CASE REPORTS/CLINICAL VIGNETTES

Scurvy Is Still Present in Developed Countries

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INTRODUCTION: Scurvy, while uncommon, still occurs in developed countries despite the widespread availability of vitamins and fortified foods. A vitamin C deficiency prevalence of 10 to 14% in adults was reported in the National Health and Nutrition Examination Survey (NHANES) in 1994.

OBJECTIVE: We report the case of a 57-year-old male who presented with a combined vitamin C deficiency (scurvy) and a Zinc deficiency.

INTERVENTIONS: He came to the emergency department complaining of a painful swollen ecchymotic leg and dyspnea. Prescriptions for narcotics did not relieve his symptoms. When a detailed dietary history was obtained, we added scurvy to the differential diagnosis. An extensive evaluation excluded trauma, coagulopathies, neoplasia, and vasculitides.

MAIN RESULT: The combination of a classic skin biopsy and a low vitamin C level confirmed the diagnosis.

CONCLUSION: This presentation illustrates the necessity of including scurvy in the differential diagnosis of ecchymoses and demonstrates specific populations at risk: single adults and the elderly with deficient diets.

KEY WORDS: scurvy; 57-year-old male patient; vitamin C; developed country; ecchymosis.
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INTRODUCTION

Most physicians consider scurvy a historical disease associated with starving populations deprived of vitamin C intake. In the 15th to 18th centuries, sailors commonly developed scurvy when only non-perishable food could be stored in ships sailing overseas.¹ Scurvy occurred when people had little access to foods containing vitamin C for extended periods, e.g., the great Potato Famine in Ireland in 1845, the American Civil War, the exploration of the North Pole, and the California Gold Rush.^{2,3,4} The last major documented outbreak occurred in Afghanistan in 2002 after civil war and a severe drought.⁵ Scurvy presently

occurs in developed countries among poor elderly patients, those institutionalized with diets devoid of fresh fruits and vegetables, and those with a history of alcoholism, mental illness, or chronic illness.^{6,7} Scurvy still occurs despite widespread availability of multiple enriched and fortified food products.

This case reports an older man who complained of a swollen ecchymotic leg, petechiae, and dyspnea. His dietary history suggested nutrient deficiencies. An extensive investigation excluded common causes of bleeding: trauma, coagulopathies, vasculitides, and malignancy. A skin biopsy and a low vitamin C level confirmed the diagnosis of scurvy. This case reminds physicians to consider the possibility of scurvy and other vitamin and mineral deficiencies. This patient's history reinforces the importance of obtaining a complete dietary history that could reveal nutritional deficiencies.^{7,8,9,10}

CASE REPORT

A 57-year-old white male presented to the emergency department complaining of a painful, swollen left thigh ecchymosis, which had spread to the ankle over the past 2 weeks. He had 2 previous emergency department visits with similar complaints, being discharged with narcotic pain medications only. The patient presented with worsening symptoms and new onset shortness of breath. The patient denied hematemesis, hematochezia, hematuria, bleeding gums, and trauma as well as any chest pain, cough, or fever. History revealed a reduced appetite and weight loss of 20 lbs over the last 2 months. His past medical history was negative. His only current medication was an opioid analgesic. Socially, he lived alone and was divorced. He smoked 2 packs of cigarettes a day and smoked marijuana episodically. He had a history of previous alcohol abuse but stopped drinking 6 years ago. He was sexually inactive. Family history was positive for diabetes mellitus. His dietary history revealed that he ate mostly TV dinners, canned soups, and canned stewed vegetables, with almost no fresh fruits or vegetables.

On physical examination, the vital signs showed a temperature of 96.5 F, heart rate of 77 beats/min, and a respiratory rate of 24/min, a blood pressure of 104/65 mmHg, and an oxygen saturation of 100% on room air. His height was 6 feet, and weight was 150 lbs, with a body mass index of 20.3 kg/m². He appeared older than his stated age, malnourished, and in mild distress, with a flat affect and irritability. He had male pattern baldness. He was toothless without evidence of gingivitis. Pulmonary and cardiovascular exam were within normal limits. Skin exam revealed a purple hematoma on the left thigh (Fig. 1) partly involving the entire circumference with some spreading to the left calf and ankle, but the patient did not have any palpable purpura. The patient also had non-

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Figure 1. Hematoma in the left thigh and perifollicular petechiae.

blanching non-palpable petechiae in a diffuse perifollicular distribution (Fig. 2) on the upper and lower extremities including the dorsal feet. There were bruises of various sizes on the extensor surfaces of the hands, wrists, and forearms. Remainder of physical examination was unremarkable.

Laboratory data showed a normal white cell count. The hemoglobin was 9.7 g/dL and hematocrit was 29.8% (previous value of 13.0 g/dL and 38.8% 2 weeks before), platelet of $190 \times 10^3/\text{mL}$, mean cell volume (MCV) was 87 fl. A reticulocyte count was 3.5%. A comprehensive metabolic panel was within normal limit as were prothrombin and partial thromboplastin time. D-dimer level was 0.92 mg/L (normal <0.5 mg/L). Cardiac enzymes, electrocardiogram (EKG), and chest x-ray were within normal limits. Echocardiogram showed preserved left ventricular ejection fraction without valvular abnormalities or pericardial effusion. A computed tomography (CT) angiograph of the chest showed bullous emphysema but no pulmonary embolism. CT of abdomen and pelvis were negative. A Doppler of the left lower extremity venous showed a normal deep venous system. Left femoral, tibia, and fibula x-rays were negative for fracture. Large bony infarcts were described in both the proximal and distal tibia and a sclerotic lesion involving the proximal tibia was noted.

Hepatitis A, B, and C antibodies were all negative. HIV was non-reactive. A urine drug screen was positive for marijuana. An alcohol level was undetectable. An erythrocyte sedimentation rate was mildly elevated at 37 mm/h. Antineutrophil cytoplasm antibodies (c-ANCA), antineutrophil perinuclear antibodies (p-ANCA), and antinuclear antibodies (ANA) were all negative. Serum protein electrophoresis (SPEP) was negative. Ristocetin cofactor and Von Willebrand Ag were normal. A platelet function analysis revealed a normal collagen epinephrine (Epi) and negative cell-bound platelet autoantibody. The vitamin C level was $<0.1 \mu\text{mol/L}$ (RR 0.6–2.0) and the zinc level was 0.62 mcg/mL (RR 0.66–1.10).

Vitamin C and zinc were below the expected normal range. We had performed a skin biopsy near one of the perifollicular petechiae before getting the vitamin C level back. It showed classic perifollicular hemorrhage, irregularly shaped hair follicles with hyperkeratosis, and coiled irregular hair shafts. Based on his vitamin C level, dietary history, clinical picture, and skin biopsy, we made a diagnosis of scurvy.

We treated the patient with vitamin C 1 gm orally once a day and encouraged a diet rich in vitamin C. He showed clinical and hematological improvement within 4 days time and was continued on the same dose at discharge as well as encouraged to start a daily multivitamin. At a follow-up phone call at 2 weeks, he reported a complete recovery of his skin lesions and dyspnea as well as continued compliance with vitamin C intake.

DISCUSSION

Scurvy is a disease rarely seen in developed countries where fortified food products and multiple supplements fill the market. However, poor diets devoid of fresh fruits and vegetables and low food variety can still cause this often forgotten disease.

Our patient's initial diagnostic work-up considered conditions that are more common than scurvy, such as trauma, coagulopathies, vasculitides, and malignancy. We excluded leg trauma and physical abuse by history and a negative x-ray.

Subsequently deep vein thrombosis was ruled out with a normal vein duplex. An extensive work-up was done for dyspnea. Cardiac illness was excluded with a normal EKG, negative cardiac enzymes, and a normal chest x-ray and echocardiogram. Pulmonary embolism was in the differential with an increased D-dimer; the negative Doppler and CT angiograph of the chest excluded it. The history of tobacco use and incidental finding of bullous emphysema on CT, as well as a new-onset anemia, were considered contributory factors of dyspnea. Thrombotic thrombocytopenic purpura, idiopathic thrombocytopenic purpura, and hemolytic uremic syndrome were excluded by a normal platelet count, urinalysis, and basic metabolic panel. We also considered other important possibilities such as abnormal qualitative platelet function; drug hypersensitivity induced purpura, primary vasculitis, secondary vasculitis,²¹ Von Willebrand disease, vitamin K deficiency, and neoplasia. A similar clinical picture has been observed in patients with these different conditions.

Our patient's Epi collagen test result was negative, as was his platelet antibodies, which ruled out purpura induced by drug hypersensitivity or neoplasia. A C-ANCA and P-ANCA came back negative, a urinalysis was normal, and a CT scan of

Figure 2. Microscopic picture showing perifollicular hemorrhage, extravasation of erythrocytes.

the chest showed only bullous emphysema, ruling out primary vasculitis, such as Wegener's granulomatosis and polyarteritis nodosa. In a similar way, we excluded secondary vasculitis induced by infectious diseases such as hepatitis C or HIV.

We considered vitamin K deficiency secondary to liver disease as our patient had a previous history of alcohol abuse. However, he had normal liver function tests and a normal coagulant profile (prothrombin time and partial thromboplastin time). We also considered the different types of Von Willebrand disease, but his Von Willebrand Ag was normal and his ristocetin cofactor ruled out a qualitative abnormal VIII factor.

Neoplastic disease can also present in a similar way. We considered diseases like leukemias, lymphomas, multiple myeloma, myelodysplasia, and disseminated malignancy. Our patient had a normal white blood cell count, normal peripheral smear, a normal protein electrophoresis, and a CT of the chest; abdomen, and pelvis were negative for malignancy.

As we could not find any common cause for his petechiae and hematoma, we reconsidered his presentation. We obtained a detailed dietary history in our patient that revealed poor dietary habits with lack of fresh fruit and vegetables, based on canned or overcooked food. He did eat what sounded like a daily intake of meat, so we did not check a prealbumin. His poor intake of vitamin C containing foods led us to consider scurvy in our differential diagnosis and consider less common diseases like senile purpura secondary to zinc deficiency.

Zinc deficiency can contribute to senile purpura, acrodermatitis enteropathica, cognitive decline, depression and alopecia.^{11,16,20} Our patient had purpura, male pattern baldness, and was initially irritable with a flat affect. We ordered a zinc level that came back low (0.62 mcg/mL) but not low enough to be significant. Alkaline phosphatase level was 62 IU, in the lower range of normal. It is interesting to note that after several months of treatment with multivitamins, his alkaline phosphatase went up to 92 u/L. Serum alkaline phosphatase is low in severe zinc deficiency and returns to normal after treatment.^{22,23} We did not order zinc levels in neutrophils and lymphocytes.

Our patient's vitamin C was very low and his skin biopsy showed classic perifollicular hemorrhages that confirmed the diagnosis of scurvy.¹³ He also had the classic corkscrew hairs noted on biopsy. The biopsy excluded the possibility of leukocytoclastic vasculitis (small vessel vasculitis) triggered by Hepatitis C, cryoglobulinemias, or neoplasias.

Vitamin C participates in the transformation of lysine proline on hydroxylproline that is part of collagen synthesis. Patients with deficient Vitamin C produce an abnormal collagen that affects blood vessel integrity, leading to capillary fragility, perivascular edema, and red cell extravasations.^{7,14} Classical signs of vitamin C deficiency include weakness, anemia, tooth loss, gum bleeding, bruises, and petechiae.

Our patient had a significant anemia, which was not present at his earlier visits to the emergency department 2 weeks back. Reticulocyte count was mildly increased at 3.5%. This, along with a normal MCV, essentially ruled out any severe deficiency of nutrients required for the development of red blood cells, such as iron or vitamin B¹². Serum lactate dehydrogenase (LDH) and haptoglobin levels were normal and the patient's anemia improved with vitamin C supplementation. Anemia is a frequent sign of scurvy secondary to blood loss and decreased iron absorption in the small bowel.

Decrease of the osteoid matrix formation and bone mineralization has been reported in children with scurvy and noted in our patient as evidenced by several bony infarcts and sclerotic lesions described on the tibia x-ray.²³ Hemorrhagic manifestations and poor wound healing are evident symptoms after 3 to 6 months of decreased dietary intakes of vitamin C.⁹ Humans are unable to synthesize vitamin C and thus depend on external sources to fulfill these requirements.⁸ The usual sources of vitamin C include raw fresh fruits and vegetables. Boiling, cooking, and canning fruits and vegetables lower the vitamin C content by 33%.^{25,26}

Many adults consume insufficient vitamin C. Eighty-five percent of the institutionalized elderly population is undernourished and 50% have vitamin and mineral intake that is less than the recommended dietary allowance.^{7,8} The most common population at risk for scurvy is an adult or older person living alone, edentulous or toothless, with poor self-care, lack of social support, having destructive behaviors such as alcoholism, drug abuse or smoking, and those adults who are mentally or chronically ill. They share a common denominator, a lack of food variety with chronic deprivation of vitamin C and other nutrients. Smokers require higher doses of vitamin C (110–125 mg/day) compared with normal population's requirement of 60–90 mg/day because of increased demand as a result of increased oxidative stress.⁶ Some researchers feel that the elderly require more vitamin C for the same reason.^{6,17,18,24}

The micronutrient and macronutrient deficiencies in the geriatric population cause new cases of scurvy, pellagra, and Zn deficiency.^{8,14,16}

This patient showed classical symptoms of bleeding caused by vitamin C deficiency: petechiae, hematomas, weakness, anemia, as well as radiologic bone changes, giving a typical picture of scurvy.

We generally consider scurvy an uncommon disease in developed nations, but it is still present. As scurvy presents with clinical features similar to many other common conditions, we must remember it and consider it as part of an expanded differential diagnosis. We strongly recommend having a high index of suspicion for this disease in the specific populations at risk, described as the socially isolated adult or elderly patient with a history of destructive behavior, living alone, and mentally or chronically ill.

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