

## Case Report

# Vitamin C Deficiency Leading to Hemodynamically Significant Bleeding

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## Abstract

Vitamin C deficiency generally results from little to no intake of ascorbic acid, and it often presents with small-volume bleeding. Rarely vitamin C deficiency causes hemodynamically significant bleeding. Here we present a middle-aged man who was admitted to the hospital several times for hypotension, recurring lower extremity ecchymoses, and persistent anemia. Extensive workup from previous hospitalizations did not identify the etiology of his recurrent bleeds. Ultimately a detailed dietary history uncovered a lack of fresh fruit and vegetable intake. Vitamin C deficiency was suspected and subsequently confirmed. After receiving multiple blood transfusions to correct his hypovolemic hypotension, he was treated with high-dose ascorbic acid. This case highlights the potential damage of vitamin C deficiency in the industrialized world, and it reminds clinicians to be vigilant of this rare but significant cause of anemia.

## CASE PRESENTATION

A 56 year old otherwise independent and functional man presented to our hospital after an episode of syncope associated with an expanding hematoma in his left leg. Over the previous three months, the patient had been experiencing recurrent episodes of spontaneous leg and thigh hematomas, which had been temporally associated with subsequent syncopal episodes. He adamantly denied any traumatic association with the hematomas. His legs had become increasingly edematous and ecchymotic, leading to significant impairment in his level of activity.

Prior to this admission, he had been admitted twice in the preceding months to outside hospitals for similar recurrent lower extremity hematomas and loss of consciousness. He was found to be profoundly anemic and orthostatic, both of which responded to a total of 6 units of packed red blood cells. He had underwent a seemingly exhaustive work up for bleeding disorders, including multiple CT angiograms, INR/aPTT, clotting factor levels/activity, and bleeding time, all of which were found to be normal. He was in the hematology clinic for evaluation prior to this admission, where his syncope occurred.

His past medical history includes esophageal cancer, which was treated with esophagectomy and chemotherapy eight years prior. He had no known complications from this. He denied a history of bleeding diatheses. He denied any trauma, family history of bleeding disorders, or use of anticoagulants (including

warfarin, aspirin, or NSAIDs). He had a 45 pack year smoking history and was a former heavy drinker who quit both in 1993.

The patient had orthostatic changes by blood pressure and heart rate criteria. His other vital signs were normal. Physical examination on admission showed an obese, slightly pale middle aged Caucasian man in no apparent distress. Examination of the lower extremity revealed tender ecchymoses extending from the medial aspect of the right middle leg up to the mid-thigh. Small punctate, perifollicular hemorrhages were noted on each of his thighs. No hemorrhages or ecchymoses were seen elsewhere on skin exam. Rectal examination showed brown stool which was negative of occult blood. Eye exam showed conjunctival pallor, with the remainder of his exam including the heart, lungs, abdomen, and skin otherwise unremarkable. He was edentulous Table 1.

Laboratory results revealed anemia. Iron studies were consistent with iron deficiency. The reticulocyte studies were appropriately elevated. Vitamin B12 and folate were normal, as were liver function tests. Albumin and pre-albumin were 2.7 (3.3-4.8 g/dL) and 10, respectively. Hemolysis labs showed haptoglobin 317 (34-200 mg/dL) and LDH 161 (90-208 IU/L). PT, aPTT, thrombin time, and factor VII activity were within normal limits.

A CT angiogram of the lower extremities showed no bleeding vessels and no retroperitoneal bleeding. Ascorbic level was 0 mg/dL.

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- Anemia
- Collagen
- Hemorrhage

## DISCUSSION

Prior to admission, ascorbic levels had not been checked, although lab data one month earlier included low Collagen/Epi (54), prolonged Collagen/ADP (300), and increased levels of both von Willebrand Factor Ag (235%, normal 50-150) and vWF activity level (254%, normal 50-170), which may be explained by the patient's anemia and possible vascular damage secondary to defective collagen synthesis in the setting of scurvy. In light of an undetectable ascorbic acid level along with current laboratory data, imaging studies negative for sources of acute blood loss, as well as previous hematologic workup that was unrevealing for any factor deficiencies (including V, VIII, IX, X, and XIII), we determined the etiology of the patient's normocytic anemia to be the result of severe vitamin C deficiency after further questioning of the patient's dietary habits revealed a dearth of vegetable and fruit intake on a daily basis. He attributed this to his ill-fitting dentures which did not allow him to eat regular solid food. He also admitted to small meals due to his total esophagectomy with gastric pull-through in 2003.

**Table 1:** Select laboratory results.

Hemoglobin	7.0 g/dL
Hematocrit	22.2%
WBC	7,100 cells/hpf
Platelets	288,000
MCV	87.4 (80-99)
RDW	16.0 (11.5 to 14.5%).
Fe	27 (40-190 mcg/DL)
TIBC	362 (260-420 mcg/dL)
Ferritin	114 (22-415 ng/mL)
Iron saturation	7 (11-46%)
Reticulocyte percentage	5.05 (0.66 to 2.85%)
Absolute reticulocyte count	128.0 (27.9 to 121.6 k/uL)

**Abbreviations:** WBC: White Blood Cell count; MCV: Mean Corpuscular Volume; RDW: Red cell Distribution Width; Fe: Iron; TIBC Total Iron Binding Capacity



**Figure 1** Right lower extremity ecchymosis.



**Figure 2** Perifollicular hemorrhages on the right lower extremity.

While scurvy seems a quaint diagnosis, often ascribed to sea-faring men during the 17th and 18th centuries, vitamin C deficiency remains a relevant differential diagnosis in the workup of anemia, even in industrialized nations with access to fresh food. In fact, the diagnosis of scurvy is still reported in recent literature [2,3,4,20]. In addition to confirming the diagnosis with an ascorbic acid level, and evaluating for other potential hematologic etiologies of anemia, taking a thorough history—including dietary habits that may be impacted by a patient's social situation—is key in arriving at the correct diagnosis of scurvy, especially when laboratory and imaging workup is unrevealing. In this particular case, our patient's physical exam findings of punctate perifollicular hemorrhages with large ecchymoses and hemodynamic instability, coupled with his history of a diet lacking in fresh fruit and vegetables due to edentulousness, led us to suspect scurvy [7,11].

Our patient was transfused two units of packed red blood cells with an appropriate hemoglobin increase. He was empirically started on vitamin C treatment doses including a one-time intramuscular dose followed by 500 mg oral vitamin C daily. One month later in clinic, his hemoglobin was 13 g/dL and ascorbic acid level was 2.2. He showed complete resolution of his symptoms with no further transfusions, syncope, or hematomas.

"As one Saint noted: It commences with a sharp pain in the ankles, swells, and finally the leg get[s] all black and in many cases it proves fatal." [5].

## ACKNOWLEDGEMENT

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