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Scurvy Induced Hemodynamic Instability

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Authors' contributions

This work was carried out in collaboration between all authors. Author LY acquired analyzed and interpreted the case; drafted and revised the article. Author JF acquired interpreted the case; drafted and revised the article. Author TK acquired analyzed and interpreted the case; drafted and revised the article and approved final version. All authors read and approved the final manuscript.

Article Information

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Case Report

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ABSTRACT

Aim: Our case report aims to inform practicing clinicians of an unusual presentation of vitamin C deficiency in the setting of a developed nation where this illness is rare and underappreciated. Case Presentation: We present the case of a noncompliant 16-year-old African American female with vertically transmitted human immunodeficiency virus/acquired immune deficiency syndrome who presented to the emergency department (ED) with a CD4 count of 4 and a hemoglobin level of 5.7 g/dL. In the ED, she was found to have persistent low-grade bleeding initially believed to be of an upper gastrointestinal origin, but which was later found come from the oral mucosa. Her stools were dark in color and guaiac positive. She was hemodynamically unstable, for which she was transfused with packed red blood cells and briefly treated with continuous norepinephrine infusion. Her initial coagulation studies were noncontributory with an international normalized ratio of 1.1, a prothrombin time of 35, and a platelet count that was also within normal limits. Esophagogastroduodenoscopy and a colonoscopy were both unremarkable. Bone marrow biopsy showed normocellular marrow with 80% cellularity and trilinear hematopoiesis. Her vitamin C level was zero. She was diagnosed with scurvy and treated with vitamin C supplementation. Discussion and Conclusion: Vitamin C deficiency can lead to an often-forgotten medical condition called scurvy. It can cause defective collagen synthesis leading to fragile capillaries, gingival bleeding, and cutaneous changes. Unrecognized, this condition can lead to significant



bleeding and can be lethal in select patient populations. Our case is unique in that it shows that vitamin C deficiency can masquerade as upper gastrointestinal bleeding and may present with significant hemodynamic instability requiring blood transfusions and vasopressor support. It is therefore imperative to keep in mind the diagnosis of scurvy as a potential cause of hemodynamic instability even in an industrialized nation such as the United States.

Vitamin C deficiency is a rare and underdiagnosed medical entity in the hospital setting that can lead to hemodynamic instability. Scurvy patients can present with melena and oral bleeding, mimicking hematemesis.

Keywords: Ovariectomy; estradiol; ibandronate; anti-oxidant enzymes; DEPPD free radical; rat's liver.

1. INTRODUCTION

Scurvy is a disease resulting from a deficiency of Vitamin C. Though described by Hippocrates about 2,000 years ago, scurvy was not effectively treated until its link to vitamin C was established in 1932 when ascorbic acid (chemical name of vitamin C) was first isolated [1]. Scurvy was once the nightmare t for many maritime activities, killing at least 2 million sailors between the 16th and 19th centuries [2].

Today, the developed world enjoys almost universal access to sources of Vitamin C. In the United States, citrus fruits are the predominant source, followed by tomatoes, tomato juice, and potatoes [3]. And yet there remain in the United States select populations who, by choice or as a result of disability, are unable to access these foods. These groups including food faddists, individuals of low socioeconomic status, the isolated elderly, the neglected young, alcoholics with poor nutrition, the institutionalized, and the mentally ill [4,5]. The combination of this syndrome's persistent presence today, uniformly fatal outcomes when left untreated, and simple solution, render it imperative for clinicians to remain familiar with the manifestations of this illness.

A previous study has indicated that approximately 13% of the US population are vitamin C deficient (serum concentrations <11.4 μ mol/L) according to the federal nutrition recommendation, indicating clinicians should remain vigilant of scurvy, particularly in the previously mentioned high-risk populations [6].

2. CASE PRESENTATION

We present the case of a noncompliant 16-yearold African American female. She acquired HIV/AIDS from her mother at birth, and while she had been placed on HAART therapy, her family reported non-compliance. This young woman presented with a six month history of dark stools and shortness of breath on exertion. She was noted to be "coughing up blood" in the emergency department, and her stool was guaiac positive. An initial CBC showed a hemoglobin level of 5.6 g/dl. Her remaining laboratory values, which include PT/PTT/INR and platelet count, were all within normal limits. Her CD 4 count was found to be 4. On presentation. she was hemodynamically unstable and her systolic blood pressure remained in high 70s and early 80s despite adequate fluid resuscitation, and consequently was treated with а norepinephrine drip. She also received packed red blood cells for her symptomatic anemia. The anemia was thought to be from ongoing chronic bleeding from an upper gastrointestinal source. Esophagogastroduodenoscopy and a colonoscopy were both unremarkable. Her clinical course was complicated by hypoxic respiratory failure prompting intubation and transfer to a medical ICU. An arterial line was placed for hemodynamic monitoring. The arterial line was removed once improved was noted, however she continued to ooze blood from the radial artery which was ultimately ligated in the operating room by the general surgery service. A bone marrow biopsy showed normocellular marrow with 80% cellularity and trilinear hematopoiesis was also complicated by oozing blood from the biopsy sites. Several days into her admission it was discovered that the patient was bleeding from her gums which was often obscured by her habit of frequently licking her gingiva. After having ruled out all the other common causes of GI bleed, a vitamin C level was drawn and ordered as a send-out lab. Upon the return of the result a week later, the medical team was surprised to find that the patient's vitamin C level was zero. She was diagnosed with scurvy and treated with vitamin C supplementation.

3. DISCUSSION

Ascorbic acid is crucial in collagen production, aiding with the hydroxylation of collagen.

Furthermore, it is needed in the synthesis of dopamine, norepinephrine, epinephrine, and carnitine [7]. Deficient vitamin C uptake usually leads to onset of the scurvy syndrome within 3 months. Early symptoms include petechiae, ecchymoses, bleeding gums, coiled hairs, hyperkeratosis and impaired wound healing. Late systemic symptoms include weakness, malaise, joint swelling, arthralgias, edema, depression, neuropathy, and vasomotor instability and if left untreated, death [8].

In our case, several significant clinical findings led us to the suspicion of vitamin C deficiency. These include persistent arterial line bleeding that required surgical ligation, hemorrhage from the bone marrow biopsy site despite compression as well as chronic but profuse gingival bleeding in the absence of coagulopathy which suggested a deficiency in primary hemostasis. The absence of childhood bleeding and family history of bleeding disorders deemed congenital primary hemostasis disorder less likely. A normal platelet count left an acquired deficiency of primary hemostasis most likely. Thus, a vitamin C deficiency was suspected in the setting of her disadvantaged socioeconomic background.

The occurrence of scurvy in HIV patients, though uncommon, has been documented in the past. The author's search discovered three such case reports which describe patients who presented with multiple comorbidities ranging in severity from spontaneous bruising, odynophagia and rash to DIC [9-11]. In our case, the anemia exhibited by patient, at 5.7 g/dL, is significantly lower than that found in the literature of a similar case, 9.3 g/dL [11]. This degree of anemia is rare among documented cases of Vitamin C deficiency, and may be attributable to the combined effect of bone marrow suppression effect by HIV, persistent low-grade oral bleeding and patient's delay in seeking medical advice.

Furthermore, the presentation of hemodynamic instability in our case is very unique. The onset of hemodynamic instability suggests acute blood loss played a major role in her severe anemia. More commonly, tissue bleeding with nonpalpable purpura is an early finding, and can also be attributed to capillary fragility secondary to defective collagen synthesis. If fact, case studies have suggested the first sign of Vitamin C deficiency in patients is petechial hemorrhage. Notably, only dentulous patients have been found to experience gum changes [5]. Last but not least, our case masqueraded as upper gastrointestinal bleeding, which was later ruled out by EGD and colonoscopy. A current hypothesis regarding the development of Dieulafoy's lesion describes a pulsatile vessel extending through a mucosal tear. A similar mechanism involving capillaries extending through weakened mucosa may be present in scurvy. This would consequently cause capillary instability manifested as petechiae, ecchymosis, gum bleeding, hemarthrosis, and bone hemorrhage. However, no formal relationship has been established to date [5].

The recommended treatment for scurvy in adult is oral vitamin C supplementation 100 mg 3-5 times a day followed by 100 mg/day. (7) Symptoms usually improve within 3 days, and most physical findings resolve in 2 weeks. Other nutritional deficiencies should be sought in newly diagnosed cases since vitamin C deficiency is not usually an isolated finding [12]. Our patient was treated with 300 mg oral vitamin C daily followed by 100 mg vitamin C supplementation. Her symptom and physical findings improved after replenishment of vitamin C.

Vitamin C deficiency can happen in all age groups, though it most commonly affects the very young and elderly, due to their inability to manage their own nutrition. Risk factors for child neglect are those which suggest a disorganized family system and include parental substance abuse, unemployment, physical disability, or cognitive impairment [13,14]. There are a few cases reports and series mainly dealing with vitamin C deficiency in children and found that the affected age group ranged from infants to adolescents [15]. Our patient was heavily dependent on carbonated beverages and processed food. She was an adolescent patient from a family of low socioeconomic status. Thus, the prevalence of vitamin C deficiency the need demonstrates for community awareness programs, particularly for those from impoverished backgrounds.

Our case report is limited in that it describes an unusual presentation absent in most vitamin C deficient patients, who remain asymptomatic. Further large-scale cross-sectional study may help identifying the accurate prevalence of symptomatic vitamin C deficient patients in highrisk populations. Also, further retrospective studies are also needed to identify specific risk factors to better define high-risk patient groups.

4. CONCLUSION

Mild vitamin C deficiency is not uncommon in United States. Clinicians should be vigilant for the diagnosis of scurvy in high-risk populations as it can be universally fatal if not appropriately treated. Tissue bleeding, including gingival bleeding, is a common manifestation in scurvy patients and may masquerade as upper gastrointestinal bleeding and severe anemia.

CONSENT

All authors declare that 'written informed consent was obtained from the patient (or other approved parties) for publication of this case report and accompanying images.

ETHICAL APPROVAL

It is not applicable.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

REFERENCES

- 1. Stone I. On the genetic etiology of scurvy. Acta geneticae medicae et gemellologiae. 1966;15(4):345-50.
- 2. Whitehead P. The history of scurvy and vitamin C: Med Hist. 1987;31(2):231-2.
- Institute of Medicine Panel on Dietary A, Related C. Dietary Reference Intakes for Vitamin C, Vitamin E, Selenium, and

Carotenoids. Washington (DC): National Academies Press (US) Copyright 2000 by the National Academy of Sciences. All rights reserved; 2000.

- Stephen R, Utecht T. Scurvy identified in the emergency department: A case report. The Journal of emergency medicine. 2001;21(3):235-7.
- Pimentel L. Scurvy: Historical review and current diagnostic approach. The American Journal of Emergency Medicine. 2003;21(4):328-32.
- Schleicher RL, Carroll MD, Ford ES, Lacher DA. Serum vitamin C and the prevalence of vitamin C deficiency in the United States: 2003-2004 National Health and Nutrition Examination Survey (NHANES). The American Journal of Clinical Nutrition. 2009;90(5):1252-63.
- Leger D. Scurvy: Reemergence of nutritional deficiencies. Canadian Family Physician Medecin De Famille Canadien. 2008;54(10):1403-6.
- 8. Wang AH, Still C. Old world meets modern: a case report of scurvy. Nutrition in clinical practice: Official Publication of the American Society for Parenteral and Enteral Nutrition. 2007;22(4):445-8.
- Burdette SD, Polenakovik H, Suryaprasad S. An HIV-infected man with odynophagia and rash. Clinical infectious diseases: An Official Publication of the Infectious Diseases Society of America. 2005;41(5): 686-8:744-7.
- Khonsari H, Grandiere-Perez L, Caumes E. Spontaneous bruising in an HIV-positive patient. La Revue de Medecine Interne/ Fondee Par La Societe Nationale Francaise De Medecine Interne. 2005; 26(12):984-5.
- 11. Maltos AL, Silva LL, Bernardes Junior AG, Portari GV, Cunha DF. Scurvy in a patient with AIDS: Case report. Revista da Sociedade Brasileira de Medicina Tropical. 2011;44(1):122-3.
- De Luna RH, Colley BJ, Smith K, Divers SG, Rinehart J, Marques MB. Scurvy: An often forgotten cause of bleeding. American Journal of Hematology. 2003; 74(1):85-7.
- Gillham B, Tanner G, Cheyne B, Freeman I, Rooney M, Lambie A. Unemployment rates, single parent density, and indices of child poverty: their relationship to different

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categories of child abuse and neglect. Child Abuse & Neglect. 1998;22(2):79-90.

- 14. Berkowitz CD. Fatal child neglect. Advances in Pediatrics. 2001;48:331-61.
- 15. Popovich D, McAlhany A, Adewumi AO, Barnes MM. Scurvy: Forgotten But Definitely Not Gone. Journal of Pediatric Health Care. 2009;23(6):405-15.

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