Scurvy in 2017 in the USA

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ABSTRACT

Vitamin C deficiency may be common in third-world countries due to malnutrition, but it is currently rare in the USA. Initially, nonspecific symptoms like exhaustion and depression may make this disease difficult to diagnose until classical dermatological manifestations appear. Diagnosis mainly relies on clinical presentation, dietary history to identify risk factors, and dramatic recession of symptoms and signs following vitamin C therapy. Human beings cannot synthesize vitamin C and hence need 90% of vitamin C intake from fruits and vegetables. As a processed carbohydrate-rich diet becomes the staple food, scurvy must be recognized before it becomes potentially fatal. We describe a 65-year-old man with dyspnea, fatigue, anemia, and bleeding diathesis from scurvy and emphasize the importance of dietary history and the critical role of vitamin C in diagnosis and management of this forgotten entity.

KEYWORDS Bleeding diathesis; collagen defects; scurvy; vitamin C

Scurvy, a disease caused by a deficiency of vitamin C, is uncommon in the modern era and is often underdiagnosed. Vitamin C serves as a cofactor in collagen synthesis, and its deficiency is characterized by hemorrhagic diathesis. We describe a case of scurvy presenting with anemia, perifollicular hemorrhages, and extensive ecchymoses and highlight the importance of recognizing vitamin C deficiency as a cause of bleeding diathesis.

CASE REPORT

A 65-year-old man with known anxiety–depression and emphysema (smoker for 30 years) presented with dyspnea and dizziness. He lived alone and his diet consisted of carbohydrates and lacked fresh fruits and vegetables. Current medications included only duloxetine and bronchodilators. Physical examination disclosed perifollicular hemorrhages and extensive ecchymoses with increased pigmentation involving the lower extremities (*Figure 1*). Duloxetine was discontinued; nevertheless, he developed a right thigh hematoma. Laboratory evaluation is summarized in *Table 1*. Vitamin C levels were $<5 \mu\text{mol/L}$ (normal $23\text{--}114 \mu\text{mol/L}$). Vitamin C supplements were administered with dramatic dissipation of ecchymosis and improvement of anemia (*Figure 2*).

DISCUSSION

Scurvy, known since the Egyptian era, is rarely seen in the modern world due to the discovery of its link to the dietary

deficiency of vitamin C. Scurvy was first described in sea voyagers by the Elbers papyrus in 1550 BC. Humans cannot synthesize vitamin C and hence require an exogenous source.¹ Following ingestion, ascorbic acid (AA) is absorbed in the small intestine, and excess amounts are excreted in urine. Although vitamin C is readily stored in tissues throughout the body, these stores are quickly exhausted. Serum AA is three times lower in smokers than in nonsmokers, significantly increasing their chances of AA deficiency.² Smokers possess a 40% increase in daily vitamin C turnover, increasing the potential for vitamin C deficiency in smokers who consume $<200 \text{ mg/d}$ of vitamin C. Other risk factors include living alone (particularly for men), advanced age, long-term alcoholism, and a voluntarily restrictive diet lacking vegetables and fruits.³

The critical role of vitamin C in humans is collagen synthesis. Collagen is an essential element in skin and blood vessels.⁴ Vitamin C helps to hydroxylate excess proline and lysine in procollagen, stabilizing the triple helix. Malfunction in collagen synthesis is the primary cause of clinical manifestations of vitamin C deficiency.⁵ Thus, it eventually leads to skin lesions and ruptured blood vessels. Vitamin C levels in the body become undetectable 41 days following dietary lack of vitamin C. Cell depletion commences after 121 days, and dermal lesions appear at 132 days following dietary vitamin C deprivation.

Early manifestations include nonspecific fatigue, lassitude, and depression and hence diagnosis is often delayed, as

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Figure 1. Right arrow showing perifollicular hemorrhages and left arrow showing extensive ecchymosis.

Figure 2. (a) Extensive ecchymosis of the lower extremity prior to the treatment. (b) Resolution of ecchymosis after vitamin C treatment.

occurred in our patient, until cutaneous manifestations occur. Dermatological manifestations are the hallmark of scurvy and range from paleness, bloated complexion, dryness, and follicular hyperkeratosis to hemorrhagic skin lesions and spontaneous hematomas.⁶ Oral findings include gingivitis, periodontitis, and mucosal hemorrhages. Ocular manifestations like conjunctival hemorrhage and flame-shaped hemorrhages of the fundus may also be evident. Late presentations include bone pain, leg edema, dyspnea, anxiety, anorexia, pulmonary hypertension, leukopenia, syncope, hypotension, and eventually death unless the vitamin C deficiency is corrected in

Table 1. Laboratory findings

Lab test	Patient's value	Normal values
Hemoglobin (g/dL)	8.3	13.5–17.5
Platelet count ($10^9/L$)	165,000	150–450,000
Reticulocyte count	4.5%	0.4%–2.4%
Prothrombin time (seconds)	13	11–14
Partial thromboplastin time (seconds)	30	20–39
Alpha 2-antiplasmin levels	153%	85%–156%
Euglobulin lysis time (minutes)	>60	90–240
Platelet function analysis		
Collagen ADP (seconds)	81	55–137
Collagen epinephrine (seconds)	123	78–199

a timely manner. Diagnosis is usually made by the impressive clinical findings discussed above and the dramatic response to vitamin C therapy; nevertheless, measurement of ascorbic acid levels aids the clinical diagnosis. Repleting the body stores and correcting the critical deficiency of AA remain the goals of therapy. AA at a dose of 300 mg for 1 month cures most of the symptoms and signs within 4 weeks, with complete recovery expected in 3 months.⁷

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