ACADEMIC: CASE REPORT

A case report on modern-day scurvy

Zhaolun Feng, Samarina Musaad

Abstract
Scurvy, from the Latin word *scorbutus* meaning to rupture or lacerate, refers to the clinical disease caused by nutritional deficiency of Vitamin C (Ascorbic acid), with characteristic mucocutaneous and musculoskeletal features. It was originally described in the 18th century by James Lind and associated with long sea voyages and insufficient citrus consumption. Despite much decrease in prevalence, it still exists in developed countries. This case reports on an older male who presented with general lethargy, gum disease and easy bruising. His dietary history suggested global nutrient deficiency. Investigations excluded common causes of bleeding: trauma, coagulopathies, vasculitis, and malignancy. A low Vitamin C level confirmed the diagnosis of scurvy. This report serves to remind clinicians to consider scurvy as a possible working diagnosis in at-risk patients.

Keywords
Scurvy, Vitamin C, Ascorbic acid, Gum

Background

Mr F is an 87-year-old man with a history of giant cell arteritis being weaned off from prednisone, atrial fibrillation on rivaroxaban and sotalol, osteoarthritis, and previous asymptomatic minimally focally active colitis. He presented to the emergency department with three weeks history of generalised unwellness, watery non-bloody diarrhoea four times a day, reduced appetite, early satiety, and significant involuntary weight loss (14kg over eight weeks). He had no significant history of alcohol use or eating disorders. There was no family history of bowel cancer. Distant relations reported finding both him and his sister frail and bony in their house. He is a carer for his sister, who had deteriorating dementia. He reported carer stress which had impacted his mood and appetite.

Physical examination on the day of admission revealed a dehydrated, jaundiced patient, with pin-prick purple discoloration at the fingertips. He was not engaging, giving one-word answers to questions or shaking/nodding his head. There were no masses or organomegaly, but his skin was thin, with petechiae, ecchymoses and purpuric rashes on his shins. First and second heart sounds were heard without any murmurs. Subsequently, he developed hypothermia and hypoglycaemia.

Investigations revealed a total bilirubin level of 40 (ref <25 umol/L), and borderline hypoalbuminemia with normal liver enzymes and lipase. Cryoglobulins were not detected, which excluded cryoglobulinemia as a cause of fingertip pathology. The coagulation screen was within the pattern of rivaroxaban use. His eGFR was 59 mL/min/1.73m^2. Abdominal ultrasound and contrast computerised tomography (CT) of the chest, abdomen and pelvis on admission demonstrated no features of malignancy. A myeloma screen was negative, with normal IgG, IgA, and IgM levels, and no monoclonal bands on serum electrophoresis. Stool was negative for *Clostridium difficile, Cryptosporidium*, and *Giardia* species. Colonoscopy was not performed due to his frail functional status, but flexible sigmoidoscopy showed mucosal erythema excluding inflammatory colitis as a cause. A biopsy of the left colon was done and showed oedematous, focally haemorrhagic large bowel mucosa, which excluded microscopic colitis. He also had normal faecal elastase >800 (ref: >200 ug/g), negative coeliac serology (tTg IgA <1.9 CU and DGP IgG <2.8 CU), non-specific calprotectin rise to 108 (ref: <50 ug/g), and normal iron studies. Vitamin C level, which returned four weeks later, was 7 (ref: 26–85 umol/L). A fibreoptic endoscopic evaluation of swallowing (FEES) study and video-fluoroscopic swallow study (VFSS) showed moderate oropharyngeal dysphagia, alongside a normal gastroscopy. The vasculitis screen was normal. Full blood counts showed normocytic anaemia with a gradual drop in haemoglobin from 137 g/L to 105 g/L over a seven-week period.

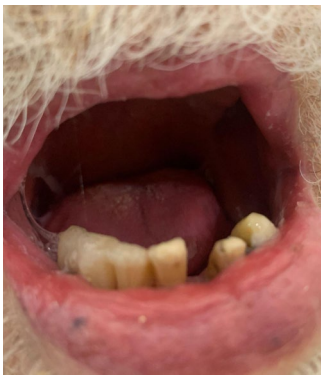

*Figure 1: Tooth loss and limited view of gum prior to Vitamin C treatment. No overt sign of scorbutic gums.**

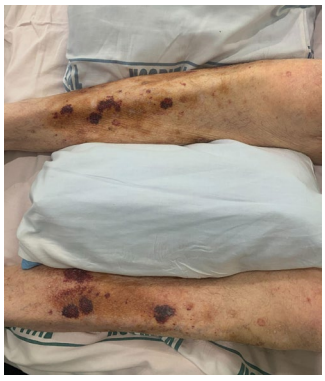

Figure 2: The legs of an 87-year-old man with scurvy, showing perifollicular haemorrhages and ecchymo

Treatment

In the second week of admission, after the vitamin C level was sent out, the patient was commenced on nasogastric feeding and received daily 1 g Vitamin C replacement initially for a week, followed by 500 mg daily in the following weeks.[2] Meanwhile, he received Vitamin B complex supplements (thiamine 10 mg, riboflavin 4 mg, nicotinamide 60 mg, pyridoxine 4 mg) daily. His episode of undifferentiated ischaemic colitis or infective colitis self-resolved. His skin lesions also resolved, and his gum disease was slowly improving. His bilirubin level normalised in subsequent readings; it was attributed to the resolution of a possible indirect bilirubin elevation due to haemolysis in the context of scurvy.[1]

*Scorbutic gums refer to gingivitis and gingival haemorrhages caused by breakdown of gingival capillary components.

Outcome and follow-up

He was transferred to an older adults' rehabilitation ward, given his protracted illness with significant deconditioning, and assessed for suitability for private hospital level of care with ongoing nasogastric feeding.

Discussion

The prevalence of vitamin C deficiency in the developed world varies from 0.4% to 26%.[3] The CHALICE Cohort Study reported a 2.4% New Zealand prevalence.[4] Two scurvy case reports in New Zealand could be identified from the PubMed database.[5,6] Depletion of vitamin C in the human body usually takes place from 4–12 weeks after intake stops. The human body is reliant on dietary vitamin C intake, mainly from fruits and vegetables, due to the lack of genes coding for hepatic enzyme L-gulonolactone oxidase, which is needed for the last step of the vitamin C synthetic pathway.[7] It plays a significant role in type IV collagen synthesis through transcription of pro-collagen catalysed by lysyl hydroxylase in the skin, blood vessels and tissue.[7] Reduced social support and recent gastrointestinal illness were risk factors for Mr F's Vitamin C deficiency leading to his gum and mucocutaneous bleeds.[8] The diagnosis of scurvy was made in the second week of admission based on the overall clinical picture of an at-risk demographic with gum disease and bruising typical of scurvy, without biochemical or radiological findings of malignancy, infection, or vasculitis. This diagnosis was confirmed by a low blood level without histopathological samples taken. This was managed with oral Vitamin C replacement as recommended by DermNet. Treatment was initiated before Vitamin C level results were available as its turnaround time is about four weeks, given it is a send-away sample to Canterbury Health New Zealand.[9] The benefit of treating outweighs the risk of over-supplementation, though its excretion in urine may increase renal oxalate excretion and promote renal stone formations.[7] Within a week of treatment, Mr F demonstrated remarkable improvement.

Lack of social support was considered a primary condition causing scurvy and was addressed with an increased level of care. Scurvy remains an under-diagnosed condition.[10] This report encourages physicians to recognise risk factors and keep a high index of suspicion based on clinical features.

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Patient consent

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Conflict of interest

There are no conflicts of interest or funding received.

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