CASE REPORT

Adult Scurvy Presenting with Painful Purpura on the Legs

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Abstract:

Prolonged vitamin C deﬁciency can result in numerous metabolic abnormalities like impaired tissue repair and defective collagen synthesis. This case report describes a middle-age Japanese man presenting painful purpura on his lower limbs, severe anemia, and altered consciousness. The patient had been eating a selective diet lacking in vegetables and fruits since childhood. A serum analysis demonstrated a low level of vitamin C. The patient was treated with vitamin supplementation and psychological intervention. Scurvy is an under-considered illness with a favorable prognosis if diagnosed early while it is still sporadically encountered in some patients with malabsorption or malnutrition even in modern times.

Key words: vitamin C, malnutrition, malabsorption, peliosis, anemia

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Introduction

Scurvy, caused by a prolonged inadequate intake of vitamin C, is a potentially lethal condition. The disease may also occur in developed countries, typically among poor elderly patients and refugees (1). It is also recognized as an occupational disease among workers such as whalers or among armies with inadequate access to fresh fruits and vegetables (2, 3). The prevalence of scurvy has decreased markedly over the years as the availability of multiple enriched and fortified food products became widespread. However, scurvy still occurs in both children and adults with abnormal dietary habits, mental illness, alcoholism, concomitant gastrointestinal disease, or physical disabilities (4-7).

We herein report an adult patient with scurvy presenting with painful purpura on his legs and gluteal region and muscle weakness. Based on detailed inquiries regarding his eating habits and serum measurement of vitamins, the patient was diagnosed early and successfully treated with vitamins and a balanced diet. The signs of scurvy greatly vary and include systemic, skin, bone, and dental manifestations. In addition, the low frequency of this disease may result in difﬁculty accurately diagnosing and treating the condition. Our case may highlight the importance of recognizing scurvy as a differential diagnosis when there are atypical presentations of purpura that cannot be explained by another disease such as vasculitis or trauma. Sharing our experience as well as including a review of the pertinent literature may help emergency physicians expedite the treatment process and improve patient prognosis.

Case Report

A 48-year-old male was admitted to the hospital with complaints of impaired consciousness, general fatigue, and leg pain. He had no noteworthy past medical history and had been living a life of seclusion. The patient had cramps in his left dorsal foot and gingival bleeding two weeks before presentation. He denied any physical trauma, insect bites, or general diseases and reported a history of eating a selective diet lacking in vegetables or fruits since childhood. His daily diet had consisted of rice with soy sauce and mayonnaise for the past month and before that, he ate exclusively instant noodles. The patient experienced occasional nasal and gingival bleeding.

His vital signs and physical examination results at presentation were as follows: body temperature of 36.7°C, height...
of 175 cm, body weight of 56.3 kg, body mass index of 23.3 kg/m², heart rate of 103 beats/min, blood pressure of 103/70 mmHg, and respiratory rate of 28 breaths/min. His Glasgow Coma Scale score was 15 (E4V5M6). Prior to arrival at the hospital, he had also experienced urinary incontinence. An examination of the skin showed symmetrically distributed purpuric petechiae with edema and tenderness in the right lower extremity (B) shows flat, non-blanchable purpura/echymoses ranging from purple to yellow around the calf and ankle.

Figure. Purpuric petechiae with edema and tenderness in the gluteal region and posterior thigh were noted (A). Image of right lower extremity (B) shows flat, non-blanchable purpura/echymoses ranging from purple to yellow around the calf and ankle.
Vitamin C is absorbed from the gastrointestinal tract via active transport and passive diffusion. The total pool of vitamin C in the body, mainly found in the liver and muscles, is 1,500-2,000 mg. The daily turnover of vitamin C is 45-60 mg, with a half-life of 10-20 days (4). Ignorance about proper nutrition, malabsorption syndromes, psychiatric disorders, alcoholism, drug abuse, pregnancy/breast feeding, and social isolation are all causes that can lead to the development of scurvy. Symptoms and signs typically develop after three months of insufficient vitamin consumption. Vitamin C, a strong reducing agent and enzyme cofactor, is essential in various biochemical pathways like antioxidant reactions and collagen synthesis. Abnormalities in collagen synthesis cause many of the symptoms of scurvy, including collagen fragility, which in turn makes blood vessels fragile and leads to poor wound healing (7, 8).

Vitamin C also promotes carnitine biosynthesis, which is necessary for biosynthesis of adenosine triphosphate in the muscle mitochondria. Therefore, vitamin C deficiency can cause muscular weakness. In addition, vitamin C deficiency results in behavioral alterations, mental disorders, and neuropathological disruption, which may be associated with altered noradrenergic and dopaminergic signaling (9). Thus, scurvy’s early clinical signs such as low grade fever, loss of appetite, and irritability, and dermatological symptoms such as ecchymoses, petechiae, corkscrew hairs, and hyperkeratosis, are not specific (4). Skin purpura without a history of trauma is a distinguishing feature of scurvy (10). Patients with scurvy are often underdiagnosed and they can also develop potentially fatal diseases including cerebral hemorrhaging, severe hemodynamic compromise, and systemic inflammatory response.

Scurvy’s differential diagnoses vary widely and include systemic conditions like hematological and rheumatological diseases and localized disorders like periodontal disease. In addition to physical and laboratory examinations, detailed information regarding the patient’s lifestyle, occupation, eating habits, food preferences, mental status, and bowel movements help in making an accurate diagnosis of scurvy. Of note, other diseases presenting similar manifestations must be ruled out. Our patient reported that he was a picky eater and a recluse, and lived in a self-imposed isolated room in his house. Under such circumstances, the patient’s daily diet may be unbalanced without essential nutrients. As with our patient, scurvy should be suspected in those with behavioral and psychiatric disorders (11). Children with physical disabilities, mental illness, or abnormal dietary habits are prone to developing this condition.

As vitamin C replacement is the only effective therapy for scurvy, early diagnosis and treatment is critical. Even patients with advanced disease usually favorably respond to administration of vitamin C. The coexistence of other biochemical complementary deficiencies of other vitamins and zinc should be considered and are required to be supplemented. Moreover, conditions that can affect compliance with treatment like gastritis, gastro-esophageal reflux, and bowel disease causing malabsorption should also be considered.

The standard treatment for scurvy is a daily oral dose of 1,000 mg of vitamin C for two weeks (12). When administered orally, vitamin C is absorbed well at low doses, but absorption decreases with increasing doses. The median bioavailability after oral administration is 87% at 30 mg, 80% at 100 mg, 72% at 200 mg, and 63% at 500 mg. At 1,250 mg, less than 50% of the dose absorbed and the remainder is excreted in the urine (8). Therefore, the doses should be divided and given throughout the day to obtain adequate absorption (4, 12, 13). Issuing nutritional guidance to the patient is necessary to prevent scurvy from reappearing after discharge from the hospital. Foods rich in vitamin C are often also rich in folic acid. Our patient required both

### Table. Laboratory Data on Admission and Two Months Later.

| Test                                      | On admission | Two months later |
|-------------------------------------------|--------------|------------------|
| White blood cells (×10⁶/µL)              | 4,800        | 5,400            |
| Red blood cells (×10¹²/µL)               | 1.33         | 4.30             |
| Hemoglobin (g/dL)                        | 5.0          | 13.8             |
| Hematocrit (%)                           | 13.7         | 38.5             |
| MCV (fL)                                 | 103.0        | 89.5             |
| MCH (pg)                                 | 37.6         | 32.1             |
| MCHC (g/dL)                              | 36.5         | 35.8             |
| Reticulocytes (%)                        | 5.3          | 1.0              |
| Platelet cells (×10⁹/µL)                 | 18.6         | 11.4             |
| Albumin (g/dL)                           | 2.8          | 3.9              |
| AST (IU/L)                               | 14           | 13               |
| ALT (IU/L)                               | 5            | 13               |
| Total-bilirubin (mg/dL)                  | 1.8          | 0.6              |
| Creatinine (mg/dL)                       | 1.01         | 0.73             |
| Urea nitrogen (mg/dL)                    | 20.2         | 11.1             |
| Sodium (mmol/L)                          | 134          | 142              |
| Potassium (mmol/L)                       | 3.3          | 3.5              |
| Chloride (mmol/L)                        | 104          | 106              |
| Iron (µg/dL)                             | 141          | 105              |
| Ferritin (ng/mL)                         | 67           | 285              |
| C-reactive protein (mg/dL)               | 1.61         | 0.11             |
| PT (s)                                   | 14.2         | 11.5             |
| APTT (s)                                 | 29.3         | 33.6             |
| PT-INR                                   | 1.2          | 1.0              |
| Vitamin C (µg/mL)                        | <0.2         | 8.8              |
| Vitamin B12 (pg/mL)                      | 80           | 404              |
| Folic acid (ng/mL)                       | 1.9          | 11.9             |

MCV: mean corpuscular volume, MCH: mean corpuscular hemoglobin, MCHC: mean corpuscular hemoglobin concentration, AST: aspartate transaminase, ALT: alanine aminotransferase, PT: prothrombin time, APTT: activated partial thromboplastin time, PT-INR: international normalized ratio of prothrombin time

25. During the two-month close follow-up period, the patient remained clinically stable without any laboratory abnormalities (Table).

**Discussion**

Vitamin C is absorbed from the gastrointestinal tract via active transport and passive diffusion. The total pool of vitamin C in the body, mainly found in the liver and muscles, is 1,500-2,000 mg. The daily turnover of vitamin C is 45-60 mg, with a half-life of 10-20 days (4). Ignorance about proper nutrition, malabsorption syndromes, psychiatric disorders, alcoholism, drug abuse, pregnancy/breast feeding, and social isolation are all causes that can lead to the development of scurvy. Symptoms and signs typically develop after three months of insufficient vitamin consumption. Vitamin C, a strong reducing agent and enzyme cofactor, is essential in various biochemical pathways like antioxidant reactions and collagen synthesis. Abnormalities in collagen synthesis cause many of the symptoms of scurvy, including collagen fragility, which in turn makes blood vessels fragile and leads to poor wound healing (7, 8).

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nutritional guidance and psychological treatment.

**Conclusion**

Vitamin C deficiency, which is often under-suspected, should therefore be considered in the differential diagnosis when examining patients with skin purpura without a history of trauma.

The authors state that they have no Conflict of Interest (COI).

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