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Vitamin C Deficiency

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Continuing Education Activity

Vitamin C deficiency, commonly called scurvy, is a well-documented nutritional disorder with historical significance that continues to impact global health today. Vitamin C is an essential nutrient for maintaining an individual's good health and well-being. Vitamin C has significant antioxidant properties that protect cells from free radical damage. In addition, vitamin C also nurtures the growth and repair of skin, cartilage, bone, and teeth. Vitamin C deficiency is often linked to low socioeconomic status, food insecurity, and limited access to nutritious foods. Classic signs of vitamin C deficiency include corkscrew hairs, perifollicular hemorrhages, and gingival bleeding. Scurvy is a rare and reversible condition that requires early diagnosis and treatment. This activity reviews the historical background, clinical manifestations, diagnosis, treatment, and preventive measures concerning vitamin C deficiency. This activity also highlights the critical role of the interprofessional team in providing care for affected patients and raising awareness about nutritional deficiencies.

Objectives:

- Identify the clinical signs and symptoms of vitamin C deficiency, such as corkscrew hairs, perifollicular hemorrhages, gingival bleeding, fatigue, and anorexia.
- Assess the effectiveness of treatment by regularly monitoring patients' clinical improvement and resolution of symptoms associated with vitamin C deficiency.
- Select suitable diagnostic tests, such as serum vitamin C levels or leukocyte vitamin C levels, to aid in accurately assessing vitamin C deficiency and treatment response.
- Collaborate with an interprofessional team, including dentists, dietitians, and pharmacists, to provide holistic care and optimize outcomes for patients with vitamin C deficiency.

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Introduction

Scurvy is a clinical syndrome resulting from vitamin C deficiency. Vitamin C is essential for the growth and repair of skin, cartilage, bone, and teeth. In addition, it has significant antioxidant properties that protect cells from free radical damage. This article defines the sources, metabolism, and functions of vitamin C, covering the diagnosis, physical manifestations, evaluation, treatment, prognosis, and prevention of vitamin C deficiency.

History of Vitamin C Deficiency

Ancient Egyptian medical practitioners first documented the symptoms of this condition in 1550 bc in the *Ebers Papyrus* and prescribed the treatment with onions and vegetables. Hippocrates coined *ileos emantis* for the disease and described it as follows: "The mouth feels unpleasant; gums are separated from the teeth; blood flows from the nostrils… ulcerations appear on the legs; skin becomes thin." During the 1700s, James Lind, a British Royal Navy surgeon, made the significant discovery that the consumption of lemons and oranges alleviated the symptoms of vitamin C deficiency. Tales from the pirates and British sailors era have made scurvy infamous across several countries. Scurvy also led to notable morbidity during the European potato famine,[1] polar expeditions, the United States Civil War, and the California gold rush.[2] In the 1920s, Albert Szent-Györgyi, a Hungarian biochemist, discovered the molecular structure of vitamin C and named it ascorbic acid, meaning anti-scurvy.[3][4][5][6][7]

Etiology

Exogenous vitamin C is necessary only for humans and other primates. Most mammals synthesize the vitamin from glucose, as it shares a close chemical resemblance. Humans lack the active form of the enzyme *L*-gulonolactone oxidase required for synthesizing ascorbic acid, making it essential to acquire vitamin C from dietary sources or supplements. The primary cause of vitamin C deficiency is due to inadequate nutritional intake.

Vitamin C is naturally present in various fruits and vegetables, such as citrus fruits, potatoes, spinach, broccoli, red peppers, strawberries, and tomatoes.[8] Approximately 90% of the vitamin C in typical diets comes from various sources of produce, including fresh, frozen, and canned options. As vitamin C is sensitive to heat, cooking foods at high temperatures can cause the vitamin to decompose. Boiling can also lead to its leaching into the water.[9] Fresh fruits and vegetables possess higher vitamin C content compared to frozen or canned foods. However, the latter can serve as a significant source of vitamin C when fresh produce is not accessible.

Vitamin C is water-soluble and gets absorbed in the distal ileum. The absorption of the vitamin is efficient at dosages up to 100 mg/d. However, when intake exceeds 1500 mg/d, the absorption of the vitamin decreases to 50% or less.[8] A small amount of vitamin C is found in leukocytes, adrenal glands, and the pituitary gland. Nevertheless, the vitamin C stores in the human body are limited due to its water-soluble nature, and any excess amount is typically excreted from the body. The human body has around 1500 mg of vitamin C in total, and clinical signs of deficiency are exhibited when the level drops below 350 mg. Symptoms of scurvy appear within 4 to 12 weeks of insufficient vitamin C intake. Plasma concentrations primarily indicate recent or current consumption rather than stores and may lack clinical utility.[10][11][12][13]

Risk Factors for Vitamin C Deficiency

Several factors can contribute to an increased risk of vitamin C deficiency, some of which are listed below.

Alcohol use disorder: Individuals with alcohol use disorder consuming more than 80 g/d of ethanol may experience increased renal excretion of vitamin C and poor dietary habits.

Infant feeding practices: Consumption of cow's milk instead of breast milk or fortified formula during infancy can pose a risk for vitamin C deficiency.

Social isolation and dietary habits: Social isolation and a limited diet, often called a "tea and toast" diet, particularly common among older individuals and institutionalized patients, can contribute to a lack of vitamin C intake.

Limited access to fruits and vegetables: Inability to obtain or afford a diverse range of fruits and vegetables can also elevate the risk of vitamin C deficiency.

Smoking: Smoking tobacco can diminish vitamin C levels in the body due to increased oxidative stress.

Eating disorders: Conditions such as anorexia nervosa and selective eating habits, including food faddism, can lead to inadequate vitamin C intake.

Type 1 diabetes: Individuals with type 1 diabetes may experience increased vitamin C requirements, potentially leading to deficiency.

Malabsorptive disorders: Conditions such as inflammatory bowel disease, celiac disease, and cystic fibrosis can hinder vitamin C absorption.

Bariatric surgery: Individuals who have undergone bariatric surgery may be at risk of vitamin C deficiency due to reduced absorption capacity.

Iron-overload conditions: Conditions involving excessive iron accumulation with renal losses of vitamin C can contribute to deficiency.

Restrictive or low carbohydrate diets: Diets significantly restricted in carbohydrates might inadvertently lead to reduced vitamin C intake.

Food allergies: Allergies to specific foods can limit dietary diversity and potentially impact vitamin C intake.

Developmental disabilities and mental illness: Conditions affecting food preferences, often seen in developmental disabilities and mental illness, can result in inadequate vitamin C consumption.

Hemodialysis: Individuals undergoing hemodialysis may experience increased renal losses of vitamin C, potentially leading to its deficiency.

In summary, a range of factors can significantly heighten the risk of vitamin C deficiency. Therefore, recognizing and addressing these risk factors to prevent and mitigate vitamin C deficiency are paramount for maintaining optimal health.[10][11][12][13]

Epidemiology

Vitamin C deficiency affects millions of individuals globally. The prevalence of this deficiency varies based on factors such as age, lifestyle, access to nutritious foods, dietary choices, and underlying medical conditions. Individuals who do not include fruits and vegetables in their diet face an elevated risk of vitamin C deficiency, although several high-risk populations have been identified. Scurvy is most commonly found in countries with endemic malnutrition, but its occurrence is global. Prevalence differs across regions, ranging from as low as 7.1% in the United States to as high as 73.9% in northern India.[14]

Pathophysiology

Scurvy arises due to inadequate vitamin C intake, which is critical in synthesizing collagen. Collagen is a vital structural protein essential for maintaining the integrity and strength of connective tissues throughout the body. Type IV collagen forms the primary building block of blood vessel walls, skin, and the basement membrane that separates the epidermis from the dermis. Collagen constitutes 75% of the dermis.[15] Vitamin C is a cofactor for proline and lysine hydroxylases, which stabilize collagen types I and VI. Furthermore, this vitamin enables crosslinking and facilitates procollagen transcription into collagen. A deficiency in vitamin C levels also results in epigenetic DNA hypermethylation, inhibiting the transcription of collagen in various tissues, including the skin and blood vessels. Insufficient collagen production in the body results in fragile skin and blood vessels, gingival hemorrhages, petechiae, and impaired wound healing. Vitamin C is a potent antioxidant that protects cellular constituents from oxidative stress. Vitamin C deficiency compromises immune function, rendering individuals more susceptible to infections and causing delays in wound healing.

Histopathology

Vitamin C deficiency exhibits distinctive histopathological features. Microscopic examination of affected tissues reveals evidence of compromised collagen synthesis. In the skin, there is evident dermal disruption and atrophy. The

number of collagen fibers is reduced, and they appear fragmented. The affected area displays signs of dilated hair follicles, fibrosis, follicular hyperkeratosis, corkscrew hairs, and perifollicular hemorrhages.[16][9] In the oral cavity, the gingival tissues exhibit hemorrhagic changes, which are characterized by capillary dilation and fibrin thrombi, subepithelial hemorrhages, and inflammatory infiltrates.[17] Within the skeletal system, the osteoid matrix is thin and disrupted, accompanied by subperiosteal hemorrhages and indications of osteopenia.[18]

History and Physical

Initially, nonspecific symptoms of scurvy emerge after 4 to 12 weeks of insufficient intake of vitamin C. Patients might present with fatigue, malaise, lethargy, and anorexia.[9] Subsequently, patients with scurvy experience oral and skin symptoms such as bleeding gums, easy bruising, skin rashes, fragility, delayed wound healing, and bone and joint aches.

Early oral and dermatologic signs that are visible in physical examination include gingivitis with bleeding, periodontal disease, loss of dentition, mucocutaneous petechiae, ecchymoses, hyperkeratosis, alopecia, corkscrew hairs, and swan neck hairs.[19] Corkscrew strands, characterized by twisted or coiled hair shafts, result from impaired collagen synthesis and are considered pathognomonic for scurvy. Swan neck hairs are hair shafts that bend at multiple points due to weakened hair follicles. Perifollicular hemorrhages are often localized to the lower extremities, as capillary fragility cannot withstand gravity-dependent hydrostatic pressure. This can lead to a condition known as "woody edema." Nail-related observations include koilonychia and splinter hemorrhages.

In addition to mucocutaneous manifestations, physical findings reflect the involvement of various other organ systems. Painful joint swelling, hemarthroses, and subperiosteal hematomas result from vascular fragility caused by impaired collagen formation.[18] Disrupted endochondral bone formation can result in fragile bones that are prone to fractures. Ocular symptoms include dry eyes, subconjunctival hemorrhages, and scleral icterus.

About 80% of children with scurvy present with musculoskeletal signs and symptoms, including arthralgias, myalgias, hemarthroses, muscle hemorrhages, and subperiosteal hematomas. Children exhibiting bone involvement might display a limp or resist bearing weight. The knee joint is frequently the most affected part of the body.[18] Another notable finding during a physical examination is the "scorbutic rosary," which are tender, angular, and sharp swellings at the costochondral junctions caused by weakened connective tissue. This differs from the "rachitic rosary" in rickets, where the prominences are rounder and non-tender.[20]

As scurvy progresses, its symptoms become increasingly severe and life-threatening. Advanced ocular manifestations include flame hemorrhages, cotton-wool spots, and retrobulbar bleeding into optic nerves, resulting in atrophy and papilledema.[21] The immune system becomes compromised and increases susceptibility to infections. If left untreated, severe scurvy can induce profound weakness, dyspnea, anasarca, hemolysis, jaundice, seizures, organ failure, and ultimately, death.

Evaluation

Scurvy is diagnosed clinically through relevant medical history, physical examination observations, and a rapid response to vitamin C supplementation.[21] The evaluation process commences with symptom assessment, dietary history collection, evaluation of risk factors for vitamin deficiencies, and a comprehensive physical examination. Dermoscopy can reveal findings such as follicular purpura and corkscrew hairs, while a 4-mm punch biopsy and histopathology of affected regions typically confirm these observations.

Serum testing for scurvy requires caution due to potentially misleading results from recent vitamin C intake or supplementation. A low plasma vitamin C level of less than 0.2 mg/dL may indicate scurvy, but chronic deficiency could be concealed.[22] The leukocytes provide a more precise measure of vitamin C levels than other methods due to

the resistance of white blood cells to dietary changes. A vitamin C level of 0 mg/dL in leukocytes indicates scurvy. A range of 0 to 7 mg/dL indicates a deficiency, whereas levels exceeding 15 mg/dL are considered sufficient.[23][24]

Patients diagnosed with scurvy might also exhibit inadequate intake of other crucial vitamins and minerals. Therefore, apart from evaluating vitamin C levels, it is also essential to conduct screening for other concomitant vitamin deficiencies. The affected patients often have low vitamin B12, folate, calcium, zinc, and iron levels. As vitamin C contributes to iron absorption, individuals with scurvy should be evaluated for anemia arising from insufficient absorption or blood loss resulting from bleeding.

Imaging studies may reveal the following:

- Fractures and dislocations
- Subperiosteal elevation
- Alveolar bone resorption, osteopenia, or osteoporosis
- Epiphyseal separation of the distal radius

Treatment / Management

The appropriate dosage of vitamin C for scurvy treatment varies based on the severity of the condition and the individual's particular requirements. Prompt vitamin C supplementation can quickly and efficiently replenish depleted vitamin levels in patients, with dosages up to 300 mg/d for children and 500 to 1000 mg/d for adults. The end point of replacement typically occurs within 1 to 3 months or upon the complete resolution of all clinical signs and symptoms. Alternative treatment regimens for adults include up to 2 g/d for the initial 3 days, 500 mg/d for 1 week, and 100 mg/d for 1 to 3 months.[9]

Treating scurvy involves consuming sufficient fruits and vegetables to prevent recurrences and addressing the underlying causes of malnutrition that lead to vitamin C deficiency. The recommended daily vitamin C intake varies based on age, sex, pregnancy, and lactation. The amount of vitamin C required for children is 15 to 75 mg, men 90 mg, women 75 mg, pregnant women 85 mg, and lactating women 120 mg. Smoking leads to a depletion of vitamin C levels, requiring smokers to consume an additional vitamin C of 35 mg/d.

Vitamin C is found in a wide range of foods. According to the United States National Institutes of Health, Office of Dietary Supplements, vitamin C can be easily obtained from the following sources:

- 1/2 cup red pepper: 95 mg
- 1 medium orange: 70 mg
- 1/2 cup strawberries: 49 mg
- 1/2 cup Brussels sprouts: 48 mg
- 1 baked potato: 17 mg

Supplements are readily available for individuals unwilling or unable to obtain adequate vitamin C solely from their diet, including standard over-the-counter multivitamin preparations.

Differential Diagnosis

The differential diagnosis encompasses a range of pathological conditions that vary based on the presenting signs and symptoms. Some possible considerations are listed below.

Purpura/ecchymoses: Immune thrombocytopenic purpura, Henoch-Schonlein purpura, disseminated intravascular coagulation, Rocky Mountain spotted fever, meningococcemia, and hypersensitivity vasculitis.

Oral and dental disease: Necrotizing gingivitis, periodontitis, candidiasis, blood dyscrasias, medication-induced gingival overgrowth, and glossitis/cheilitis arising from other nutritional deficiencies.

Rashes and skin changes: Mineral and other vitamin deficiencies.

Bone pain and limp: Osteomyelitis and septic arthritis.

Prognosis

Typically, symptoms of fatigue, body aches, and anorexia improve within 24 hours of treatment. Additional symptoms such as bruising, gingival bleeding, perifollicular hemorrhage, and weakness tend to respond within 1 to 2 weeks following treatment initiation. Corkscrew hairs usually return to their normal appearance within 1 month, and most symptoms tend to resolve completely within 3 months.[9] Surgical intervention might be necessary to manage severe and advanced bone abnormalities.

Complications

The complications associated with vitamin C treatment for scurvy are relatively infrequent, generally mild, and often associated with dosage.

Gastrointestinal disturbances: Elevated doses of vitamin C can lead to diarrhea, nausea, and abdominal cramps. These symptoms are usually self-limiting and resolve spontaneously when the dosage is adjusted or discontinued.

Renal calculi: Excessive vitamin C consumption has been linked to the development of kidney stones in male patients with a history of calculi or preexisting renal conditions. This complication is believed to be due to increased urinary oxalate excretion.[25] Maintaining proper hydration and moderating vitamin C intake can help mitigate the risk.

Interactions with medications: The antioxidant properties of vitamin C can reduce the effectiveness of chemotherapy. This property can also interfere with the effectiveness of drugs such as warfarin, statins, and niacin. Careful consideration and monitoring are essential when combining vitamin C supplementation with these medications.

Deterrence and Patient Education

Patients should be educated about the causes, symptoms, and potential consequences of vitamin C deficiency. They must understand that scurvy primarily develops due to inadequate consumption of vitamin C–rich fruits and vegetables. By providing clear and concise information, healthcare professionals can motivate their patients to make well-informed dietary decisions and integrate vitamin C–rich foods into their daily meals. Excellent sources of vitamin C–rich foods include citrus fruits, juices, berries, melons, red and green peppers, tomatoes, potatoes, and cruciferous vegetables. Individuals should receive counseling to refrain from smoking and, if necessary, limit their alcohol consumption.

In cases where scurvy stems from a malabsorptive condition, such as inflammatory bowel or celiac disease, patient education should encompass understanding the specific diagnosis to effectively address the underlying disorder and the resulting vitamin C deficiency. When social isolation or food insecurity contributes to inadequate intake of foods rich in vitamin C, it might be essential to consider social services or supplemental food programs to complement standard patient education initiatives. Individuals unable or unwilling to consume fruits and vegetables should be recommended to take a daily multivitamin supplement.

Enhancing Healthcare Team Outcomes

A multidisciplinary approach involving physicians, advanced care practitioners, nurses, dieticians, dentists, dental hygienists, and pharmacists is vital for providing patient-centered care, enhancing outcomes, and maintaining patient safety. Each healthcare team member should possess specialized skills in assessing, diagnosing, and managing vitamin C deficiency. Collaboratively, the team should develop comprehensive strategies for identifying patients at risk, implementing evidence-based interventions, and monitoring progress. This approach requires devising strategies for dietary changes and taking supplements.

Healthcare professionals should encourage adequate nutrition for their patients. Although vitamin C deficiency is uncommon in North America, it tends to develop among individuals who have inadequate consumption of vitamin C–rich foods or are affected by malabsorption or other chronic medical conditions. Patients should be educated about the significance of a nutritious diet abundant in fruits and vegetables. Furthermore, it is essential to motivate patients to abstain from smoking and limit alcohol consumption. Upon diagnosing a vitamin deficiency, it becomes imperative to conduct screenings for other potential concurrent deficiencies. As vitamin C deficiency is primarily related to inadequate dietary intake, affected individuals may also exhibit deficiencies in other essential nutrients such as vitamin B12, folate, calcium, zinc, and iron.

When patients show oral signs and symptoms, dentists may initially diagnose vitamin C deficiency. Collaboration between dentists and primary care physicians through care coordination can improve patient outcomes. In rare cases, where a patient shows limited improvement even after several weeks of treatment, seeking evaluation by a specialist might be necessary to determine the underlying cause of the vitamin C deficiency.

Effectively addressing vitamin C deficiency demands the coordinated efforts of an interprofessional team. Healthcare professionals can collectively enhance patient outcomes, safety, and overall team performance by harnessing their skills, adopting a patient-centered approach, delineating clear responsibilities, fostering open communication, and ensuring streamlined care coordination.

Review Questions

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