Scurvy: An Unusual Cause of Anemia

Scott A. Cohen, MD, and Robert J. Paeglow, MD

Nutritional deficiencies are a common cause of anemia. We describe a case of a schizophrenic patient with a rapidly declining hematocrit. The patient was a long-term inpatient at a psychiatric hospital. He was brought to the emergency department with acute anemia initially thought to be secondary to gastrointestinal bleeding. Other causes for his anemia were investigated when gastrointestinal bleeding was ruled out.

A dietary history showed that the patient's diet consisted entirely of bread, cheese, and water. The patient was transfused and given oral vitamin C. Within 48 hours his hematocrit increased from 15.8% to 28%. The patient was transferred back to the psychiatric hospital, where he continued to improve on oral vitamin C. At a follow-up visit after discharge, he had a hematocrit of 41% and no signs of additional bleeding.

The diagnosis of scurvy can often be overlooked because it is rarely encountered in present-day society. A nutritional history is important, especially in the elderly, institutionalized, eating-disordered, or psychiatric patient.

Case Report

A 40-year-old man with schizophrenia was brought to the emergency department by his caretakers, who reported a 2-week history of progressive fatigue, leg and knee pain, apparent lightheadedness, and generalized weakness. The patient's schizophrenia and delusional thinking compromised his ability to provide useful historical information. On the day of admission the patient was noted to be "refusing" to walk. The caretakers also commented on the patient's apparent decline in function during the 2 weeks before admission, noting some "bloody stools," shortness of breath, knee swelling and "easy bruising."

His medical history was remarkable for schizophrenia and anxiety. He had been hospitalized 6 months earlier with suspected dehydration. His surgical history was notable for an inguinal hernia repair and appendectomy. His medications included fluphenazine, resperidone, and fluoxetine. No allergies were reported. His substance abuse history was negative for tobacco, drugs, and alcohol. On a review of systems there were no blood or bleeding disorders, fevers, or gastrointestinal symptoms other than the bloody stools. When examined, the patient was a very thin man lying in bed with severe pallor and in mild diaphoresis. His blood pressure was 110/72 mmHg, pulse was 94 beats per minute and regular, and respiratory rate was 18/min, and he was afebrile. Findings from a head, ears, eyes, nose, and throat examination were remarkable for poor dentition, pale conjunctiva, and mild gingival erythema. Findings from a heart, lung, and abdominal examination were unremarkable. His lower extremities had large bilateral knee effusions and pitting edema (2+) to above the knees. The skin examination showed large, confluent, nonpalpable, ecchymotic areas extending posteriorly from the inferior aspect of the buttocks to below the popliteal fossae. Ecchymotic areas were also found in the right antecubital fossa. An initial rectal examination reported by the emergency department was positive for occult blood; however, subsequent rectal examinations were negative.

Laboratory studies performed at the time of admission disclosed the following values: hemoglobin 7.7 g/dL and hematocrit 22.2%, with a mean corpuscular volume of 74 μm^3 (Table 1). Results from laboratory reports 3 months and 6 months before admission were hemoglobin 10.9 g/dL and 14.6 g/dL, respectively, and hematocrit 32.3% and 41.2%, respectively. Prothrombin time, activated partial thromboplastin time, Ivy bleeding time, and platelet count were normal. The patient was admitted, and he initially refused a transfusion. Intravenous hydration was begun, along with a workup to determine the cause of his anemia. Six hours after admission his hematocrit dropped to 15%, and he became tachycardic, diaphoretic, and hypotensive. A psychiatrist was consulted and determined the patient not capable of making decisions in his best

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From the Department of Family and Community Medicine (SAC, RJP), Albany Medical Center, Albany, NY. Address reprint requests to Scott A. Cohen, MD, Bassett Healthcare–Norwich, 55 Calvary Dr, Norwich, NY 13815.

Table 1. Hematology Laboratory Results in a Man With Scurvy.

Components	Admission*	6+ Hours After Admission	6 Days After Admission
Hemoglobin (g/dL)	7.7 (13.6–16.7)	5.5	11.4
Hematocrit (%)	22.2 (40.0–49.0)	15.8	33.7
Platelets ($\times 10^3/\mu L$)	334 (130–350)	237	374
White cells ($\times 10^3/\mu L$)	7.9 (4.0–9.0)	5.4	7.1
Mean corpuscular volume (μm^3)	79.8 (82.3–93.2)	—	—
Mean corpuscular hemoglobin (g/dL)	27.7 (27.8–31.9)	—	—
Red cell distribution width (%)	12.7	—	—
Erythrocyte sedimentation rate (mm/h)	105		65
Total bilirubin (mg/dL)	2.1 (0.1–1.2)	4.0	1.7
Direct bilirubin (mg/dL)	0.4 (0.0–0.3)	0.5	0.5
Reticulocyte count (%)	2.6	—	3.1

*Normal range given in parentheses.

interests. The patient was then given 4 U of red blood cells, and his clinical condition stabilized. Acute retroperitoneal bleeding was considered when reexamination of the patient revealed diffuse abdominal tenderness; however, an abdominal computed tomographic scan was negative.

After he was stabilized, and because there was no obvious cause of his anemia, a further workup ensued. Iron studies, vitamin B_{12} and folate levels, lactate dehydrogenase, and creatine kinase were all within normal limits. Coombs test was negative. No apparent site of bleeding could be established other than the skin ecchymoses. Elevated bilirubin levels prompted a workup for a hemolytic process, but there was no evidence of hemolysis from the peripheral smear and haptoglobin levels. An underproduction type of anemia was considered when a reticulocyte count of 2.9% was obtained. The reticulocyte response was considered inadequate in relation to the degree of anemia. Hematologists were consulted to help decide whether a bone marrow biopsy would contribute to the diagnosis. They recommended a bone marrow biopsy, the results of which were essentially normal. The hematology consultants noted the anemia was most likely related to anemia of chronic disease, although no chronic disease could be determined. They attributed the rapid drop in hematocrit to aggressive hydration and blood dilution. These explanations, although, did not address the patient's worsening clinical status in conjunction with the declining hematocrit.

In retrospect, consultation and bone marrow biopsy added little useful diagnostic information. An alternate course of action would have been to manage the patient's condition with the initial transfusion to stabilize him, subsequently adding vitamin and iron supplements while awaiting laboratory studies.

We considered a diagnosis of scurvy when no other proposed cause adequately explained the patient's anemia. The clinical findings of ecchymoses, knee effusions, and gingival erythema were all consistent with the pathophysiologic effects of scurvy, which causes blood vessel fragility and bleeding into body tissues. We reviewed the patient's history with the caretakers and discovered that his diet consisted exclusively of white bread, processed cheese, and water. No vitamin supplements were added to this diet. An oral regimen of 500 mg of vitamin C twice daily was begun, and plasma ascorbate levels were measured.

The patient improved gradually, with his hematocrit first stabilizing and then increasing. He began to show an increase in his energy and activity levels. He was discharged on hospital day 6, walking, with a hematocrit of 33%. Plasma ascorbate levels were 0.0 mg/dL, reported 1 week later. Subsequent follow-up examinations after discharge showed a hematocrit of 41% with resolution of the ecchymotic areas and knee effusions.

Discussion

Petechial hemorrhages, ecchymoses, coiled or corkscrew hairs, and gingivitis are common signs of scurvy. Other manifestations, as vitamin C stores are further depleted, include extremity edema, con-

junctival hemorrhages, arthralgias, hemarthroses, anemia, gastrointestinal bleeding, wound healing defects, fatigue, weakness, and weight loss.[1] Ascorbic acid is an essential cofactor in many human biochemical processes, including iron incorporation into heme and collagen cross-linking. Blood vessel fragility thus results from impaired synthesis of the basal laminae. Manifestations of scurvy, such as poor wound healing and bleeding, are the result of defects in collagen synthesis. Unlike other animals, humans are unable to synthesize vitamin C and are dependent on vitamin C from dietary sources. Patients at risk for scurvy include alcoholics and institutionalized patients, as well as those with generally poor nutrition or malabsorption.

Scurvy has been described for centuries. It was recognized as an important problem beginning in the 15th century, corresponding with the time of lengthy seafaring voyages; during which scurvy ravaged the crews of ocean-going vessels. One of the first descriptions was from Portuguese sailor Vasco da Gama in the late 15th century. He described many of his crew as developing severe lassitude and swelling of the legs, hands, and gums. Their condition apparently resolved after eating oranges.

In 1753, the "Treatise on Scurvy" was published by James Lind. It described an experimental study using oranges and lemons to prevent and treat clinical scurvy. Unfortunately, James Lind's theories were temporarily refuted in the 1860s, when limes imported from the West Indies were used. The limes turned out to be a poor source of vitamin C. Subsequent studies led to present-day knowledge of the role of vitamin C in humans.[2,3] Many recent articles have been published describing the history and biochemical function of vitamin C.[3,4]

Recent studies have shown that as many as 2% of institutionalized psychiatric patients might have vitamin C levels less than 0.1 mg/dL.[5] Detrimental effects on immunity are thought to occur at levels three times higher. Although this patient did not undergo endoscopy or arthrocentesis, bleeding into the gastrointestinal tract, joints, and other tissues is the likely cause of his severe anemia. Anemia in scurvy patients can also be attributed to hemolysis and decreased hematopoiesis caused by a diminished ability to incorporate iron into hemoglobin, which might also explain the patient's high bilirubin levels.

Conclusion

Scurvy is a relatively uncommon diagnosis, but the consequences of a missed diagnosis can be dire. Family physicians should consider nutritional deficiencies in psychiatric, institutionalized patients and others at risk for disordered eating.[6–10] A detailed dietary history can reveal clues to the diagnosis.

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