean origin. About 100,000 Americans have SCD (~1 in every 400–500 African–Americans). Carrier frequency varies, with high rates associated with zones of high malaria incidence. Carriers are often protected against malaria.

Patients with SCD are at risk for delayed growth and sexual maturation; acute and chronic pulmonary dysfunction; stroke; aseptic necrosis of the hip, shoulders, or both; sickle cell retinopathy; dermal ulcers; and severe chronic pain (Edwards et al, 2005). The homozygous state (SS) is associated with complications and a reduced life expectancy.

Chronic anemia, pallor, and jaundice result because sickled cells do not last as long as normal blood cells. Bone marrow functions at six times the normal rate. Because there are fewer cells, the blood is thinner or anemic. When RBCs are destroyed, bilirubin is released into the blood and turns the whites of the eyes to a shade of yellow.

Inadequate dietary intakes of folate are common, whereas vitamin B_{12} intakes are usually adequate. Low RBC folate levels may occur. Serum total homocysteine (tHcy) levels may be elevated in this population; greater intakes than normal of folate may be needed. Elevated tHcy levels contribute to

thrombosis, a frequent event in this population. Children with sickle cell anemia have lower vitamin B_6 concentrations.

Infants and children who have SCD are at risk for nutritional deficiencies and loss of body mass during acute illness. Suboptimal vitamin A intake is common, with more frequent hospitalizations and poor growth. Low serum vitamin D status is highly prevalent in children with SCD; vitamin D status is associated with season and dietary intake. Prepubertal children with SCD may have zinc deficiency and may benefit from zinc supplementation.

There is an underlying defect in lipid metabolism associated with SCD, manifested during the fasting state; this abnormality in lipid homeostasis has the potential to alter RBC membrane fluidity and function in SCD patients (Buchowski et al, 2007). Individuals with SCD have reduced levels of EPA and DHA in red cells, platelets, and mononuclear cells due to peroxidation from compromised antioxidant competence (Ren et al, 2008). Because dietary omega-3 fatty acids reduce prothrombotic activity, include omega-3 fatty acids in diet and in supplemental form.

Cellular and tissue damage is caused by hypoxia, oxidant damage, inflammation, abnormal intracellular interactions, and reduced nitric oxide bioavailability (Steinberg, 2008). Young children with SCD are at a very high risk of stroke, with microvascular occlusion and painful episodes (Adams, 2007).

Aggressive antibiotic therapy and transfusions can save lives. Transfusion is indicated for symptomatic anemia and specifically to prevent stroke, during acute stroke, and for acute chest syndrome (Roseff, 2009). Although life saving, transfusion therapy has resulted in the majority of sickle cell anemia patients being at risk for iron overloading and hemosiderosis-induced organ damage (Vichinsky et al, 2005). Iron overload has become easier to manage with the introduction of an oral iron chelator (Adams, 2007).

Acute chest syndrome, triggered by infections and fat clots in the lungs, is the leading cause of death in sickle cell anemia. Treatment includes hydroxyurea therapy to decrease the frequency of painful episodes and hematopoietic cell transplantation (Mehta et al, 2006). Bone marrow transplantation requires a perfect match from a sibling. Because patients with SCD have problems with surgery, including prolonged bleeding, vitamin K should be given preoperatively (Raffini et al, 2006). Use of transcranial Doppler ultrasonography helps identify asymptomatic, at-risk children who should be considered for chronic blood transfusions (Mehta et al, 2006).

Studies of gene expression are bringing new solutions. Human progenitor cell (from bone marrow, peripheral blood stem cells, or umbilical blood) transplant can cure the disease and is used for patients with severe disease for whom conventional therapy may not be effective (Roseff, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Sickle cell anemia is an autosomal recessive disease caused by a mutation in the *hemoglobin beta gene* (*HBB*) found on chromosome 11p15.5. Genetic studies have identified regions on chromosome 6q23 and BCL11 A on chromosome 2p16 that account for 20–50% of the common variation in fetal Hgb levels in patients with sickle cell anemia (Thein et al, 2009). SNPs have also been found in KCNK6 (Sebastiani et al, 2009).

Clinical/History	Lab Work	
Height	CBC, WBC	Serum creatinine
Weight	Sickle cell test	N balance
BMI	Hgb (often low)	Alb
Diet history	Hct	CRP
BP	Serum Fe (increased from hemolysis)	Cholesterol
I & O	RBP (decreased)	Triglycerides (Trig) (decreased)
Chronic anemia, pallor	Bilirubin	MCV
Jaundice	tHcy (often elevated)	Serum ferritin
Bone pain	Transferrin saturation	Partial pressure of oxygen (pO_2)
Abdominal pain	Serum folic acid and B_{12}	Partial pressure of carbon dioxide (pCO_2)
Breathlessness	Serum and urinary zinc	Uric acid (increased)
Lower leg ulcers		PT and INR
Casts or blood in urine		
Excessive thirst		
CT scan or MRI		

INTERVENTION

OBJECTIVES

- Supplement diet with missing nutrients. Correct any malnutrition.
- Reduce oxygen debt and hemolytic crises.
- Reduce painful cramps, liver dysfunction, cholelithiasis, jaundice, and hepatitis.
- Lessen likelihood of pressure ulcers, infections, and renal failure. Infections may include pneumonia, cholecystitis, osteomyelitis, or urinary tract infections.

SAMPLE NUTRITION CARE PROCESS STEPS

Involuntary Weight Loss

Assessment Data: Weight pattern, percent desirable body weight, diet history, problems with meal planning or shopping, financial challenges.

Nutrition Diagnosis (PES): Involuntary weight loss related to sick cell anemia with inadequate caloric intake as evidenced by 10% loss of usual body weight in the last 2 months.

Intervention: Nutrition counseling, encouraging energy-dense foods and favorites. Coordination of care with referral to social service agencies for help with meal preparation and delivery.

Monitoring and Evaluation: Weight records, improvements in appetite and intake.

TABLE 12-17 Equation to Predict Energy Needs in Adolescents with Sickle Cell Disease

Basal energy requirements are higher in adolescents with sickle cell anemia than in healthy control subjects (Buchowski et al, 2002)

Males: REE (kcal/d) = 1305 + 18.6 × weight (kg) − 55.7. hemoglobin (g/dL)

REE (kJ/d) = 5461 + 77.7 × weight (kg) − 233.2 × hemoglobin (g/dL)

Females: REE (kcal/d) = 1100 + 13.3 × weight (kg) − 30.2 × hemoglobin (g/dL)

REE (kJ/d) = 4603 + 55.6 × weight (kg) − 126.2 × hemoglobin (g/dL)

- Maintain adequate hydration.
- Promote normal growth and development, which tend to be stunted in children.
- Prevent chronic hypoxia, which can lead to lower intellectual performance.
- Improve quality of life and ability to participate in the activities of daily life.

FOOD AND NUTRITION

- Include food sources of omega-3 fatty acids; vitamins D, C, A, B_{12}, and B_6; folic acid; and HBV proteins; ensure adequate zinc and riboflavin.
- Estimate fluid and energy needs; increase diet as needed (Table 12-17).
- A multivitamin–mineral supplement should be recommended; one without excess iron is important when transfusions are used. Avoid excesses of iron, including from tube feedings or parenteral nutrition.
- Energy deficits are common in this population. Nightly tube feeding can help to improve nutritional status. While supplementation with arginine has been suggested, more studies are needed.

Common Drugs Used and Potential Side Effects

- Pain medicines (such as ibuprofen) may be used. Monitor for all side effects and GI distress.
- Hydroxyurea therapy (Droxia, Hydrea) can be used to increase Hgb production.
- Rofecoxib is a cyclo-oxygenase-2 (COX-2) inhibitor approved for pain and has been tested in children with no adverse effects.
- Rituximab may be used to prevent delayed hemolytic transfusion reaction disorder in SCD.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- A phytomedicine, Niprisan, may reduce episodes of SCD crisis associated with severe pain.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Indicate which foods are good sources of folic acid, HBV proteins, zinc, riboflavin, and vitamins A, C, D, E, B_6, and B_{12}.
- Discuss ways for easy meal preparation because fatigue tends to be a problem.
- Quality of life is often decreased among adults with SCD, and health professionals should try to offer assistance that will help improve this quality (McClish et al, 2005).

Patient Education—Food Safety

If tube feeding or CPN is needed, careful handwashing procedures should be followed.

For More Information

- American Sickle Cell Association
  http://www.ascaa.org/
- National Institutes of Health (NIH)–Genes and Disease
  http://www.ncbi.nlm.nih.gov/disease/sickle.html
- NIH–Sickle Cell Anemia
  http://www.nlm.nih.gov/medlineplus/sicklecellanemia.html

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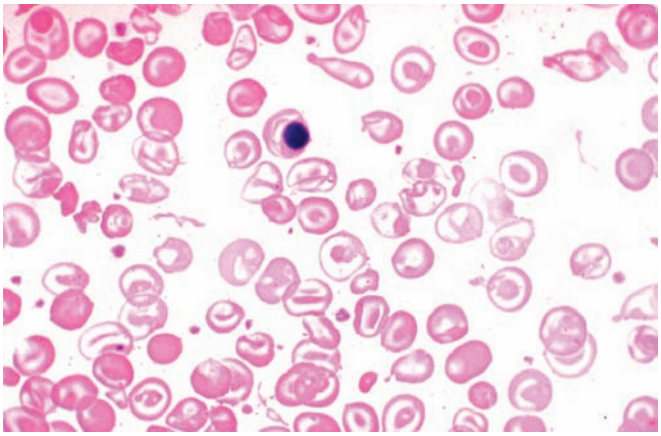
THALASSEMIAS

NUTRITIONAL ACUITY RANKING: LEVEL 1



Adapted from: Raphael Rubin, David S. Strayer, *Rubin's Pathology: Clinicopathologic Foundations of Medicine,* 5th ed. Philadelphia: Lippincott Williams & Wilkins, 2008.

DEFINITIONS AND BACKGROUND

The thalassemias are inherited hematologic disorders caused by defects in the synthesis of one or more of the Hgb chains. Alpha thalassemia is caused by reduced or absent synthesis of alpha globin chains, and beta thalassemia is caused by reduced or absent synthesis of beta globin chains (Muncie and Campbell, 2009). Hemolysis and impaired erythropoiesis occur.

Collectively, the thalassemias are among the most common inherited disorders. Alpha-thalassemic syndromes have an increased frequency in African, American Indian, and Asian populations. Beta thalassemia is well recognized in persons of Greek and Italian descent; Cooley's or Mediterranean anemia is most severe. The beta thalassemias are more common and are a worldwide clinical problem due to an increasing immigrant population (Hahalis et al, 2005).

Silent carriers of alpha thalassemia and persons with alpha or beta thalassemia trait are asymptomatic and require no treatment. Alpha thalassemia intermedia (Hgb H disease) causes hemolytic anemia (Muncie and Campbell, 2009). The RBCs are fragile and contain abnormal Hgb.

Beta thalassemia major causes hemolytic anemia, poor growth, and skeletal abnormalities during infancy. Symptoms can begin as early as 3 months of age. In the first year or two of life and in the absence of transfusion, a child can demonstrate severe anemia and expansion of the facial and other bones. These children may be pale or jaundiced, have a poor appetite, fail to grow normally, and have an enlarged spleen, liver, or heart. The incidence of gallstones is unusually common in this population. Affected children will require regular lifelong blood transfusions (Hahalis et al, 2005).

Blood transfusions and increased gastrointestinal iron absorption result in iron overload and tissue damage. If splenomegaly occurs, a splenectomy may be needed. Excess iron accumulates, leading to liver, heart, and pituitary damage and failure of these organs. Cardiac complications caused by iron deposition, such as cardiomyopathy, are major causes of death (Hahalis et al, 2005). Chelation therapy may be needed.

Recent advances have been beneficial. Bone marrow transplants can be curative for some children with beta thalassemia major. Cord blood is the blood that remains in the umbilical cord and placenta following birth; it is a rich source of stem cells that reproduce into RBCs for the immune system. Stem cell transplantation offers opportunities for individuals with thalassemia to lead a more normal life.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: In thalassemia, variations are caused by the severity of the genetic mutations. In thalassemia major or intermedia, reduction in the number of alpha globin genes can ameliorate the disease phenotype; conversely, excess alpha globin genes convert beta thalassemia trait to a clinical picture of thalassemia intermedia (Rund and Fucharoen, 2009). An increase in Hgb F level is variably associated with the presence of beta thalassemia trait. Levels relate to the presence of a polymorphism in the (G)gamma-158 (C>T) gene (Mosca et al, 2009).

Clinical/History

- Height
- Weight
- BMI
- Diet history
- BP
- Growth failure
- Jaundice
- I & O
- Leg ulcers
- Bone abnormalities
- Enlarged spleen
- Hypogonadism
- Anemia
- Jaundice

Lab Work

- RBC, CBC
- H & H (low?)
- Quantitative Hgb A2 and Hgb F
- Serum Fe (increased)
- FEP
- Transferrin iron saturation percentage (increased)
- Serum lead
- Transferrin (decreased)
- Superconducting Quantum Interference Device (SQUID)
- Serum ferritin (increased)
- TIBC (decreased)
- Thyroid-stimulating hormone (TSH)
- Thyroxine (T4)
- Alkaline phosphatase (Alk phos)
- Gluc
- Vitamin B_{12}
- Serum zinc
- Alb
- CRP
- Hypoparathyroidism

INTERVENTION

OBJECTIVES

- Offer temporary relief with blood transfusions; this will improve hematological status with oxygen availability.

SAMPLE NUTRITION CARE PROCESS STEPS

Unintentional Weight Loss

Assessment Data: Growth failure in 6-year old, weight loss of 3# in past 12 months. Poor oral intake and anorexia. Hx of thalassemia intermedia, diagnosed at age 2.

Nutrition Diagnoses (PES): Unintentional weight loss related to poor oral intake as evidenced by weight loss of 3# in past year and growth failure (previously 40th percentile, now at 25th percentile for age).

Interventions: Food–nutrient delivery: enhance meals and snacks with high-density foods such as milkshakes with dry milk powder and peanut butter added. Educate parents about ways to enhance energy and nutrient density in meals and snacks. Counsel about when to contact the physician (e.g., signs of jaundice, additional weight loss, or chronic anorexia).

Monitoring and Evaluation: Improved intake of energy-dense foods and beverages. Growth chart improving over past 3–6 months, closer to 40th percentile again.

Correct side effects of iron overloading from the necessary transfusions.
- Correct failure to thrive and GI problems.
- Prevent slow or stunted growth. Impaired growth is a problem in children.
- Reduce or correct infections. Promote healing of any ulcerations.
- Manage any hyperglycemia.

FOOD AND NUTRITION

- A diet high in quality protein, energy, B-complex vitamins (especially folic acid and vitamin B_{12}), and zinc will be beneficial. To prevent iron overloading, avoid use of multivitamin–mineral supplements that contain iron and vitamin C in large amounts.
- Provide adequate fluid intake.
- If hyperglycemia and diabetes are present, use carbohydrate counting and other accepted techniques for managing glucose levels.

Common Drugs Used and Potential Side Effects

- Iron-chelating therapy with deferoxamine in patients with thalassemia major has dramatically improved the prognosis of this disease (Taher et al, 2005). Side effects include allergic reactions, tinnitus, and erythematous rash. Overchelation may cause growth retardation and mineral deficiency.
- Oral iron-binding agents are capable of preventing dietary iron absorption from the diet; oral chelator deferiprone (Ferriprox) is one.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss ways to improve nutritional intake, when deficient.
- Discuss importance of diet in the maintenance of hematological health.
- Persons with thalassemia should be referred for preconception genetic counseling. Women with alpha thalassemia trait should consider chorionic villus sampling to diagnose infants with Hgb Bart's, which increases the risk of toxemia and postpartum bleeding (Muncie and Campbell, 2009).

Patient Education—Food Safety

If tube feeding or CPN is needed, careful handwashing procedures should be followed.

For More Information

- Cooley's Anemia Foundation
  www.thalassemia.org
- Cord Blood Information
  http://www.thalassemia.com/cord_blood.html
- Thalassemia International Federation
  http://www.thalassaemia.org.cy/

THALASSEMIA—CITED REFERENCES

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Other Blood Disorders

BLEEDING DISORDERS: HEMORRHAGE AND HEMOPHILIA

NUTRITIONAL ACUITY RANKING: LEVEL 2

DEFINITIONS AND BACKGROUND

The circulatory system is a closed system, with low volume and high pressure. It provides efficient delivery of nutrients to all tissues. When there is volume loss, a large decrease in nutrient delivery occurs.

Hemorrhage is the excessive discharge of blood from a ruptured vessel. Bleeding (bright red in spurts from an artery; dark red and steady flow from a vein) can be external, internal, or into skin or other tissue. When massive, a hemorrhage can cause such symptoms as rapid, shallow breathing; cold, clammy skin; thirst; visual disturbances; and extreme weakness. Loss of more than 20% of blood volume causes hypotension and tachycardia; loss of more than 1 quart of blood may lead to shock. Peptic ulcer, hemophilia, spontaneous liver rupture, or stroke may lead to a hemorrhage. In some cases, surgery may be necessary. In chronic myelogenous leukemia (CML), a slowly progressive disease, platelets are increased in number and easy bleeding occurs.

To stop a hemorrhage, blood must clot properly. Blood clots when its fibrinogen is converted to fibrin by action of thrombin. Vitamin K works as a coenzyme that converts glutamic acid to gamma-carboxyglutamic acid; this helps to bind calcium and is required for the activation of the seven vitamin K–dependent clotting factors in the coagulation cascade (Table 12-18).

Hemophilia is an inherited bleeding disorder. Diagnosis may be early in life, or later after surgery or trauma. In severe cases, serious bleeding may occur without any cause. While internal bleeding may occur anywhere, bleeding into joints is common. Standard treatment involves replacing the missing clotting factor. In pregnant women who carry the trait, a C-section is often recommended. **Von Willebrand disease** is the most common hereditary bleeding disorder, where bleeding gums, abnormal menstrual bleeding, nose bleeds, and bruising are the symptoms. Desamino-8-arginine vasopressin (DDAVP) is given to raise the levels of von Willebrand factor, which reduces the bleeding tendency.

The immune response to coagulation factors VIII or IX with formation of inhibitory antibodies complicates the treatment of hemophilia; regulatory T cells (Treg) are an important component of the mechanism by which tolerance is maintained (Cao et al, 2009). New gene therapy and immune tolerance protocols are under study.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Hemophilia A, which affects 80–90% of cases, shows a mutation in the FVIII gene. People with Hemophilia B have low or missing levels of clotting factor IX. Hemophilia affects mostly males, although women carry the trait.

Clinical/History

Height
Weight
BMI
Diet history
BP
Pulse
Temperature
I & O
Blood in urine or stool?
Petechiae
Hemophiliac arthropathy
Excessive or easy bleeding
Excessive bruising
Nose bleeds
Abnormal menstrual bleeding

TABLE 12-18 Blood Clotting Factors That Involve Nutrition

The coagulation cascade involves a series of steps that stop bleeding through clot formation. Vitamin K–dependent coagulation factors are synthesized in the liver. Consequently, severe liver disease results in lower blood levels of vitamin K–dependent clotting factors and an increased risk of uncontrolled bleeding (hemorrhage). The following factors involve nutrition:

I. Fibrinogen
II. Prothrombin
III. Thromboplastin
IV. Calcium

In hemostatic (bleeding) disorders, it is important to evaluate for bleeding problems in the family history, history of heavy menses or easy bruising, and prior blood transfusions. Bleeding disorders include a number of conditions in which people tend to bleed longer. Clotting involves about 20 different plasma proteins (clotting factors). Normally, clotting factors form fibrin that stops bleeding. In bleeding disorders, the process does not occur normally

Some bleeding disorders are present at birth (hemophilia and von Willebrand's disease), or they can be acquired (such as vitamin K deficiency, severe liver disease, use of anticoagulant drugs or prolonged use of antibiotics, bone marrow problems, leukemia, pregnancy-associated eclampsia, or snake bite). In these disorders, vision loss can occur from bleeding into the eye, or anemia may result, or there may be neurological problems or even death. Gene therapy may one day be available to treat the bleeding disorders.

Lab Work

Coagulation testing
PT, International normalized ratio (INR)—prolonged?
Activated partial thromboplastin time (aPTT)
Thrombin time (thrombin added to plasma, and time to clot measured)
Fibrinogen
Platelet count (may be normal)
Von Willebrand factor level (reduced?)
Transferrin
RBC
Alb
BUN
CBC
H & H
Serum Fe
Serum folic acid and B_{12}
TIBC (increased)
Creatinine
CRP

INTERVENTION

OBJECTIVES

- Medical management is designed to control bleeding, take care of the underlying cause of the bleeding, and replace lost blood. Transfusions may be needed. Less severe hemorrhages may require iron, vitamin B_{12}, and folic acid to help replace RBCs.
- Support erythropoiesis.
- Control intestinal impact of gastrointestinal bleeding, which can cause a protein overload.
- Prevent hypovolemic shock (low cardiac output, decreased blood pressure, and decreased urinary output) from uncontrolled bleeding.

FOOD AND NUTRITION

- Ensure that diet is rich in proteins, iron, folic acid, vitamin B_{12}, and copper.
- Check need for vitamin K. Patients with intestinal or liver disease may become deficient. If medications to replace vitamin K are used, diet should provide a balance without excess. Monitor content of meals or enteral feedings and multivitamin supplements carefully to ensure that all RDAs are met without excesses (Table 12-19).

SAMPLE NUTRITION CARE PROCESS STEPS

Inadequate Vitamin Intake

Assessment Data: Bleeding disorder (Hemophilia A) with easy bruising and blood in urine and stool. Currently taking Alphanate; scheduled for dental surgery in 2 weeks. Serum vitamin K levels low. Diet hx shows little intake of any vitamin K–rich foods.

Nutrition Diagnoses (PES): Inadequate vitamin K intake related to hereditary bleeding disorder and minimal dietary intake as evidenced by low serum vitamin K levels, easy bruising and blood in urine and stool even while taking Alphanate.

Interventions: Food–nutrient delivery: identify foods that could be included and tolerated. Educate about the sources of vitamin K from diet. Counsel about multivitamin–mineral supplements that contain a desired dose of vitamin K (10–120 μg per dose).

Monitoring and Evaluation: Fewer episodes of blood in urine and stool; less easy bruising. Tolerance for multivitamin–mineral supplement and foods. Improved serum levels of vitamin K. No difficulty with dental surgery and excessive bleeding.

TABLE 12-19 Food Sources of Vitamin K

Food	Serving	Vitamin K (μg)
Kale, raw	One cup (chopped)	547
Broccoli, cooked	One cup (chopped)	420
Parsley, raw	One cup (chopped)	324
Swiss chard, raw	One cup (chopped)	299
Spinach, raw	One cup (chopped)	120
Leaf lettuce, raw	One cup (shredded)	118
Watercress, raw	One cup (chopped)	85
Soybean oil	One tbsp	26
Canola oil	One tbsp	20
Mayonnaise	One tbsp	12
Olive oil	One tbsp	7

Source: U.S. Department of Agriculture. USDA national nutrient database for standard reference, release 16. Available at http://www.nal.usda.gov/fnic/foodcomp/Data/SR16/wtrank/wt_rank.html.

Common Drugs Used and Potential Side Effects

- Avoid aspirin, NSAIDs, and other blood thinners. Oral anticoagulants, such as Warfarin, inhibit coagulation through antagonism of the action of vitamin K. Inadequate gamma-carboxylation of vitamin K–dependent proteins will inhibit clot formation. Patients taking these drugs are cautioned against consuming very large or highly variable quantities of vitamin K in their diets; they need a reasonably constant dietary intake.
- If vitamin K is needed, it is available in multivitamins and other supplements in doses that range from 10 to 120 μg per dose.
- Alphanate (antihemophilic factor) is approved to decrease bleeding in patients with bleeding diseases who must have surgery or other invasive procedures. People with hemophilia and their families can be taught to give factor VIII concentrates at home at the first signs of bleeding. XYNTHA is a new recombinant factor VIII product for both the control and prevention of bleeding episodes and surgical prophylaxis.
- FEIBA therapy, consisting of activated prothrombin complex concentrate (aPCC) and recombinant activated factor VII (rFVIIa), is effective and safe for reducing bleeding in hemophilia A (Valentino, 2009).

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.
- Potential adverse effects of high vitamin E intakes in humans, such as bleeding, are not clear (Hathcock et al, 2005).

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Blood donors should be alerted to the need to replace daily iron intake by 0.7 mg for a year. Every pint is equivalent to 250 mg of iron lost.
- Discuss adequate dietary replacement for lost nutrients. A multivitamin–mineral supplement may be indicated.

Patient Education—Food Safety

If tube feeding or CPN is needed, careful handwashing procedures should be followed.

For More Information

- All About Bleeding
  http://www.allaboutbleeding.com/
- Anemia from Excessive Bleeding
  http://www.merck.com/mmhe/sec14/ch172/ch172b.html
- Blood Line
  http://www.bloodline.net/
- International Society on Thrombosis and Haemostasis
  http://www.isth.org/
- National Hemophilia Foundation
  http://www.hemophilia.org/about/programs.htm
- World Federation of Hemophilia
  http://www.wfh.org/2/docs/Publications/Diagnosis_and_Treatment/Gudelines_Mng_Hemophilia.pdf

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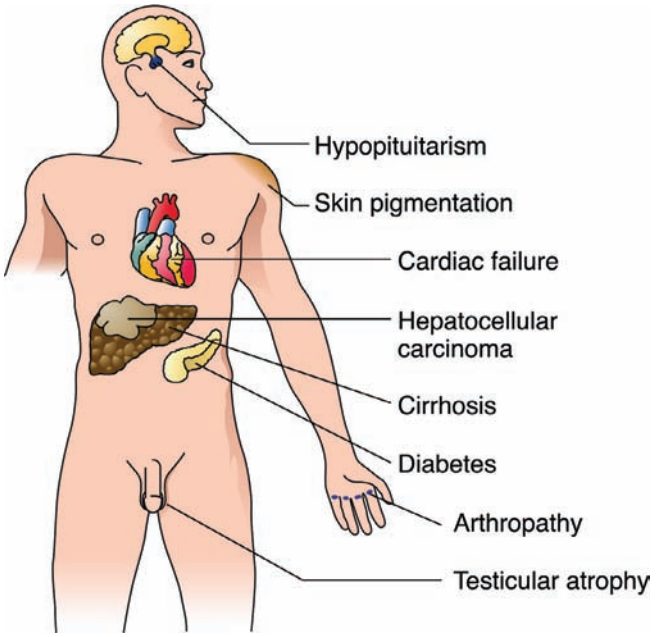
Hathcock JN, et al. Vitamins E and C are safe across a broad range of intakes. *Am J Clin Nutr.* 81:736, 2005.

Valentino LA. Assessing the benefits of FEIBA prophylaxis in haemophilia patients with inhibitors. Haemophilia. 2009 Dec 16. [Epub ahead of print]

HEMOCHROMATOSIS AND IRON OVERLOAD

NUTRITIONAL ACUITY RANKING: LEVEL 2



Adapted from: Rubin E MD and Farber JL MD. *Pathology*, 3rd ed. Philadelphia: Lippincott Williams & Wilkins, 1999.

DEFINITIONS AND BACKGROUND

Hereditary hemochromatosis (HH) is one of the most common autosomal recessive disorders among Caucasians. One in 200–400 individuals of Northern European ancestry is at risk for hemochromatosis (Camaschella and Merlini, 2005). It is also common in Hispanics or people of Mediterranean descent and is 10 times more common in males than females. Irish Americans and African–Americans have double the usual frequency. Tragically, hemochromatosis remains underdiagnosed.

In hemochromatosis, iron stores are deposited in excess, often from excess intake or liver/pancreatic diseases, renal dialysis, or frequent and long-term transfusions. Healthy people may accumulate up to 1 g of iron, but people with this condition accumulate 15–30 g. Increased iron absorption leads to excessive accumulation of iron deposits within cells of the liver, heart, pituitary gland, pancreas, and other organs, gradually causing tissue damage.

Because hemochromatosis has many possible symptoms, it often goes undiagnosed. However, early detection is important and may prevent organ failure that can occur if it is left untreated. Long-term complications include liver cirrhosis, diabetes, cardiomyopathy, hypogonadism, arthropathy, skin pigmentation, and susceptibility to liver cancer (Camaschella and Merlini, 2005) (Table 12-20).

Iron overload patients may have diagnoses other than HH: non-alcoholic fatty liver disease (NAFLD), chronic hepatitis C, and chronic alcohol use are most common (Dever et al, 2009). Iron toxicity can also occur in aplastic anemia, chronic hemolytic anemia, porphyria cutanea tarda, sideroblastic anemia, thalassemias, diabetes, rheumatoid arthritis, or transfusional iron overload. Sometimes, individuals with Alzheimer's or Parkinson's disease may have heavy metal toxicities that contribute to an iron overload. Free iron is destructive to cells, and too much iron can be a carcinogen because cancer cells need it for their DNA synthesis. With chronic kidney disease, keep serum ferritin levels below 500 ng/dL.

Porphyrias are rare disorders caused by lack of the enzymes necessary for production of heme; this causes heme precursors, porphyrins, to accumulate in the bone marrow, liver, and bloodstream (MedlinePlus, 2009). The porphyrins may also

TABLE 12-20 Facts About Hemochromatosis

1. Undetected or untreated excess iron kills after inflicting injury to a variety of body organs. The physician's concern must be to detect any excess iron instead of establishing the diagnosis
2. Some literature suggests treatment when ferritin alone is elevated. Giving blood does no harm and, instead, is beneficial to health. About one fourth of patients have low hemoglobin; treatment is the same unless the anemia is so severe that blood transfusions are required. Severely anemic patients require iron removal by an iron chelator, Desferal
3. Iron overloading is preventable. When diagnosis is in doubt, the patient should begin a trial of weekly phlebotomies at the blood bank. Four to 6 weeks will usually provide the answer, and getting rid of a little excess iron will improve health
4. The patient should be taken to the blood bank upon the physician's order for weekly phlebotomies
5. A liver biopsy is not always necessary, and waiting can delay important treatment. DNA testing is not useful because it cannot detect all of the known mutations
6. When iron levels test low, the cause must be found. It is dangerous to medicate with iron without testing first and then finding the reason for any deficiency
7. Symptoms vary. Chronic fatigue, arthritis, anemia (iron-loading anemia is one symptom), and elevated liver enzymes must not be ignored. Hemoglobin level does not indicate iron status. A disorder of thyroid or any part of the body can be a symptom of iron overload
8. Excess iron lowers immunity. Many diseases (such as cancer, hepatitis, and AIDS) will show a poor outcome unless any excess iron is removed. Excess iron stored in the brain exacerbates severity in Alzheimer's, multiple sclerosis, Lou Gehrig's disease, Parkinson's disease, psychological problems, autism, and other diseases

Adapted from: Iron Overload Diseases Association, http://www.ironoverload.org/, accessed December 23, 2009.

be excreted in the urine or stool. Most porphyrias are hereditary, but attacks may also be triggered by drugs, alcohol, hormones, or infections.

Acute, hepatic porphyrias affect the nervous system. Symptoms include nerve damage with pain or paralysis, abdominal pain and liver damage, red or brown urine, anxiety and delirium, muscle pain or weakness, numbness or tingling, tachycardia, loss of deep tendon reflexes, low blood pressure, and electrolyte imbalances. Constipation or diarrhea may occur. A diet high in carbohydrate (55–60% of total kilocalories) and beta carotene may be beneficial (MedlinePlus, 2009).

Porphyria cutanea tarda (PCT) can occur without an inherited enzyme deficiency. The porphyrins accumulate in the liver and skin, causing photosensitivity, skin damage, and cirrhosis. Phlebotomy removes excess iron, and chloroquine or hydroxychloroquine removes the excess porphyrins from the liver (Anderson, 2007).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: HH is recessive, requiring the gene from two carrier parents. There are several types of genetic hemochromatosis: type I or classic (HHC); type II a, b, or juvenile (JHC); type III or transferrin receptor mutation; and type IV or ferroportin mutation. Iron overload can also occur in individuals with the HFE C2824 gene in 1 out of 200 people.

Clinical/History

- Height
- Weight
- BMI
- Diet history
- BP
- I & O
- Bronzing of the skin
- Profound fatigue (in HH)
- Arthralgia (in HH)
- Loss of body hair
- Loss of libido
- Lack of menstruation or early menopause
- Abdominal pain
- Chronic intermittent diarrhea
- Irregular heartbeat
- Cardiomegaly with congestive failure
- Hepatomegaly
- Enlarged spleen
- Hypothyroidism
- Depression
- Liver biopsy
- Bone marrow studies

Lab Work

- Transferrin-Iron Saturation Percentage[a] (normal 25–35%)—best test
- TIBC (normal, 12–45%)
- Transferrin (increased)
- Serum Cu (increased)
- Alb
- Serum Fe
- Ferritin (increased >1000 ng/mL?)
- Hgb (desirable = 10 g/dL)
- Hct (desirable = 30–35%)
- Gluc
- Serum B_6
- Serum B_{12}
- Serum folic acid
- Thyroid tests
- Liver function tests
- CRP

[a]Divide total serum iron by TIBC for percentage of tissue saturation (TS). Divide the serum iron level by TIBC for percentage of transferrin saturation.

INTERVENTION

OBJECTIVES

- Remove excess iron from body (usually with phlebotomies of 500 mL weekly, performed by the physician over several months). Then therapy is repeated several times annually for rest of the life.
- Prevent liver cancer, heart attack, or stroke by unloading storage iron as fast as possible; keep serum ferritin at low normal range.
- If excess iron intake is a chronic problem, discontinue use in supplements and fortified foods (such as iron-fortified cereals). Read labels carefully.

SAMPLE NUTRITION CARE PROCESS STEPS

Excessive Mineral Intake

Assessment Data: Male with transferrin-iron saturation percentage (46%), ferritin (160 ng/mL), elevated H & H. Diagnosis of iron overloading. Diet hx reveals high intake of animal proteins and heme iron (about 20 g/day).

Nutrition Diagnoses (PES): Excessive iron intake related to diet high in animal proteins and heme iron as evidenced by transferrin-iron saturation percentage (46%), ferritin (160 ng/mL), elevated H & H.

Interventions: Food–nutrient delivery—encourage more vegetarian meals and fewer ounces of meats at mealtime. Educate about the role of heme iron intake in iron overloading disorders. Counsel and provide meal planning tips and portion guides for intake of meats.

Monitoring and Evaluation: Improvement in serum laboratories (transferrin-iron saturation percentage, ferritin, and H & H) with levels closer to normal. Diet hx reveals improved intake of 8–10 g/day.

- Teach principles of nutrition and menu planning to incorporate adequate intake of other nutrients that may be depleted with excessive phlebotomies (e.g., folate and other B-complex vitamins, protein).

FOOD AND NUTRITION

- Provide a normal diet unless renal or hepatic function is altered. Do not consume foods or take supplements high in vitamin C. Read cereal labels and avoid those with 100% or more of the daily allowance for iron and vitamin C. A low-iron diet is not recommended.
- Ensure adequate protein and sufficient energy intake to meet estimated needs and activity levels.
- Avoid alcohol because of potential damage to a vulnerable liver.

Common Drugs Used and Potential Side Effects

- Avoid use of multivitamin supplements that contain iron and vitamin C because these can increase iron absorption.
- An iron chelator may be needed, such as deferoxamine (DFO). This is given intravenously 8–12 hours for up to five times in a week. It can be neurotoxic.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- All blood relatives of the patient must be evaluated and monitored yearly for iron overloading.
- Genetic testing of other family members is also recommended for those with inherited type.
- Discuss avoidance of alcohol and raw seafood. *Vibrio vulnificus* in some raw seafood kills people every year; many are those with undetected iron overload.
- Discuss nutrient sources as appropriate for the individual.

Patient Education—Food Safety

- If tube feeding or CPN is needed, careful handwashing procedures should be followed.
- Avoid eating raw seafood.

For More Information

- Iron Disorders Institute
  http://www.irondisorders.org
- Iron Facts
  http://ods.od.nih.gov/factsheets/iron.asp
- Iron Overload Diseases Association, Inc.
  http://www.ironoverload.org/
- Iron Tests
  http://www.irondisorders.org/Forms/irontests.pdf

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Dever JB, et al. Phenotypic characteristics and diagnoses of patients referred to an iron overload clinic. *Dig Dis Sci.* 2009 Dec 24. [Epub ahead of print]

MedlinePlus. Porphyrias. Web site accessed December 28,2009, at http://www.nlm.nih.gov/medlineplus/ency/article/001208.htm.

POLYCYTHEMIA VERA

NUTRITIONAL ACUITY RANKING: LEVEL 1

DEFINITIONS AND BACKGROUND

Polycythemia vera (PV) is a chronic, progressive disease in which increased blood volume and increased erythrocyte production occur. Other names include erythremia, Osler–Vasquez disease, and polycythemia rubra vera. Hematological disorders like PV can result in elevated levels of cobalamin, which is released during hepatic cytolysis.

The cause of PV is unknown, and the disease is considered a hematological malignancy. The disease develops

slowly and may progress to acute myelogenous leukemia. The average age at diagnosis is 50–60 years. Incidence is highest among those of Jewish ancestry, occurring in 2 of 100,000 of the population. Increased viscosity of the blood and number of platelets result in a high risk for clot formation and stroke, hemorrhage, or myocardial infarction.

Patients with PV frequently develop hyperhomocysteinemia due to discrete depletion of cobalamin or folate; vitamin therapy should be considered. With treatment, individuals with this condition may live for 15–20 years. Phlebotomy or medications may be used.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: The somatic V617 F mutation in the Janus kinase (JAK) 2 gene, which causes a valine to phenylalanine substitution at position 617, has recently been found in the majority of patients with PV (Meyer, 2009).

Clinical/History

Height
Weight
BMI
Diet history
I & O
BP (hypertension?)
Belching, fullness
Flatulence
Peptic ulcer?
Constipation
Headache
Vertigo
Lassitude
Tinnitus
Pruritus after bathing
Transient blurred vision, diplopia
Dyspnea
Chest pain
Dusky reddish skin on face and hands
Hemorrhagic tendency
Seizures, confusion
Splenomegaly
Tinnitus
Paresthesias
Gout

Lab Work

Hgb (>18 g/dL)
Hct (>52% for men; >47% for women)
Platelets (elevated)
Leukocytes (elevated)
Serum B_{12} (elevated)
Erythropoietin (low)
TIBC
Erythrocyte sedimentation rate (ESR)
Leukocyte Alk phos
Serum ferritin
Gluc
RBC (7–12 million)
Oxygen saturation >92%
CRP
Alb, transthyretin
CRP
Chol, Trig
BUN, Creat
Uric acid (elevated)
Bone marrow biopsy

INTERVENTION

OBJECTIVES

- Prepare patient for phlebotomy by ensuring adequate nutrient stores.
- Prepare, as needed, for chemotherapy or radiation therapy, which may be provided.

SAMPLE NUTRITION CARE PROCESS STEPS

Abnormal GI Function

Assessment Data: BMI 20. Constipation, flatulence, history of gastric bleeding, and ulceration.

Nutrition Diagnoses (PES): Abnormal GI function related to discomfort and pain as evidenced by constipation, flatulence, GI ulceration, and bleeding.

Interventions: Food–nutrient delivery—use comfort foods and adequate CHO, fiber, and fluid intake; reduce acidic foods and items not well tolerated. Educate about good nutrition and inclusion of B-complex vitamins. Counsel about individualizing tolerance for medications with appropriate food, fluid, and snacks.

Monitoring and Evaluation: Daily use of prescribed medications. Improvement in GI function; resolution of constipation and flatulence. No additional GI bleeding.

- Correct or control condition.
- Manage any side effects such as heart failure, peptic ulcer disease, gastric bleeding, gout, leukemia, and seizures.

FOOD AND NUTRITION

- A high CHO diet with preferred foods and balanced meals should be offered. Monitor for the need for vitamin or mineral supplementation. Include foods rich in beta carotene.
- Extra fluids will be helpful (35–40 mL/kg, unless contraindicated, as with heart failure).
- Changes in dietary texture or content may be needed if radiation or chemotherapy alters nutrient or dietary needs.

Common Drugs Used and Potential Side Effects

- Myelosuppressive agents may be prescribed. Anagrelide hydrochloride (Agrylin) is an oral imidazoquinazoline agent that has been shown to reduce elevated platelet counts and the risk of thrombosis. Interferon alpha may be used in younger patients; pegylated interferon alpha-2a (PEG-IFN-alpha-2a) is beneficial (Quintas-Cardama et al, 2009).
- The antimetabolite hydroxyurea may be used. Side effects include anemia and skin ulcers.
- Chemotherapeutic agents (busulfan, chlorambucil, and cyclophosphamide) may cause nausea and vomiting or weight loss.
- Low-dose aspirin is sometimes used in patients with thrombotic or ischemic conditions. It can relieve some of the burning sensations in the feet and hands. Antihistamines can help reduce itching sensation.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss need to maintain a healthy lifestyle and to eat adequate protein and calories because of the frequent phlebotomies, where completed.
- Discuss ways to make meals that are nutritious yet simple to prepare.
- Tepid oatmeal baths may help reduce pruritus.

Patient Education—Food Safety

If tube feeding or CPN is needed, careful handwashing procedures should be followed.

For More Information

- Mayo Clinic – PV
  http://www.mayoclinic.com/health/polycythemia-vera/DS00919
- Merck Manual–Blood Disorders
  http://www.merck.com/mmhe/sec14/ch178/ch178b.html
- Myeloproliferative Disorders
  http://www.acor.org/diseases/hematology/MPD/

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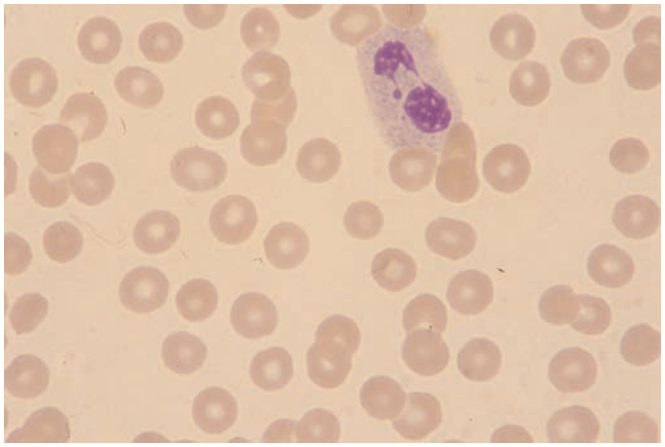
THROMBOCYTOPENIA

NUTRITIONAL ACUITY RANKING: LEVEL 1



Adapted from: *Anderson's Atlas of Hematology;* Anderson, Shauna C., PhD. Copyright 2003, Wolters Kluwer Health/Lippincott Williams & Wilkins.

DEFINITIONS AND BACKGROUND

Thrombocytopenia purpura, a myeloproliferative disorder, is a blood disease affecting the clotting factor (platelets) of the blood, with an abnormally low platelet count and shorter than normal (10 days) platelet survival time. Thrombocytopenia is the most common cause of bleeding, usually from small capillaries. Women are more affected than men.

There are many reasons for the development of decreased marrow production or platelet destruction that causes thrombocytopenia, including some hereditary causes. These can sometimes be determined by examination of bone marrow. Idiopathic thrombocytopenic purpura (ITP) is caused by platelet destruction by antibodies. Thrombotic thrombocytopenic purpura (TTP) is manifested by vascular lesions.

Plasma exchange (plasmapheresis) is used to remove the abnormal antibody from the blood and replace the missing enzyme. Mortality of TTP has decreased from 90% to 10% (George, 2009); survival improved dramatically with plasma exchange treatments after the 1980s (Kremer-Hovinga et al, 2009). Unfortunately, adults with TTP of any etiology have a high risk for persistent minor cognitive abnormalities (George, 2009).

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: Mutations in the ADAMTS13 gene cause the familial form of TPP. Alterations in the ADAMTS13 gene reduces instructions for the normal process of blood clotting.

Clinical/History

Height
Weight
BMI
Diet history
I & O
BP
Nosebleeds
Bleeding from other sites
Bruising
Pinpoint red spots on skin
Headache
Slurred speech
Numbness and weakness of extremities
Fever?
Pallor
Jaundice
Shortness of breath

Lab Work

CBC (low platelets)
H & H (decreased)
Alb, transthyretin
N balance
PT and PTT (normal)
Casts in urine
Proteinuria
CRP
Ca^{++}
Na^{+}, K^{+}

SAMPLE NUTRITION CARE PROCESS STEPS

Self-Feeding Difficulty

Assessment Data: BMI at lower end of normal, but some weight loss noted. Dx of TPP with numbness and weakness in hands and feet. Inability to feed self and remain independent; depression and easy frustration noted at mealtimes.

Nutrition Diagnoses (PES): Self-feeding difficulty related to numbness in hands as evidenced by inability to hold traditional utensils.

Interventions: Food–nutrient delivery—alter food choices to simplify options and offer more finger foods. Educate about use of adaptive feeding equipment that can be used for more independence. Counseling with tips on meal simplification.

Monitoring and Evaluation: Improved ability to feed self independently. No further weight loss. Less depression and frustration at mealtimes.

INTERVENTION

OBJECTIVES

- Avoid infections, especially upper respiratory infections and flu to prevent coughing, which increases intracranial pressure.
- Reduce bleeding tendency and complications, such as intracranial hemorrhage or GI bleeding (Goldman, 2007).
- Rest frequently.
- Prepare patient for splenectomy, if indicated. Ensure adequate nutrient stores.

FOOD AND NUTRITION

- Maintain diet of preference. Use small, frequent feedings if patient has nausea or vomiting.
- Adequate folic acid will be needed.
- Increase fluids (e.g., 3 L/d) unless contraindicated.
- After splenectomy, patient will need adequate protein, energy, zinc, and vitamins A and C for wound healing. Vitamin K from the diet and supplements may need to be monitored.

Common Drugs Used and Potential Side Effects

- Most drugs are stopped because nearly any drug may aggravate the condition.
- Corticosteroids such as prednisone may be used to control bleeding. Side effects are numerous and may affect nutritional status (e.g., decreased serum calcium, potassium, and nitrogen; increased serum sodium; and glucose intolerance may occur).
- Myelosuppressive agents are often prescribed. Anagrelide hydrochloride (Agrylin) is an oral imidazoquinazoline agent that has been shown to reduce elevated platelet counts and the risk of thrombosis. Interferon alpha may be used.
- Rituximab seems to be a promising drug in the treatment of refractory autoimmune thrombocytopenia.

Herbs, Botanicals, and Supplements

- Herbs and botanical supplements should not be used without discussing with the physician.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Discuss altering nutrients as needed, depending on medications ordered and their use over time; surgery, if required; and ability to eat adequately.

Patient Education—Food Safety

If tube feeding or CPN is needed, careful handwashing procedures should be followed.

For More Information

- The ITP Society of the Children's Blood Foundation
  http://www.childrensbloodfoundation.org/
- Platelet Disorder Support Foundation
  http://www.pdsa.org/

THROMBOCYTOPENIA—CITED REFERENCES

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SECTION 13

Cancer

CHIEF ASSESSMENT FACTORS

American Cancer Society's Seven Warning Signs of Cancer

- Change in Bowel/Bladder Habits
- Indigestion or Dysphagia
- Nagging Cough or Hoarseness
- Obvious Change in Wart or Mole
- Sore That Does Not Heal
- Thickening or Lump in Breast or Elsewhere
- Unusual Bleeding or Discharge

Other Factors

- Anorexia or Chronic Nausea
- Changes in Food Intake, Usual Functional Capacity, Energy Levels
- Depression
- Diarrhea
- Dry Mouth
- Dysphagia, Esophagitis, Mouth Sores, Mucositis
- Edema or Ascites
- Fever of Unknown Origin (Hematological, Liver, Pancreatic, Brain, Kidney Cancers)
- History of Carcinogen Exposure, Tobacco Use, Excessive Alcohol Use
- Intolerance for Nauseating Odors
- Muscle Wasting
- Nutrient Intake and Immune Function
- Pain
- Participation in Complementary and Alternative Medicine Treatments
- Side Effects of Medications
- Vomiting
- Weight Changes—Unintended Weight Loss or BMI Less Than 22

TABLE 13-1 Cancer Definitions

Term	Definition
Adenocarcinoma	Cancer that starts in the glands.
Adenoma	Benign growth that may or may not transform into cancer.
Antiangiogenesis	Process of stopping a tumor from growing new blood vessels.
Antibodies	Proteins in the immune system; in cancer, antibodies are used to recognize specific cancer cell receptors and to act as smart bombs.
Basal cell carcinoma	Most common form of skin cancer, affecting 800,000 Americans each year. Chronic exposure to sunlight causes most basal cell carcinomas, which occur most frequently on exposed parts (e.g., face, ears, neck, scalp, shoulders, and back).
Biotherapy	Treatment to stimulate or restore the ability of the immune system to fight infection and disease and to lessen side effects that may be caused by some cancer treatments; also known as immunotherapy, biological therapy, or biological response modifier (BRM) therapy.
Cancer	Abnormal, uncontrolled growth of cells in a lump or mass that also destroys normal tissue. Oncogenes in a tumor cell may be identifying markers.
Carcinoma	Cancer involving epithelial tissue and coverings of internal and external surfaces; lungs, colon, breast, stomach, uterus, skin, and tongue cancers. 80–90% of all cancers.
Chemotherapy	Use of medications to kill malignant cells.
Curative Therapy	Permanent removal of the cancer from the body.
Endocrine system cancers	Adrenocortical carcinoma; gastrointestinal carcinoid tumor; pancreatic islet cell carcinoma; parathyroid cancer; pheochromocytoma; pituitary tumor; and thyroid cancer.
Epithelioma	Carcinoma consisting of many epithelial cells.
Gastrointestinal (GI) cancers	Anal cancer; bile duct cancer; colon cancer; esophageal cancer; gallbladder cancer; GI carcinoid tumor; liver cancers; pancreatic cancer; rectal cancer; small intestine cancer; stomach cancer.
Gynecological cancer	Female reproductive system: cervical cancer; endometrial cancer; gestational trophoblastic tumor; ovarian epithelial cancer; ovarian germ cell tumor; ovarian low malignant potential tumor; sarcoma; vaginal cancer; and vulvar cancer.
Hormonal therapy	Treatment by surgery or by shrinking or killing hormone-dependent cancers.
Leukemia	Cancer where bone marrow–produced abnormal white blood cells crowd out normal white blood cells, red blood cells, and platelets.
Lymphoma	AIDS-related lymphoma; cutaneous T-cell lymphoma; Hodgkin's lymphoma; mycosis fungoides; non-Hodgkin's lymphoma; primary central nervous system lymphoma; Sézary's syndrome; and Waldenstrom's macroglobulinemia.
Male reproductive cancers	Penile, testicular cancers, prostate cancers.
Meningiomas	Tumors affecting the meninges.
Mesothelioma	Rare cancer affecting the lining of the chest, heart, and abdominal cavity from exposure to asbestos.
Metastasis	Transfer of disease from one organ to another that is not directly connected to it; particularly the spread of carcinoma.
Monoclonal antibodies	Targeted therapy to locate and bind cancer cells. May be used alone or used to deliver drugs, toxins, or radioactive material directly to tumor cells.
Neuroma	A tumor composed of nerve cells, which may occur along any nerve.
Oat cell carcinoma	A rapidly spreading, highly fatal cancer of the bronchus.
Oncology	Scientific study of tumors.
Osteosarcoma	Most common bone cancer, which develops in new tissue in growing bones, affecting young people and more males.
Palliative therapy	Pain relief but not expected to cure the disease. Given to improve quality of life as much as possible.
Radiation	Treatment with high-energy rays to kill or damage cancer cells. May be external rays or internally placed radioactive material.
Sarcoma	Cancer arising from bone or connective tissue, which sometimes spreads into blood or lymphatic tissues.
Small cell carcinoma	Carcinoma that most commonly arises in the lung but can occur as a cancer in other body sites including the prostate, cervix, and head and neck; responsive to chemotherapy and radiation therapy.
Vaccine	To stimulate the immune system to mount a defense against cancer cells. Example – the cervical cancer vaccine.

Definitions: http://www.cancer.gov/dictionary/ and Types of cancers: http://www.cancer.gov/cancertopics/alphalist/a-d, accessed December 29, 2009.

For More General Information on Cancer: Table 13-1 provides a list of cancer definitions.

- AMC Cancer Research Center and Foundation
  http://www.amc.org/
- American Cancer Society
  http://www.cancer.org
- American Dietetic Association Oncology Nutrition Dietetic Practice Group
  http://www.oncologynutrition.org/
- American Institute for Cancer Research (AICR)
  http://www.aicr.org/
- Cancer Care
  http://www.cancercare.org/
- Cancer Screening Guidelines
  http://www.aafp.org/afp/20010315/1101.html
- Caring 4 Cancer
  http://www.caring4cancer.com/go/cancer/nutrition

- Eating Hints for Cancer Patients
  http://www.cancer.gov/cancertopics/eatinghints
- Genetics 101
  http://www.ornl.gov/sci/techresources/Human_Genome/project/info.shtml
- Harvard–Dana-Farber Cancer Institute
  http://www.dfci.harvard.edu/
- Harvard Center for Risk Analysis
  http://www.hcra.harvard.edu/#
- Hospice Net
  http://www.hospicenet.org/
- Human Carcinogens
  http://www.cancer.org/docroot/PED/ped_1_1.asp
- Journal of the National Cancer Institute
  http://jncicancerspectrum.oxfordjournals.org/
- Lance Armstrong Foundation
  http://www.laf.org
- National Cancer Institute
  http://www.nci.nih.gov/
- National Coalition for Cancer Survivorship
  http://www.canceradvocacy.org/
- National Institutes of Health Gene Testing
  http://www.genetests.org/
- National Toxicology Program
  http://ntp-server.niehs.nih.gov/index.cfm
- North American Cancer Registry
  http://www.naaccr.org/
- Oncology Association of Naturopathic Medicine
  http://www.oncanp.org/
- Oncology Nursing Society
  http://www.ons.org/
- Online Human Genome Resources
  http://www.genome.gov/10000464
- Vital Options
  http://www.vitaloptions.org/

CANCER PREVENTION AND RISK REDUCTION

Cancer results from dysregulated cell growth control and is caused by an interaction of dietary, genetic, and environmental risk factors. There are over 100 variations of cancer. Cancer has a strong genetic component, associated with initiation, promotion, and metastatic growth. The Human Genome Project has identified 30,000 human protein-coding genes. Individualized DNA methylation helps to control gene expression. Genotyping resources allow cancer prevention investigators to identify which genetic subsets of patients are likely to benefit most from chemoprevention and interventions. This emerging science of nutritional genomics is very promising (Kauwell, 2005).

Natural carcinogens include ultraviolet (UV) radiation, dyes, environmental chemicals from smoke or mines, viruses, nitrosamines, aflatoxins, and safrole. The most consistent carcinogen is tobacco. Approximately 30% of cancers also have a nutrition or dietary component (Williams and Hord, 2005). Functional food components greatly impact the incidence and treatment of cancer. In addition, food intake, aging, and immune function share a complex relationship; selenium, EPA, DHA, vitamin A, and sodium seem to be particularly important (Wardwell et al, 2008).

Nutritive and nonnutritive dietary constituents can either promote or hinder the development of cancer, individualized by genetic predisposition. Diets rich in carotenoids, antioxidative vitamins, phenolic compounds, terpenoids, steroids, indoles, and fibers reduce the risk of cancer and related chronic diseases (Aggarwal and Shishorida, 2004). Studies support the role of flavonoids, carotenoids, curcumin, ascorbic acid, and citrus liminoids (Patil et al, 2009). Polyphenols are the most abundant antioxidants in the average diet and are constituents of fruits, vegetables, cereals, dry legumes, chocolate, tea, coffee, and wine.

The strongest evidence linking specific foods to a decreased risk of certain cancers is related to the consumption of fruits, vegetables, and whole grains. Antioxidants protect against free radical damage, improving the resistance of cells to oxidative stress. In a study using HANES data, daily intakes of antioxidants from both diet and supplements averaged 208 milligrams of vitamin C, 20 milligrams of alpha-tocopherol, 223 retinol activity equivalents (RAE) of carotenes, 122 micrograms of selenium, and 210 milligrams of dietary flavonoids (Chun et al, 2009). Women, older adults, Caucasians, nonconsumers of alcohol, nonsmokers, and those with a higher income and exercise level than other tended to have better intakes (Chun et al, 2009). Key nutrients, chemoprotective phytochemicals, and functional food ingredients are listed in Table 13-2.

Promoting fruits, vegetables, and whole grains is the key message. Choose "better for you" foods, and make vegetables the central focus of the plate. Minimize meats, or use leaner cuts. Enhance the diet with nuts and whole grains (insoluble types to increase stool bulk and push bile out; soluble types for their cholesterol-lowering effect). A complete "anticancer" grocery list includes dark green, yellow, and orange fruits or vegetables; red grapes; cruciferous vegetables; orange juice; tomatoes; olive and canola oils; garlic; legumes; strong coffee; whole grains; soy; and other plant estrogens.

Dietary factors and physical inactivity contribute to approximately one-third of all cancers. Table 13-3 provides a list of important dietary factors. Excess body weight increases the risk of several cancers. There are five lifestyle habits to promote: maintain BMI <25, get 30 minutes of exercise, limit alcohol to—one to two drinks daily, do not smoke, and choose a healthy diet rich in phytochemicals.

Many cancer patients try CAM therapies; fish oil is the leading choice of adults. Yet the emphasis remains on food sources, not supplements or pills. The intake of whole foods and fortified, enriched, or enhanced foods has the most beneficial impact on health (American Dietetic Association, 2009).

TABLE 13-2 Phytochemicals, Functional Food Ingredients, and Cancer

Phytochemicals are functional foods or ingredients that occur naturally in fruits and vegetables and whole grains, often to protect against microorganisms. They are not "essential" nutrients, but some phytochemicals function as antioxidants to squelch free radicals.

Food	Functional Ingredient	Possible Roles in Reducing Cancer Risk
Apples (MALUS sp., Rosaceae)	Hydroxycinnamic acids, dihydrochalcones, catechins and oligomeric procyanidins	Also contain triterpenoids in apple peel and anthocyanins in red apples. Protect against skin, lung, breast, and colon cancer (Gerhauser, 2008).
Apples, black tea, grapefruit, onions, arugula	Quercetin, kaempferol, myricetin, isorhamnetin (flavonols)	Decreases ascorbate-dependent free radical oxidation; decreases inflammation and tumorigenesis. May be protective against colorectal cancer. Since apple skins retain pesticides, choose organic.
Beans and legumes, soybeans, whole grains, alfalfa, lentils, bean sprouts	Saponins: oleanic acid, hedagenin (terpenes)	Triterpene glycosides that neutralize enzymes in the intestine that may cause cancer; they boost immunity. Consume only moderate amounts of soy as part of a healthy plant-based diet.
Beef, lamb, yogurt, some cheeses and dairy products	Conjugated linoleic acid	Maintains immune function and normal body composition; some antitumor properties. Marinate meats in red wine or beer to cut heterocyclic amine exposure; do not burn meats.
Bell peppers, citrus fruits	Vitamin C	Protects against damage from free radicals.
Blueberries (particularly skins), blackberries, raspberries, strawberries, cherries, red grapes, red cabbage, eggplant, red onion, kidney beans, red beans, beets, black currants, elderberries, purple sweet potato skin, prunes	Anthocyanins (polyphenols/ flavonols)	Bolsters cellular antioxidant defenses particularly against UV radiation; maintain brain function and motor function; neutralize free radicals; have antimicrobial action.
Brazil nuts, lean meats, tuna, salmon, seafood	Selenium and Glutathione	Increases immune cell functioning, DNA methylation, and regulation of cytokine production. Protects from damage from free radicals. SELECT study found that a 200-milligram selenium supplement actually promotes prostate cancer.
Broccoli, broccoli sprouts, horseradish, cauliflower, cabbage, bok choy, Brussels sprouts, kale	Sulforaphane; thiols	Isothiocyanates (ITCs) protect the body from cancer by inducing detoxification enzymes such as quinone reductase. They increase periods of cancer latency and are effective agents against fungi such as *Aspergillus*. Protects against stomach and skin cancers. Lightly cooked broccoli has more; use a mix of raw and cooked vegetables.
Broccoli, peas, beans, and other vegetables; soybeans, clover, alfalfa	Coumestans (phytoestrogen)	High level of estrogenic activity that may reduce the risk for lung cancer. Estimated daily intake of coumestans is 0.6 micrograms; broccoli is the main source.
Cabbage, sauerkraut	Glucosinolate	May lower the risk of hormonal cancers
Carrots, American ginseng roots	Falcarinol, falcarindiol, panaxydol (polyacetylenes)	Inhibits cell proliferation in normal and cancer cells through synergism of bioactive polyacetylenes. More effective if whole and not chopped before cooking.
Carrots, sweet potatoes, pumpkin, butternut squash, cantaloupe, mangoes, apricots, peaches, papaya, watermelon	Beta-carotene (terpenes)	Beneficial effects on human cancer prevention; increases the activity of killer cells slightly; photoprotective; neutralize free radicals; serves as antioxidants. Avoid excesses of supplements as they act as pro-oxidants. Dietary fat is needed for proper absorption. Boil or steam to protect the antioxidants.
Cereals, legumes, nuts, sesame seeds, soybeans, brown rice, corn and wheat brans	Inositol	Found as phytic acid in plants. Used by all cells to relay outside messages to the cell nucleus. Aids in the metabolism of calcium and other minerals.
Cherry juice	Chlorogenic acid (phenolic acid)	Strong antioxidant properties.
Cinnamon, cocoa, apples, strawberries, purple grapes and wines, peanuts, cranberries	Proanthocyanidins	Decreases oxidative stress; supports urinary tract health.
Citrus fruits, lemon, cantaloupe, pomegranate, potato skins, wild leafy greens, celery stalks, lettuce, chili and sweet peppers, spinach, parsley, watermelon, whole grains, tomato sauce, red wines	Apigenin, luteolin (flavones)	Tumor growth inhibition and chemoprevention; may protect against skin cancer and ultraviolet damage. May protect women against ovarian cancer.
Citrus fruits, apples, pears		
Some vegetables	Caffeic acid, ferulic acid (flavanones)	Bolsters antioxidant defenses.

(continued)

TABLE 13-2 Phytochemicals, Functional Food Ingredients, and Cancer *(continued)*

Food	Functional Ingredient	Possible Roles in Reducing Cancer Risk
Corn, soy, wheat, wood oils	Plant stanols	Inhibits cholesterol absorption.
Cruciferous vegetables: Broccoli, cabbage, sauerkraut, Brussels sprouts, bok choy, arugula, Swiss chard, watercress, cauliflower, kale, kohlrabi, turnips, rutabaga	Indoles (indole-3-carbinol); glucosinolates (thiols)	Antimutagenics that may enhance detoxification of undesirable compounds. May contribute to a healthy immune system; downregulate estrogen and tumor formation. May yield a lower risk of breast, colon, prostate, and cervical cancers.
Cruciferous vegetables (*Brassica* family)	Jasmonates (thiols)	Plant signaling compounds that activate the coordinated gene expression in ascorbate and glutathione metabolic pathways. Important in defense responses to oxidative stress and biosynthesis of glucosinolate, a defense compound.
Dairy products	Vitamin D_3, calcium, sphingomyelin	May inhibit tumor cell growth and aid in cell death. Vitamin D_3 may protect against some skin cancers, but not melanoma. A level of 2000–4000 IU per day of vitamin D_3 is considered necessary for most adults. Sun exposure, diet, and supplements may be needed.
Flaxseed, rye, whole grains, berries, carrots, spinach, broccoli, tea, asparagus, linseeds; alcoholic beverages (red and white wines).	Lignans: matairesinol and secoisolariciresinol	Phytoestrogens attach to estrogen receptors and block real estrogen, lower cholesterol levels, and decrease cancer activity. Lignans may prevent prostate or lung cancer. Median intake of lignans is 578 micrograms (mainly from berries).
Garlic, onions	Allyl sulfides; selenium	Boosts levels of naturally occurring enzymes that may help maintain a healthy immune system. Garlic also contains arginine, oligosaccharides, sulfur and flavonoids. May be protective against cancers of the stomach, esophagus, colon, breast, and pancreas.
Ginkgo biloba	Ginkgolide A and B	Taken for 6 months or longer, may lower the risk of ovarian cancer. Involved in anti-inflammation processes.
Ginseng	Triterpene glycoside	Radioprotective capability, attributed to the ginsenosides, which are saponins with antioxidant properties. May increase lymphocyte production, stimulate natural killer cells and other immune activity; inhibit cancer cell growth; antioxidant.
Grapes, wine, raspberries, strawberries, tomatoes, citrus fruits, carrots, whole grains, nuts	Ellagic acid, Ferulic acid (phenolic acids)	May block the production of enzymes needed for cancer cells to reproduce. Protect against *Salmonella* and *Staphylococcus aureus* infections (particularly ellagitannins in raspberries).
Grape juices from green and black grapes	Gallic acid (phenolic acids)	Strong antioxidant properties
Green tea, oolong tea, black teas, dark chocolate, red wines, licorice root, cranberries and cranberry products	Catechins: epigallocatechin gallate (EGCG); glycyrrhizin; procyanidins/tannins (flavonols)	Decreases the growth of hydroquinone oxidase; decreases *COX-2* gene expression and cancer cell growth; and neutralizes free radicals. Tannins in green and black tea and strong coffee inhibit the proliferation of cancer cells in prostate, ovarian, breast, lung, and possibly other sites. Avoid excesses of caffeine, which dilute the effect of the tea.
Green tea extract, watermelon, prunes, raisins, plums, eggplant, grapes, berries, cherries, apples, cantaloupe	Polyphenols	May neutralize free radicals to help block damage to DNA. Superoxide anion radical (SOR) has scavenging activity; protects against oxidation of low-density lipoprotein and protects vision.
Green vegetables: Turnip, collard, and mustard; kale, spinach, broccoli. Green peas, kiwi, cilantro, parsley, lettuce. Corn; egg yolk.	Lutein and zeaxanthin (terpenes)	Antioxidants; anti-inflammatory; DNA repair. Good for healthy vision (protects against macular degeneration). Raw spinach is higher than cooked. Lutein may improve skin health and protect against skin cancer.
Leafy greens	Folic acid	Role in DNA synthesis, mitosis, and gene expression.
Legumes, whole grains, bananas, fish, chicken	Vitamin B_6	Supports a healthy immune system and increases lymphocyte numbers.
Licorice *Glycyrrhiza uralensis* and orange *Citrus* spp.	Coumarins; benzo-alpha-pyrones	Inhibits proteolysis and lipoxygenase; anti-inflammatory and antitumor effects.
Milk and dairy products, fish, liver	Vitamin A	Protects cells against free radical damage.
Nuts, nut butters, wheat germ, whole grains, oils, mayonnaise, creamy salad dressings, egg yolks, cereals, seeds	Vitamin E	Increases antibody production and B- and T-cell functioning; protects cells against free radical damage. SELECT study found that a 400 IU supplement actually promotes prostate cancer.
Oats	Avenanthramides; selenium	These polyphenols have strong antioxidant, anti-inflammatory properties (Meydani, 2009).

(continued)

TABLE 13-2 Phytochemicals, Functional Food Ingredients, and Cancer *(continued)*

Food	Functional Ingredient	Possible Roles in Reducing Cancer Risk
Oats, lima beans, navy beans, black beans, Brussels sprouts, ground psyllium seeds, peas, carrots, apples, barley, pectins, gums, mucilages	Soluble fiber	May lower cancer risk; helps lower cholesterol levels.
Olive and canola oils; tree nuts	MUFAs	Monounsaturated fats decrease tumorigenesis. Virgin olive oil phenols may reduce colorectal carcinogenesis.
Onions, garlic (particularly oil), leeks, chives, scallions, shallots	Allium and allicin (allyl sulfides, *S*-allylcysteine [SAC])	Organosulfurs decrease tumor cell growth; inhibit kinase activity; may protect the immune system, assist the liver in rendering carcinogens harmless, and reduce cholesterol production in the liver. Diallyl sulfide (DAS) inhibits the effects of PhIP that can cause DNA damage or transform substances into carcinogens.
Onions, garlic, leeks, shallots, inulin, Jerusalem artichokes, fructooligosaccharides (FOS); polydextrose in whole grains, some fruits, honey	Prebiotics	Support GI health and improve calcium absorption. Inulin may help protect against colon cancer.
Orange, grapefruit, lemon; cherries; citrus fruit peel	Limonoids/limonene (Terpenes); Naringenin, Hesperetin, Eriodictyol (flavanones)	Citrus glycosides decrease bacterial and fungal growth; decrease cancer cell growth by detoxifying enzymes in liver. Also cause cell apoptosis with certain types of cancers. Stimulate DNA repair with naringenin. Avoid grapefruit in estrogen+ breast cancer.
Oranges, grapefruit, lemons, tangerines, peaches, apricots, broccoli, tomatoes	Bioflavonoids, vitamin C	Minimizes damage to neutrophils; induces apoptosis; inhibits histamine
Pomegranate fruit and juice	Punicalagin (phenolic acid)	Potent antioxidant; protective against prostate cancer.
Red pepper, paprika	Capsaicin (8-methyl-*N*-vanillyl-6-nonenamide)	Red peppers of the genus *Capsicum*; it contains carotenoids that may be protective against cancer.
Salmon, mackerel, sardines	Coenzyme Q10 (ubiquinone) Omega-3 fatty acids	May reduce chemotherapy-related heart damage.
Seafood, canola oil, walnuts, flaxseed, marine and other fish oils	Omega-3 PUFAs	Reduce inflammation; may reduce cancer cachexia. Genetic variations affect response of COX-2 and its inflammatory impact on cancers such as prostate.
Soybean, corn and safflower oils	Omega-6 PUFAs	Keep omega-6 to omega-3 at a ratio of 5–10:1.
Soybeans (tofu, vegetable soy milk, soy nuts); legumes such as chick peas, beans, peas; nuts; grain products, coffee, tea; raisins and currants	Isoflavones: genistein, daidzein, biochanin A	Phytoestrogens attach to estrogen receptors and block real estrogen. Red clover and soy extracts contain isoflavones, with high affinity to estrogen receptor-alpha (ERα), estrogen receptor-beta (ERβ), progesterone receptor (PR), and androgen receptor (AR). Daily dose may be 40–50 milligrams of isoflavones (biochanin A, daidzein, formononetin, and genistein). Avoid excesses.
Spices: Cumin and turmeric, ginger, mint, rosemary, garlic, thyme, oregano, sage, basil, coriander, caraway, fennel, chili powder, black pepper, mint	Myricetin	Neutralizes free radicals, supports antioxidant defense system; preserves alpha-tocopherol, decreases inflammation; decreases ATPase; protects plasma DNA from radiation damage. May protect against prostate cancer.
Supplements, herbal	Astralgus, Echinacea, Silymarin	May increase macrophage activity and enhance immunity (interferon, killer cells, Interleukin-2). Silymarin protects the liver.
Sweet potatoes, carrots, turnips, spinach, papaya, tomato, red or green bell peppers, oranges	Retinoids; beta carotene	Important for cell growth, differentiation and death. May prevent some leukemias. Sweet potatoes are high in fiber, potassium, choline, vitamin C, magnesium, iron, and calcium as well.
Tomatoes and tomato products, ketchup, peppers; pink grapefruit; watermelon	Lycopene; vitamin C	Potent antioxidant; may reduce risk of prostate cancer. Tomato products must be cooked.
Vegetable oils, soy, peanuts, rice bran	Sterols: beta-sitosterol, campesterol, stigmasterol, squalene	Can reduce the risk of lung cancer and may reduce the tumor growth of other cancers. Sterols are found in vegetable oils. Sitosterol is the most studied.
Walnuts	Phytosterols, omega-3 fatty acid (ALA)	Antioxidant, anti-inflammatory properties that reduce tumor growth.
Wheat germ, lean beef, seafood, black-eyed peas	Zinc	Increases neutrophil function and killer cell numbers; decreases cytokines.
Whole grains, beans, seeds (soybeans, oats, barley, brown rice, whole wheat, flaxseed)	Phytates; insoluble fiber; vitamin E	Decreases oxidative damage to cells. Reduces the risk for breast or colon cancer.

(continued)

TABLE 13-2 Phytochemicals, Functional Food Ingredients, and Cancer *(continued)*

Food	Functional Ingredient	Possible Roles in Reducing Cancer Risk
Whole grains	Oligosaccharides; protease inhibitors	These increase short-chain fatty acid formation; decrease cholesterol and lower insulin levels; inhibit action of protein-splitting enzymes. May prevent cancer cell formation or decrease tumor size.
Wine, red grapes, grape juice, peanuts	Resveratrol; stilbenes (flavonoids)	Phytoalexin produced in plants in response to exposure to ultraviolet light or fungi. Decreases platelet activity, lowers cholesterol, suppresses proliferation of a variety of tumor cells.
Yogurt with *Lactobacilli*, *Bifidobacteria*; fermented milk such as Kefir.	Probiotic bacteria	Normalizes the intestinal microflora, blocks the invasion of potential pathogens in the gut, prevents colon cancer, modulates immune function, inhibits *H. pylori*, enhances calcium absorption; synthesizes niacin, folic acid, vitamin B_6, and biotin.

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TABLE 13-3 Cancer Risk Factors by Site

Factors That are Known to Increase the Risk of Cancer

Cigarette Smoking and Tobacco Use

- Acute myelogenous leukemia (AML).
- Bladder cancer.
- Cervical cancer.
- Esophageal cancer.
- Kidney cancer.
- Lung cancer.
- Oral cavity cancer.
- Pancreatic cancer.
- Stomach cancer.

Infections

- Human papillomavirus (HPV) increases the risk for cancers of the cervix, penis, vagina, anus, and oropharynx.
- Hepatitis B and hepatitis C viruses increase the risk for liver cancer.
- Epstein-Barr virus increases the risk for Burkitt lymphoma.
- Helicobacter pylori increases the risk for gastric cancer.

Two vaccines to prevent infection by cancer-causing agents have already been developed and approved by the U.S. Food and Drug Administration (FDA). One is a vaccine to prevent infection with the hepatitis B virus. The other protects against infection with strains of human papillomavirus (HPV) that cause cervical cancer. Scientists continue to work on vaccines against infections that cause cancer.

Radiation

- Ultraviolet radiation from sunlight: This is the main cause of nonmelanoma skin cancers.
- Ionizing radiation from medical X-rays and radon gas in our homes: Scientists believe that ionizing radiation causes leukemia, thyroid cancer, and breast cancer in women. Ionizing radiation may also be linked to myeloma and cancers of the lung, stomach, colon, esophagus, bladder, and ovary.

Factors That May Affect the Risk of Cancer

Alcohol

- Oral cancer.
- Esophageal cancer.
- Breast cancer.
- Colorectal cancer (in men).
- Liver cancer
- Female colorectal cancer.

Diet

Foods may protect against cancer and other foods may increase the risk of cancer.

- Fruits and nonstarchy vegetables may protect against cancers of the mouth, esophagus, and stomach.
- Fruits may protect against lung cancer.
- Diet high in fat, protein, calories and red meat may increase risk of colorectal cancer, but studies have not confirmed this.
- It is not known if a diet low in fat and high in fruits, vegetables and fiber lowers the risk of colorectal cancer.

Physical Inactivity

- Colorectal cancer
- Postmenopausal breast cancer
- Endometrial cancer.

Obesity

- Postmenopausal breast cancer.
- Colorectal cancer.
- Endometrial cancer.
- Esophageal cancer.
- Kidney cancer.
- Pancreatic cancer.
- Gallbladder cancer

Adapted from: National Cancer Institute, http://www.cancer.gov/cancertopics/pdq/prevention/overview/Patient/page3, accessed December 29, 2009.

For More Information on Cancer Prevention

- Cancer Prevention and Control
  http://www.cdc.gov/cancer/
- Cancer Research and Prevention
  http://www.preventcancer.org/
- Complementary Treatments for Cancer
  http://nccam.nih.gov/health/cancer/
- Patient Advocate Foundation
  http://www.patientadvocate.org
- Wellness Community
  http://www.thewellnesscommunity.org/

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CANCER TREATMENT AND TIPS FOR LONG-TERM SURVIVAL

CANCER: TREATMENT GUIDELINES

DEFINITIONS AND BACKGROUND

Cancer patients can be divided into three groups: those receiving standard or experimental therapy, those who have become unresponsive to these therapies, and those in remission who are at risk for recurrence or a second new cancer.

Cancer cachexia—a wasting syndrome characterized by weight loss, anorexia, early satiety, progressive debilitation, and malnutrition—may lead to organ dysfunction and death (Mattox, 2005). Fatigue is the most common experience among cancer patients. Otherwise, each type of cancer has its own set of treatments and side effects.

TABLE 13-4 Use of Nutrition Support in Cancer Patients

ASPEN Cancer Guideline Updated (2009)

Nutrition support therapy should not be used **routinely** in patients undergoing major cancer operations. • Grade: A

Perioperative nutrition support therapy may be beneficial in moderately or severely malnourished patients if administered for 7–14 days preoperatively, but the potential benefits of nutrition support must be weighed against the potential risks of the nutrition support therapy itself and of delaying the operation. • Grade: A

Nutrition support therapy should not be used **routinely** as an adjunct to chemotherapy. • Grade: B

Nutrition support therapy should not be used **routinely** in patients undergoing head and neck, abdominal, or pelvic irradiation. • Grade: B

Nutrition support therapy is appropriate in patients receiving active anticancer treatment who are malnourished and who are anticipated to be unable to ingest and/or absorb adequate nutrients for a prolonged period of time. • Grade: B

The palliative use of nutrition support therapy in terminally ill cancer patients is rarely indicated. • Grade: B

Omega-3 fatty acid supplementation may help to stabilize weight in cancer patients on oral diets experiencing progressive, unintentional weight loss. • Grade: B

Indications for Enteral Feeding	Contraindications for Enteral Feeding
Inability to consume 50% of estimated needs orally for 1 week or longer—estimated or actual	Severe malabsorption that cannot be corrected with enteral nutrition
Functioning gastrointestinal (GI) tract with adequate capacity for nutrient absorption	Intestinal obstruction below feeding placement site
Patient willingness to use tube feeding method	Condition such as high-output fistula or high aspiration risk

Adapted from: ASPEN Guidelines. http://www.nutritioncare.org/wcontent.aspx?id=4054, accessed January 1, 2010.
Dixon SW. Nutrition care issues in the ambulatory (outpatient) head and neck cancer. *Support Line.* 27:3, 2005.

Weight loss and cachexia are common. Malnourished cancer patients commonly have high protein turnover and loss of nitrogen, significant loss of muscle mass, and impaired physical capacity. Tumor factors such as proteolysis-inducing factor (PIF), tumor necrosis factor (TNF), and lipid mobilizing factor (LMF) all tend to promote catabolism. Nutritional inadequacy mobilizes protein stores and, thus, causes a loss of lean body mass. Altered nutrient utilization causes glucose intolerance, insulin resistance, increased glucose turnover, lipolysis, hyperlipidemia, and increased protein turnover. Properly nourishing patients, particularly when malnourished, is essential therapy. The Subjective Global Assessment tool and scoring sheet is used for cancer patients. Indications for use of nutrition support are listed in Table 13-4. Parenteral nutrition (PN) should not be used to prolong life for patients at the end stages of disease but may be appropriate for patients with responsive cancers when enteral and oral feedings are poorly tolerated.

Many cancer therapies cause unpleasant side effects. Table 13-5 defines the types of side effects and treatments in cancer therapy. Patients who are unresponsive to standard or experimental therapies have few treatment options and usually experience poor quality of life for the remainder of their lives. An active nutritional protocol including high doses of multiple dietary antioxidants (vitamin C, alpha-tocopherol, and natural beta-carotene), when administered as an adjunct to other therapies, may increase tumor response and decrease toxicity. A maintenance nutritional protocol with lower doses of antioxidants, in addition to a modified diet and lifestyle, may reduce the risk of recurrence of the original tumor and development of a second cancer among survivors.

TABLE 13-5 Side Effects of Treatment and Common Problems of Cancer

Side Effect	Comments
Anemias	About half of the patients coming to cancer treatment are anemic. Use a balanced diet with high-quality proteins, B-complex vitamins, and vitamin C. Eat small meals every 2–3 hours. Heme sources of iron will increase iron bioavailability. Use beef, chicken, fortified grains, dried fruits such as prunes, nuts, and seeds, and blackstrap molasses. Avoid the long-term use of iron supplementation.
Anorexia	Medications, GI distress, altered sensory experiences often leads to cachexia. Treat symptoms, such as pain, constipation, and GI symptoms. Encourage small, frequent feedings. Consider pharmacological therapy with appetite-enhancing medications. Rinse mouth with baking soda or water before eating. Ginger ale or mint can mask metallic tastes; use plastic utensils if needed. Add flavorings to food, or suck on hard candy. Try chilled, frozen, sweet, or tart foods. Avoid unpleasant odors.
Aversion to foods or flavors	A lower threshold for urea causes aversion to meat; it "smells rotten." Substitute milk, cottage cheese, eggs, peanut butter, legumes, poultry, fish, and cheese. In addition, patient may have a decreased ability to taste salt and sugar. Add other seasonings, sauces, and more salt or sugar as desired by the patient; however, do not allow sweet foods to replace nourishing foods. Clear palate prior to meals by brushing teeth, gums, and oral cavity. Rinse with baking soda and salt water.
Cachexia	Cachexia is the clinical consequence of a chronic, systemic inflammatory response. Depletion of skeletal muscle and redistribution of the body's protein occur. Nutritional deprivation at diagnosis can lead to further depletion with treatments. Anorexia cachexia syndrome (ACS) is caused by numerous factors. Use small, frequent feedings and supplements. Teach ways to increase calories and protein. Fortify foods when possible. Relieve symptoms before meals whenever possible. Anabolic and anticatabolic agents, such as Megace and Oxandrin, may help. Use of omega-3 fatty acids (EPA and DHA) can disrupt the cachexia.
Chemotherapy	With all types (given daily, weekly, monthly for 1–2 months or even years), prompt attention to side effect management and appropriate use of supportive care (medications, nutrition, etc.) will be needed. Increase fluid intake for adequate hydration. After chemotherapy, cardiac, kidney, or pulmonary toxicity may occur. Some chemotherapy agents may cause infertility in both men and women. Nausea and vomiting can now be well controlled with Zofran (ondansetron), Kytril (granisetron), and Anzemet (dolasetron). Hemopoietic agents (e.g., Neupogen, Procrit, granulocyte colony-stimulation factor, granulocyte-macrophage colony-stimulating factor) may be needed if red blood cell production is too low; transfusions are a last resort. Avoid the risk of infection and cuts during chemotherapy. Monitor for nosebleeds, bruising, black or bloody stools, or reddish urine. Glutamine supplementation has been used with some success.
Cold food preference	Cold foods may be better accepted than hot foods. Use cold, clear fluids, carbonated beverages, ices, gelatin, watermelons, grapes and peeled cucumbers, cold meat platters, ice cream, and salted nuts. Serve supplements over ice between meals. Shakes, puddings, and custards are other alternatives.
Constipation	Establish an appropriate bowel program, including regular use of pharmacological agents. Add fiber and extra fluids to the diet. Milk is beneficial, if tolerated. Fresh or dried fruits, all vegetables, bran and a hot drink may help. Get adequate exercise, such as walking. Drink hot beverages, or use prunes or prune juice. Over-the-counter bulking agents may be useful in some cases. Avoid gas-forming foods in excess. Report any blood, vomiting, or no stool for 3+ days to the doctor.
Dental Caries	Avoid sweets and use sodium fluoride three times daily. Mouth care should be provided several times daily. Persons receiving irradiation to the head and neck area may benefit from use of fluoride trays and stannous fluoride.

(continued)

TABLE 13-5 Side Effects of Treatment and Common Problems of Cancer *(continued)*

Side Effect	Comments
Diarrhea	Evaluate all medications carefully. Assess hydration status and associated symptoms. Alter fiber in diet. Beware of lactose intolerance secondary to disease process, drug therapy, or abdominal or pelvic radiation therapy. Increase fluids that contain sodium and potassium; use of Gatorade or Pedialyte may be helpful. Use cool or room temperature foods. Avoid dairy products if lactose-intolerant. Consume small amounts of food throughout the day instead of three large meals. Decrease fatty, spicy, or acidic foods; caffeine; gas-forming vegetables; or carbonated beverages. Plain rice, potatoes, eggs, mild fish or skinless chicken may be well tolerated. Limit sorbitol and sugar substitutes. Oral glutamine may be useful.
Difficulty swallowing (dysphagia)	Modify diet consistency and follow swallowing techniques provided by speech pathologist. Use moist foods; add sauces or gravies. Semi-solid foods may be better tolerated than liquids, and pureed foods rather than regular items. Patient should sip fluids throughout meal. To prevent aspiration, try placing liquid under the tongue. Some patients find that tilting their heads back helps. Thickeners are available for liquids, if thin beverages are not tolerated well (as with choking, coughing with each swallow). Use of a straw may be beneficial. Spoons are easier to control than forks in the mouth. Avoid very hot or very cold foods. Chew sugarless gum or candy. Use artificial saliva if needed. Consider feeding tube if needed.
Dry mouth (xerostomia)	Surgical removal of salivary glands, atrophy of mucous membranes, or permanent damage from radiation to salivary glands may cause difficulty in eating and swallowing. Use salivary substitutes, lip balm, sugarless gum and candies, gravies, and sauces. Increase fluids and use softened, moist foods (custard, stews, and soups). Cut food into small pieces, or use pureed foods. Ice chips and popsicles also can help. Avoid salty foods. Tart foods, lemon drops, and lemonade may help to stimulate saliva production; avoid tart items if there are oral lesions. Sip on water or other fluids frequently throughout the day and with each bite of food. Synthetic saliva products such as Optimoist or MouthKote may help. Patients benefit from a thorough dental examination before treatment. Use fluoride trays, rinses, other measures. Avoid caffeine, alcohol and tobacco products. Salagen (pilocarpine) is approved to reduce radiation-related dry mouth.
Early Satiety	Rather than serving plain water, encourage a calorie-containing beverage. Take liquids between meals. Avoid fatty, greasy foods because they are more slowly digested and absorbed. Use small meals and frequent snacks between meals. Add protein and calories using extra butter, margarine, cheese, dry milk powder.
Edema	Fluid retention may require elevating the legs at rest, staying physically active (walking), and reducing salt intake overall. The doctor may prescribe a diuretic.
Fatigue	Fatigue is very common. Assess and treat causes such as anemia, infection, pain, neutropenia, depression, or medication side effects. Meals may be prepared in quantity when the patient is less tired. Use foods that require less chewing and provide frequent rest periods, particularly before meals. Exercise daily to build stamina. Maintain adequate sleep/nap patterns.
Graft-versus-host disease (GVHD), neutropenia	Fever, chills, sweating, coughing, shortness of breath, diarrhea. Avoid people with colds or flu. Wash hands frequently and use safe food handling procedures. Wash all produce carefully and cook thoroughly. Avoid raw eggs. Cook meats well.
Insulin resistance	Insulin resistance is common from the tumor itself, or may occur after pancreatic surgery. Control of CHO intake and oral agents may be indicated.
Loneliness, Emotional Changes	Social eating may improve food intake. Visitors should be encouraged to bring gifts of food, as appropriate. If anxiety occurs, discuss with healthcare provider.
Loss of lean body mass	Protein wasting and unintentional weight loss are common. Exercise is extremely helpful. Endurance activities can counteract loss of physical performance and improve lower and upper body strength. Patients who exercise also have less fatigue and depression. Taking hormones such as growth hormone, insulin-like growth factor (IGF-I), thyroid hormone, androgens, and cortisol makes a difference as well. Muscle protein synthesis can be increased accordingly.
Malabsorption	Elemental diets can only be used if patient has an intact duodenum and jejunum. Total parenteral nutrition should be used only in some cases, considering risk of infection. Tart beverages such as lemonade can be mixed with elemental products if they are to be taken orally.
Meal interruptions	Encourage a good breakfast and snacks to make up for interrupted meals. Keep kitchen well stocked. Meals-on-Wheels may be a useful way to serve meals to this population at home.
Mouth blindness (dysgeusia)	To alleviate disinterest and aversion to foods, emphasize the aroma and colors of foods. Foods that are served warm or hot have more flavor and aroma. Provide a variety of foods and use garnishes. Acidic foods (such as lemonade) may help stimulate patient's ability to taste foods. Use highly flavored foods and sauces. Try milk shakes that are coffee or mint flavored. Fresh vegetables, special breads, highly flavored snacks, olives and pickles may be well received.
Mouth or throat soreness or dry mouth (stomatitis, mucositis, esophagitis)	Swish mouth with lidocaine, mild saline, or sodium bicarbonate before meals. Changes in taste or enjoyment of foods may occur. Avoid acidic juices, salty foods or soups, dry toast, and coarse or grainy breads or cereals. Grind meats; use a "mechanical soft diet" as needed. Offer cold or tepid fluids frequently; use a straw if needed. Popsicles may help. Smaller meals are useful. Cut foods into small pieces; grind or puree if needed. Mix food with sauces or gravies to make it easier to swallow. Avoid smoking and use of alcohol. Oral glutamine has been used with some success. Avoid alcohol-based mouthwashes. Salagen (pilocarpine) reduces dry mouth and throat sprays or cough drops may be useful.

(continued)

TABLE 13-5 Side Effects of Treatment and Common Problems of Cancer *(continued)*

Side Effect	Comments
Muscle wasting	Muscle weakness is frequently associated with tumor growth. Include adequate amounts of protein and amino acids in the diet or in enteral feeding, particularly arginine, glutamine and leucine. Depression, altered moods, immobility, and bed rest contribute to loss of muscle mass. Structured exercise, including resistance training and aerobic exercises, can improve muscle mass and strength. Increased physical activity improves emotional stability, self-confidence, and independence. Active patients have less fatigue, nausea, and insomnia; quality of life improves.
Nausea	Treated with slow, deep breathing, ice chips, or sips of ginger ale, tea, or candied dried ginger. Try a dry diet with liquids between meals. Eat small meals; rest upright afterward. Offer toast, yogurt, sherbet, popsicles, pretzels, angel food cake, canned fruit, baked chicken, lemonade, hot cereal such as oatmeal, clear liquids, or broth. Cut down on greasy, spicy, fried, fatty foods; foods with strong odors and excessive sweetness. Sit upright for meals and snacks; avoid tight clothing. If breakfast is the best meal, it can also be the largest of the day. Keep crackers or light snacks at hand; do not skip meals. Use antiemetics as directed by physician; underusage may aggravate the nausea. Drink plenty of water/liquids the day before and after chemotherapy.
Pain	Prevention is the key. Give pain medications with the first few bites of a meal or have the patient eat when pain is lowest. Encourage trying foods again after time lapse. Try biofeedback, acupuncture, massage, muscle relaxation techniques. Keep a pain journal and report any side effects to the doctor.
Radiation therapy	Radiotherapy may involve high-energy radiation from x-rays, cobalt-60, or radium. Brachytherapy provides internal, continuous local delivery of radiation to site of malignancy (concealed). Teletherapy provides external radiation to a localized area; 7000 rads usually causes damage, particularly to small intestine (radiation enteritis). Radiation therapy (daily for 2–8 weeks) can cause nausea or vomiting if administered to the brain or abdominal/pelvic fields. A light meal is encouraged before treatment. Diarrhea may occur in radiation enteritis; glutamine may be useful in supplements or in tube feeding (TF)/CPN. Formulas containing multiple antioxidants for biological protection against radiation damage in humans are needed. Use of oral glutamine has been used with some success. **In head and neck cancer: anorexia, dysgeusia, weight loss, odynophagia, dysphagia, difficulty chewing, xerostomia. In the thorax: nausea, esophagitis, vomiting. In abdominal, intestinal: lactose intolerance, diarrhea, distention, abdominal pain, nausea and vomiting; later–intestinal stenosis, edema, fluid and electrolyte loss, weight loss.
Radiation enteritis or colitis	About 50–80% of patients who have radiation to the pelvis end up with radiation enteritis; onset can occur up to years later. Symptoms include nausea, vomiting, mucoid diarrhea, abdominal pain, and bleeding; later, there may be colic, a decrease in stool caliber, and progressive obstipation with stricture and fibrosis. Serious injury to the intestinal epithelium and arterioles of the small or large intestines results in cell death, fibrosis, and obstruction. Radiation to the ileum is particularly devastating. If radiation must be given chronically, resection may be needed and the ability of the intestines to become hyperplastic and increase absorptive capacity is thus prevented. Of these persons, many will require home CPN.
Surgery, curative	Direct efforts at restoring nutritional health to pre-illness status. After GI surgery, effects include—Oropharynx: Difficulty with chewing and swallowing, dysgeusia, xerostomia. Esophagus: Heartburn, loss of normal swallowing, decreased motility, obstruction. Stomach: Dumping syndrome, delayed emptying, anemia, malabsorption. Small intestines: Lactose intolerance, bile acid depletion, steatorrhea, fat malabsorption, vitamin B_{12} deficiency and anemia, short-gut syndrome. Colon: Loss of electrolytes and water; diarrhea, constipation, gas, bloating.
Thick saliva	Thick, ropy saliva can produce more caries. Use less bread, milk, gelatin, and oily foods. Puree foods such as fruits and vegetables. Encourage the intake of plenty of fluids to decrease the viscosity of oral secretions. Encourage good oral intake and regular oral rinses.
Tooth loss	Loss of teeth makes the patient's mouth more sensitive to cold, heat, and sweets. Try serving foods at room temperature. Use ground, chopped, or pureed foods as needed until dental repair is possible.
Vomiting	Sip clear liquids every 10–15 minutes after vomiting episodes cease; keep head elevated. "Flat" carbonated beverages are useful. Call doctor if abdominal pains persist. Take antiemetic medications prior to meals. Use small feedings, avoid spicy or acidic foods and those with strong odor, use liquids between meals. Use low fat, light meals.
Weight loss	Calculate 40–45 kcal/kg for repletion. Add fats to foods, dry milk to mashed potatoes and shakes, and extra sugar to coffee and cereals. Use small, frequent feedings and the patient's favorite foods. Add cream sauces, extra meat or cheeses in casseroles, and gravies. Encourage patients to be as physically active as possible, particularly using long muscles to promote lean body mass.

Derived from: National Cancer Institute, http://www.cancer.gov/cancertopics/pdq/supportivecare/nutrition/Patient, accessed January 5, 2010.

ASSESSMENT, MONITORING, AND EVALUATION

CLINICAL INDICATORS

Genetic Markers: HMGB1 protein—a danger signaling protein—can act as a proinflammatory and proangiogenic mediator when actively secreted by macrophages or passively released from necrotic cells; this plays an important role in the pathogenesis of cancer (Winter et al, 2009). Other genetics information for cancer can be found at http://www.cancer.gov/cancertopics/genetics-terms-alphalist/a-e.

Sample Clinical/History

- Height
- Weight
- Weight changes
- Body mass index (BMI)
- Diet history
- Input and output (I & O)
- Pain?
- Limited use of affected area
- Fatigue
- Warmth in a local area?
- Fever, temperature
- Cough
- X-ray
- Dual-energy x-ray absorptiometry (DEXA) scan
- Computed tomography (CT) scan or magnetic resonance imaging (MRI)

Lab Work

- Glucose (Gluc)
- Hemoglobin and hematocrit (H&H)
- Serum Fe, ferritin
- Serum folate, B_{12}, B_6
- Serum homocysteine
- Ca^{++}, Mg^{++}
- Na^{+}, K^{+}
- Albumin (Alb)
- C-reactive protein (CRP)
- Total lymphocyte count (TLC) (varies)

SAMPLE NUTRITION CARE PROCESS STEPS

Knowledge Deficit

Assessment: Medications, lab values, current use of herbs and botanical products.

Nutrition Diagnosis (PES): Knowledge deficit as evidenced by patients requesting information regarding the proper use of herbs and botanicals for cancer treatment.

Intervention: Education about herbs and botanical products in cancer; resources and Web sites; label reading. Counseling with responses to specific questions according to type of cancer, prevention versus treatment, side effects.

Monitoring and Evaluation: No reports of adverse side effects with herbs and botanicals, medications, or foods (such as allergic reactions).

INTERVENTION

OVERALL OBJECTIVES IN CANCER TREATMENT

- Coordinate total care plan with doctor, nurse, patient, family, caregivers, and other team members.
- Review each case individually and honor patient's wishes regarding more aggressive intervention.
- Prevent or minimize weight changes. Some patients are hypometabolic; others are hypermetabolic by 10–30% above normal rates. Greatest losses occur from protein stores and body fat.
- Use indirect calorimetry to determine energy requirements; Resting Metabolic Rate (RMR) or Resting Energy Expenditure (REE) is measured after 30 minutes of recumbent rest, preferably fasting but not necessarily early in the morning, with as little physical activity as possible before the measurement (American Dietetic Association, 2010).
- Diminish toxicity of treatments and improve quality of life. Good nutritional status early on is a good prognostic indicator.
- Correct cachexia from weakness, anorexia, redistribution of host nutrients, and nutritional depletion.
- Prevent depletion of humoral and cellular immunity from malnutrition. Improved nutritional status may allow neoplastic cells to become more susceptible to medical treatment.
- An improved nutritional status reduces side effects, promotes better rehabilitation, and improves quality of life while perhaps increasing survival rates. Malnutrition can potentiate the toxicity of antineoplastic agents.
- Prevent infection or sepsis, further morbidity, or death.
- Control complications such as anemia or multiple organ dysfunction.
- Preserve body mass through structured exercise programs and specialized nutritional supplementation.
- Control gastrointestinal symptoms, which are more common with weight loss greater than 10%.
- Work with the interdisciplinary team using a sample algorithm (courtesy of RD411.com). (See Algorithm on page 743)

FOOD AND NUTRITION

- Determine cancer-specific energy and protein requirements (American Dietetic Association, 2010).
- If indirect calorimetry is not available, calculate energy as 30 kcal/kg body weight to maintain and 35–45 kcal/kg body weight to replete lost stores or if the patient is febrile, septic, or very active.
- In general, the intake of protein should be high (1–1.5 g/kg body weight to maintain; 1.5–2 g/kg body weight to replete lean body mass) to protect from muscle wasting, malnutrition, cachexia, and treatments.
- Provide appropriate and adequate, but not excessive, micronutrient supplementation. Avoid excesses of iron, but correct anemias when diagnosed. There is no evidence for use of vitamin E or arginine prior to radiation

INTERDISCIPLINARY NUTRITION CARE PLAN
Cancer

Client Name: _____ **#:** _____ **Initiated by:** _____ **Date:** _____

SCREEN
Nutrition Screen diagnosis: Cancer
Signed: _____ Date: _____

ASSESS *(Check any/all)*
- ❑ **Receiving chemotherapy/radiation therapy**
- ❑ **Weight loss: _____lb/wk**
- ❑ **Receiving enteral or parenteral nutrition or complex diet order**
- ❑ **Dehydration**

Nutrition Impact Symptoms*
- ❑ Problems chewing/swallowing
- ❑ Mouth pain/dryness
- ❑ Nausea/vomiting
- ❑ Diarrhea/constipation
- ❑ Fatigue
- ❑ Anorexia
- ❑ Altered taste perception

Signed: _____ Date: _____

1 or more

HIGH-RISK INTERVENTIONS *(Check any/all)*
- ❑ **Build Up Your Diet** provided and explained
- ❑ **Food Record** provided and explained
- ❑ **Fluid intake stressed**

Obtain Dr. orders as needed:
- ❑ RD referral for home visit(s)
- ❑ Monitor weight q:_____
- ❑ Monitor I & O q: _____
- ❑ BID/TID supplements
- ❑ **Other:**_____
  (See notes for documention.)

Signed: _____ Date: _____

Next visit

ASSESS RESPONSE *(Check any/all)*
- ❑ Further weight loss
- ❑ Continued dehydration
- ❑ Exhibiting Nutrition Impact Symptoms*
- ❑ **Other:**_____
  (See notes for documention.)

Signed: _____ Date: _____

1 or more

OUTCOMES NOT ACHIEVED
Reassess/evaluate need for EN/PN (refer to Tube Feeding Nutrition Care Plan). Document on Nutrition Variance Tracking form.

GOALS (Check any/all):

❑ Maintain or improve nutritional status in_____ (goal time).

❑ Prevent hospitalization due to dehydration/poor nutrional intake in ____ (goal time).

❑ Prevent or alleviate nutrition-related complications of cancer or cancer therapy in ____ (goal time).

❑ Avoid delay of cancer therapy due to poor nutrition in _____ (goal time).

None

MODERATE RISK INTERVENTIONS
(Check any/all)
- ❑ **Build Up Your Diet** provided and explained
- ❑ **Food Record** provided and explained
- ❑ **Fluid intake** encouraged

Obtain Dr. orders as needed:
- ❑ RD chart consult
- ❑ Monitor weight q:_____
- ❑ BID/TID supplements
- ❑ **Other:**_____
  (See notes for documention.)

Signed: _____ Date: _____

Next visit

ASSESS RESPONSE *(Check any/all)*
- ❑ Further weight loss
- ❑ Exhibiting Nutrition Impact Symptoms*
- ❑ Dehydration
- ❑ **Other:**_____
  (See notes for documention.)

Signed: _____ Date: _____

1 or more

None

OUTCOMES ACHIEVED
- ❑ Weight stabilized or improved
- ❑ Hydration status maintained or improved
- ❑ Cancer therapy initiated without delay
- ❑ **Other:**_____
  (See notes for documention.)
- ❑ Repeat Nutrition Risk Screen in ____ days

Signed: _____ Date: _____

None

OUTCOMES ACHIEVED
- ❑ Weight stabilized or improved
- ❑ Hydration status maintained or improved
- ❑ Cancer therapy initiated without delay
- ❑ **Other:**_____
  (See notes for documention.)
- ❑ Repeat Nutrition Risk Screen in ____ days

Signed: _____ Date: _____

or chemotherapy (American Dietetic Association, 2010). Use foods that are high in phytochemicals and antioxidants.
- Use adequate fluid for hydration.
- Schedule larger meals earlier in the day. If needed, schedule five to six small meals daily, with tube feeding or intravenous feeding. If the gut works, use it.
- Parenteral nutrition (PN) is not likely to benefit advanced cancer patients who are unresponsive to treatment and should be used with caution in current or potentially septic patients because of the risks (American Dietetic Association, 2010). Use CPN if enteral nutrition is contraindicated and if the patient is at low risk for infection.
- After surgery or abdominal radiation, glutamine may be useful to protect from enteropathy, lower morbidity, augment tumor cell kill, and boost natural killer (NK) cell activity.

Common Drugs Used and Potential Side Effects

- Drugs used will be matched to the specific type of cancer. Tables 13-6 and 13-7 list drugs and some common side effects.
- See the NCI list available at http://www.cancer.gov/drugdictionary/.

Herbs, Botanicals, and Supplements

- Answer questions about the use of herbs and botanicals in cancer treatment plans. Use of complementary and alternative medicine (CAM) therapy is common in the cancer population.
- Some products are harmless, but some may lead to serious problems. Table 13-8 describes herbs that are commonly used and some general comments.

TABLE 13-6 Cancer Drugs and Chemotherapy Agents

There are many chemotherapy drugs available. With chemotherapy, patients may suffer severe side effects such as nausea, hair loss, infection, and injury to the GI tract. Serotonin antagonists such as Anzemet (dolasetron), if administered at the same time as chemotherapy, can prevent nausea and vomiting, but abdominal pain, headache, and constipation may occur. Biologic therapies such as interferon and interleukin may cause flu-like symptoms and myalgias, shortness of breath, or edema. Monoclonal antibodies such as Herceptin and Rituxan are also used to treat cancer and may cause chills, fever, lethargy and muscle aches. With antineoplastic agents, side effects include nausea, anorexia, stomatitis, diarrhea, taste alterations, some vomiting, and possibly sloughing of colonic mucosa (see Table 13-7 also).

Drug	Description
Alkylating agents: cyclophosphamide, fluorouracil	These drugs kill cancer cells by stopping their growth or by making it hard for cancer cells to repair damage. Nausea, vomiting, hyperuricemia.
Antiangiogenic agent: humanized monoclonal antibody bevacizumab (Avastin)	Tumors require nutrients and oxygen in order to grow; angiogenesis provides access to these nutrients.
Antimetabolites: flucytosine	This is a DNA substrate analog that leads to incorrect DNA synthesis affecting the cancer cells. Nausea, vomiting, diarrhea, and stomatitis can occur.
Antiemetics: granisetron or ondansetron; medical cannabis, Marinol; domperidone, promethazine (Phenergan); metoclopramide (Reglan)	May be useful for anorexia/cachexia syndrome. Also used to relieve nausea and vomiting after chemotherapy. Headache may result. Other side effects include nausea, diarrhea, increased gastric emptying, or drowsiness.
Aspirin and anti-inflammatory agents	May prevent some types of cancer, including colon cancer. The use of herbal nonsteroidal anti-inflammatory drugs (NSAIDs) may be recommended with these medications to enhance effectiveness.
Irinotecan (Camptosar)	For the treatment of stage 1–4 breast, lung, prostate, colon, skin, and most other metastatic or nonmetastatic forms of cancer.
Corticosteroids: prednisone	Hyperglycemia, sodium and fluid retention, weight gain, and calcium losses can occur.
Folate antagonist: methotrexate	Use of folate preparations can alter drug response. Folate, lactose, vitamin B_{12}, and fat are less well absorbed. Mouth sores are common.
Immunotherapy: interleukin-2 and interferon	Lymphokine is administered to decrease tumor growth. Nausea, vomiting, abdominal pain, fatigue, and anorexia can result. In addition, low levels of folate and vitamins A and B_6 may result.
Monoclonal antibodies (MAbs): cetuximab, Campath-1 H, rituximab (Rituxan), and Bexxar	These drugs correct the abnormal enzyme that causes cancerous cells to grow out of control. These attack only abnormal elements of cells ("kinder and gentler" cancer therapies). Cetuximab specifically binds to the epidermal growth factor receptor with high affinity.
Vinca alkaloids: vincristine, vinblastine	Nausea and vomiting can occur.

Derived from: American Cancer Society, http://www.cancer.org/docroot/CDG/cdg_0.asp, accessed January 3, 2010.
Chemotherapy Drugs http://www.chemocare.com/BIO/.
National Cancer Institute http://www.cancer.gov/DRUGDICTIONARY/.
Oncology Channel http://www.oncologychannel.com/chemotherapy/medsideeffects.shtml.

TABLE 13-7 Antineoplastic Agents: Generic and Brand Names

Generic	Brand	Generic	Brand
Altretamine	Hexalen	Interferon-α2 a	Roferon-A
Asparaginase	Elspar	Interferon-α2b	Intron-A
Bevacizumab	Avastin	Interferon-αn3	Alferon-N
BCG	TheraCys, TICE BCG	Irinotecan	Camptosar
Bleomycin sulfate	Blenoxane	Leucovorin calcium	Wellcovorin
Busulfan	Myleran	Leuprolide	Lupron, Lupron-Depot
Carboplatin	Paraplatin	Levamisole	Ergamisol
Carmustine	BiCNU	Lomustine	CeeNU
Chlorambucil	Leukeran	Megestrol	Megace
Cisplatin (*cis*-platinum, *cis*-diammine-dichloroplatinum)	Platinol, Platinol-AQ	Melphalan, L-phenylalanine mustard, L-sarcolysin	Alkeran (R)
Cladribine, 2-chlorodeoxyadenosine	Leustatin	Melphalan hydrochloride	IV Alkeran
Cyclophosphamide	Cytoxan, Neosar	Mercaptopurine	Purinethol Tablets
Cytarabine, cytosine arabinoside	Cytosar-U	Mesna	Mesnex
Dacarbazine, imidazole carboxamide	DTIC-DME	Mechlorethamine, nitrogen mustard	Mustargen
Dactinomycin	Cosmegen	Methylprednisolone	Solumedrol, Medrol
Daunorubicin, daunomycin	Cerubidine	Methotrexate, amethopterin	Trexall
Dexamethasone	Decadron, Tobradex	Mitomycin	Mutamycin
Doxorubicin	Adriamycin	Mitoxantrone	Novantrone
Erlotinib	Tarceva	Paclitaxel	Taxol
Etoposide (epipodophyllotoxin)	VePesid	Plicamycin, mithramycin	Mithracin
Floxuridine	FUDR	Prednisone	Deltasone
Fludarabine	Fludara	Procarbazine	Matulane
Fluorouracil	Fluorouracil Injection	Streptozocin, streptozotocin	Zanosar
Fluoxymesterone	Halotestin	Tamoxifen	Nolvadex
Flutamide	Eulexin	6-Thioguanine	Tabloid
Goserelin	Zoladex	Thiotepa, triethylene thiophosphoramide	Thiotepa
Hydroxyurea	Hydrea	Vinblastine	Velban
Idarubicin HCL	Idamycin	Vincristine	Oncovin
Ifosfamide	IFEX	Vinorelbine tartrate	Navelbine Injection
Interferon-alfa	Roferon-A, Intron-A		

Source: Medicine Online. Antineoplastic agents generic abbreviations, http://www.medicineonline.com/reference/Health/Conditions_and_Diseases/Cancer, accessed January 3, 2010.

NUTRITION EDUCATION, COUNSELING, CARE MANAGEMENT

- Educate family about special patient needs (Dixon, 2005).
- There is evidence that cancer survivors who adopt a healthy lifestyle reap physical and emotional benefits. See Table 13-9 for more guidance and patient education tips.

Patient Education—Food Safety for Cancer Patients

- Clean: Wash hands and surfaces often.
- Separate: Don't cross-contaminate. Keep raw meat and poultry apart from cooked foods.
- Cook: Use a food thermometer to be sure meat and poultry are safely cooked.
- Chill: Refrigerate or freeze food promptly.
- Avoid:
  - Hot dogs, luncheon, and deli meats unless they are reheated until steaming hot.
  - Refrigerated plate, meat spreads from a meat counter, smoked seafood, and raw or undercooked seafood.
  - Raw (unpasteurized) milk and foods that contain unpasteurized milk.
  - Soft cheeses such as Feta, queso blanco, queso fresco, Brie, Camembert cheeses, blue-veined cheeses, and Panela unless it is labeled as made with pasteurized milk.
  - Salads made in the store such as ham salad, chicken salad, egg salad, tuna salad, or seafood salad.
  - Soft-boiled or "over-easy" eggs, as the yolks are not fully cooked.

TABLE 13-8 Herbs, Dietary Supplements, and Cancer

Most patients diagnosed with cancer explore complementary and alternative medicine (CAM), particularly herbal medicine. Dietetics professionals must evaluate the risks and benefits of the use of herbs and botanical products in various cancers; indicate whether **"guidance" or "promotion"** is being offered. Alternative therapies should be reviewed in the light of potential harm. Herbs should be appropriately labeled to alert consumers to potential interactions when used with drugs, and consultation with a general practitioner is recommended.

Ashwagandha (Withania somnifera)	Used for cancer treatment, diabetes, epilepsy, fatigue, gastrointestinal (GI) disorders, pain, rheumatoid arthritis (RA), skin infections, and stress.	Should not be used in pregnant women because it is an abortifacient.
Astragalus	Stimulates interferon and positively impacts the immune system. Possibly reduces the effectiveness of chemotherapy. Strong immune booster.	A type of legume used for years in Chinese medicine. No convincing evidence in cancer.
Black cohosh (Remifemin)	May relieve the symptoms of menopause. No known side effects with chemotherapy. Source of vitamin A and pantothenic acid. Drug interactions: may increase the toxicity of doxorubicin and docetaxel.	Used to lower hot flashes, which can be a challenge for breast cancer patients.
Bromelain	Bromelain (from pineapple extract) positively impacts the immune system. Improved tumor boundaries.	Studies have not demonstrated evidence in cancer therapy.
Cat's claw	May have some effect on the immune system, but more comprehensive studies are needed. Antioxidant.	Contains alkaloids.
Chamomile	No proven efficacy in cancer. May promote sedation or allergic reactions.	
Chili powder	Capsaicin may actually have tumor-promoting effects; chili powder has been implicated in several GI cancers, but the results are conflicting.	In Mexico, higher use of chili powder is related to more stomach cancer.
Chinese herbal medicine	Chinese herbal medicine uses a variety of herbs, in different combinations, to restore balance to the body.	See Astragalus, Ginkgo, Ginseng, Green tea, and Siberian ginseng.
Chinese PC-SPES	Contains chrysanthemum, isatis, licorice, Panax ginseng, saw palmetto, skullcap; Rabdosia rubescens is the most potent ingredient. PC-SPES contains flavonoids, alkaloids, polysaccharides, amino acids, and trace minerals such as selenium, calcium, magnesium, zinc, and copper.	Antiestrogenic effects.
Cloves	Contains eugenol, which reduces lipid peroxidation and reduces cancer cell proliferation.	
Dehydroepiandrosterone (DHEA)	DHEA is a steroid hormone produced by the adrenal gland and converted into estrogen and testosterone.	It is normally found in humans, plants, and animals. DHEA extracted from a wild yam plant is available as a dietary supplement.
Echinacea	No evidence of usefulness in reducing incidence or symptoms of cancer.	May reduce colds or flu for some people.
Eleuthero (Siberian ginseng)	May boost energy. More studies are needed.	
Essiac	Mixture of four herbs: burdock root (Arctium lappa), sheep sorrel (Rumex acetosella), slippery elm bark (Ulmus rubra), and Indian rhubarb root (Rheum officinale) to make a tea. Watercress (Nasturtium officinale R. Br.), blessed thistle (Cnicus benedictus L.), red clover (Trifolium pratense L.), and kelp (Laminaria digitata [Hudson] Lamx.) have been added to a product sold as Flor Essence.	Potent antioxidant and DNA-protective activity. Possibly estrogenic, antioxidant, anti-inflammatory, antimicrobial, and anticarcinogenic.
Evening primrose oil or gamma linolenic acid (GLA)	Proposed to reduce the effects of cancer treatments. GLA is an omega-6 unsaturated fatty acid made in the human body from other essential fatty acids. The main supplemental sources of GLA are oils of the seeds of evening primrose, borage, and black currant plants.	GLA is found in human breast milk. Claimed to slow cancer cell growth. Increases effectiveness of chemotherapy; boosts efficiency of tamoxifen; antioxidant; boosts immune system.
Falcarinol	A cancer-fighting substance found only in carrots.	Human studies are needed.
Flaxseed	Flaxseed supplements along with low-fat diets may be useful in men with early-stage prostate cancer.	Controlled clinical studies are needed.

(continued)

TABLE 13-8 Herbs, Dietary Supplements, and Cancer *(continued)*

Garlic	Seems to have reduced gastric and prostate cancers. Sulfur compounds tend to be the most chemoprotective. Useful in treatment as well as prevention. Garlic appears to induce cytochrome P-450 3A4 and may enhance metabolism of many medications such as cyclosporine and saquinavir. Antimicrobial properties are helpful.	Supplements are not as effective as real garlic for allicin and S-allylcysteine activity. Garlic poultices may cause burns in infants. Used as spice and to treat hyperlipidemia, hypertension, atherosclerosis, cancer, and infections, but sustained response has not been found. Mixed effects regarding reduction of blood glucose levels, blood pressure, or cardiovascular diseases. Garlic should not be used in patients on anticoagulants and patients with platelet dysfunction.
Ginger (6-gingerol)	May help to reduce the side effects of cancer treatments as an antiemetic, anti-inflammatory agent. Effective in preventing nausea and vomiting in some patients. It is a relatively safe herb, but patients taking blood thinners or about to undergo surgery should avoid ginger supplements. Interaction with many drugs including antacids, anticoagulants and antiplatelets, antidiabetics, antihypertensives, H_2 blockers, proton pump inhibitors (PPI), and barbiturates.	Ginger may be effective in treating chemotherapy-induced nausea and vomiting.
Ginkgo biloba (maidenhair tree)	Antioxidant and anti-inflammatory effects; role in cancer is being studied (see Table 13-2). Stimulates blood circulation and helps improve memory.	Ginkgo causes bleeding when combined with warfarin or aspirin (acetylsalicylic acid), raises blood pressure when combined with a thiazide diuretic, and causes coma when combined with trazodone. Lowers the threshold for seizures in seizure-prone individuals.
Ginseng, Asian (Panax ginseng)	The dried roots of the plants are used in some traditional medicines to treat a variety of conditions, including cancer. Rh2 is a ginsenoside extracted from ginseng that has effects on cell proliferation, induction of apoptosis, and stimulation of natural killer cells and other immune activity.	Asian ginseng may prevent some cancers. Proposed to give strength and stamina. Interactions with monoamine oxidase (MAO) inhibitors.
Ginseng, American (Panax quinquefolius)	A plant with similar (but not exactly the same) properties, is grown mainly in the United States.	Used for health maintenance, strength, stamina, and immunostimulation. Contraindicated in patients with hypertension and in premenopausal women.
Glucarate (calcium glucarate)	Proponents claim that glucarate may reduce the risk of colon, lung, liver, skin, prostate, and other cancers by increasing the body's ability to eliminate cancer-causing toxins that come from diet and the environment. May help the body remove excess estrogen and other hormones that promote these diseases.	Glucarate is found in many fruits and vegetables including apples, grapefruit, broccoli, Brussels sprouts, and bean sprouts. It also occurs naturally in the body in very small amounts.
Green tea (Camellia sinesis)	Contains polyphenols and may slow the delivery of nutrients to cancer cells by inhibiting the formation of new blood vessels (angiogenesis). Recent research has focused on green tea for the prevention of breast, prostate, skin, esophagus, stomach, colon, pancreas, lung, and bladder cancers.	The use of skin products that contain green tea may be somewhat protective against skin cancers. Epigallocatechin gallate (EGCG). EGCG may cause cancer cells to die and may stop new blood vessels from forming, thereby cutting off the supply of blood to cancer cells. Do not use with pregnant or lactating mothers. Use with caution with many drugs particularly anticoagulants.
Isatis root (Ban lan gen, Radix isatidis baphicacanthi, Isatis tinctoria, Isatis indigotica)	Used for common cold, sore throat, mumps, respiratory ailments, and malignant tumors. Leaves are used in one of the herbal formulas to treat prostate cancer. No adverse reactions known.	This herb is also used to treat chronic myelogenous leukemia. Studies also indicate that this plant has antiviral and immunostimulatory effects.
Licorice	Licorice root is an ingredient in many traditional Chinese herbal remedies. More research is needed to find out whether licorice extract has any role in cancer prevention or treatment.	May cause serious side effects, including hypertension or muscle weakness or paralysis.

(continued)

TABLE 13-8 Herbs, Dietary Supplements, and Cancer *(continued)*

Lipoic acid (alpha lipoic acid)	Lipoic acid plays an important role in metabolism. Recent research has shown that it is beneficial in treating nerve damage in diabetics. It may be helpful for other conditions as well. There is currently no evidence that lipoic acid prevents the development or spread of cancer.	Lipoic acid is an antioxidant found in certain foods including red meat, spinach, broccoli, potatoes, yams, carrots, beets, and yeast. It is also made in small amounts in the human body. Its possible role as a form of complementary therapy to reduce the side effects of radiation therapy or chemotherapy is still unclear.
Lyprinol (green-lipped mussel)	This is a fatty acid complex extracted from Perna canaliculus—a green-lipped mussel (shellfish) native to New Zealand. It contains omega-3 fatty acids. Lyprinol is promoted as a dietary supplement with anti-inflammatory properties to work against leukotrienes.	It is available in capsule form as a dietary supplement.
Macrobiotic diet	The standard macrobiotic diet today consists of 50–60% organically grown whole grains, 20–25% locally and organically grown fruits and vegetables, and 5–10% soups made with vegetables, seaweed, grains, beans, and miso (a fermented soy product). Early versions of the diet included no animal products at all.	Potatoes, tomatoes, eggplant, peppers, asparagus, spinach, beets, zucchini, and avocados are excluded. The diet also advises against eating bananas, pineapples, and other tropical fruits. The use of dairy products, eggs, coffee, sugar, stimulant and aromatic herbs, red meat, poultry, and processed foods is discouraged.
Melatonin	May aid in the effectiveness of chemotherapy and in improving survival in numerous types of cancer. Melatonin inhibits tumorigenesis with the suppression of tumor linoleic acid (LA) uptake and its metabolism. Melatonin may also stimulate natural killer cells, which attack tumors. Inhibits cachexia.	Circadian rhythm may be enhanced with use of melatonin; this may help to alleviate fatigue associated with cancer.
Milk thistle (Silymarin)	Antioxidant for treating liver diseases such as cirrhosis or chronic hepatitis. May help with cancer prevention; human studies are needed.	It contains flavonolignans; perhaps has a role in decreasing skin or prostate cancer.
Mistletoe (Iscador)	Lectin-rich mistletoe extract should be further evaluated.	There may be toxic side effects.
Mulberry	Anthocyanins in mulberry have an anticancer effect.	More research is needed.
Mustard seed (Brassica campestris)	Mustard seeds enhance the antioxidant defense system and provide protection against the toxic effects of carcinogens.	May protect against stomach and uterine cancers.
Oleandrin, odoroside (Nerium odorum)	Raw leaves are toxic. May cause apoptosis in various cancer cell lines.	Side effects include nausea, vomiting and diarrhea, tachycardia, and arrhythmia.
Noni juice (Morinda citrifolia)	An immunomodulatory polysaccharide-rich substance from the fruit juice is rich in potassium.	Common in Polynesian diets. High sugar content.
Pokeweed (Poke salad)	Pokeweed antiviral protein has anti-tumor effects in mice and laboratory studies. Clinical trials have not yet been done.	All parts of the mature plant contain chemically active substances such as phytolaccine, formic acid, tannin, and resin acid; all are mildly poisonous when eaten.
Probiotics	Evidence suggests the following beneficial effects: normalization of the intestinal microflora, the ability to block the invasion of potential pathogens in the gut, prevention of colon cancer, modulation of immune function, inhibition of H. pylori.	Regular use of yogurt and other natural functional foods may be useful for cancer patients. Daily intake of Bifidobacterium lactis enhances natural immune function.
Pycnogenol; pine bark extract (Pinus pinaster)	Pycnogenol is the name of a group of bioflavonoids with proanthocyanidins taken from a number of natural sources, such as grape seeds.	The maritime pine tree contains naturally occurring proanthocyanidins.
Quercetin	Quercetin is promoted to help prevent or treat different types of cancer.	See Table 13-2.
Reishi mushroom (Ganoderma lucidum)	The medicinal mushroom Reishi has been widely used to treat cancer, diabetes, and neurasthenia in many Asian countries. Used for fatigue, high cholesterol, HIV and AIDS, hypertension, immunostimulation, inflammation, strength and stamina, and viral infections.	Can interfere with immunosuppressants and chemotherapeutic drugs. Adverse reactions may include dry throat and nose, GI upset, itchiness, nausea, and vomiting.
Rosemary and marjoram (ursolic acid)	Terpenoids in these spices provide anticancer effects.	Triterpenoid compound that occurs naturally in a large variety of vegetarian foods, medicinal herbs, and plants.

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