Scurvy

Dear Editor,

Scurvy is a nutritional disorder which can develop after prolonged (>1-3 months) severe vitamin C deficiency. Vitamin C is a cofactor in several enzyme reactions involved in collagen synthesis. The defect in collagen causes blood vessel fragility, poor wound healing, mucocutaneous bleedings, hair abnormalities, bone pains, and joint contractures due to periosteal and intraarticular bleeding (1,2). Risk factors for scurvy development are undernutrition, low socioeconomic status, older age, male sex, alcoholism, tobacco smoking, and severe psychiatric illnesses (1-3). The required daily intake for vitamin C is ~60 mg, and this amount of vitamin C can be found in only one medium-sized orange. For this reason, the disease is rarely encountered in developed countries and is often underrecognized by healthcare personnel. Herein, we present an illustrative case of scurvy in order to raise the awareness of this disorder.

A 61-year-old Caucasian man was admitted to hospital due to fatigue, hypotension (80/50 mmHg), severe normocytic anemia (hemoglobin 76 g/L), kidney failure (estimated glomerular filtration rate of 6 mL/min/1.73m^2) and mild elevation in C-reactive protein (30.9 mg/L). Prior medical history included radical cystoprostatectomy with an ileal conduit performed eight years ago due to a bladder tumor and moderate chronic kidney disease with recurrent urinary tract infections. The patient was also an alcoholic and tobacco smoker, with a very low-income and a poor diet. He did not use any medications. Heteroanamnestically, the current clinical state had developed slowly over several weeks. At admission, the patient was afebrile, lethargic, malnourished, and immobile due to generalized weakness, bone pains, and hip and knee contractures. He had generalized edema, mostly related to kidney failure, as well as severe

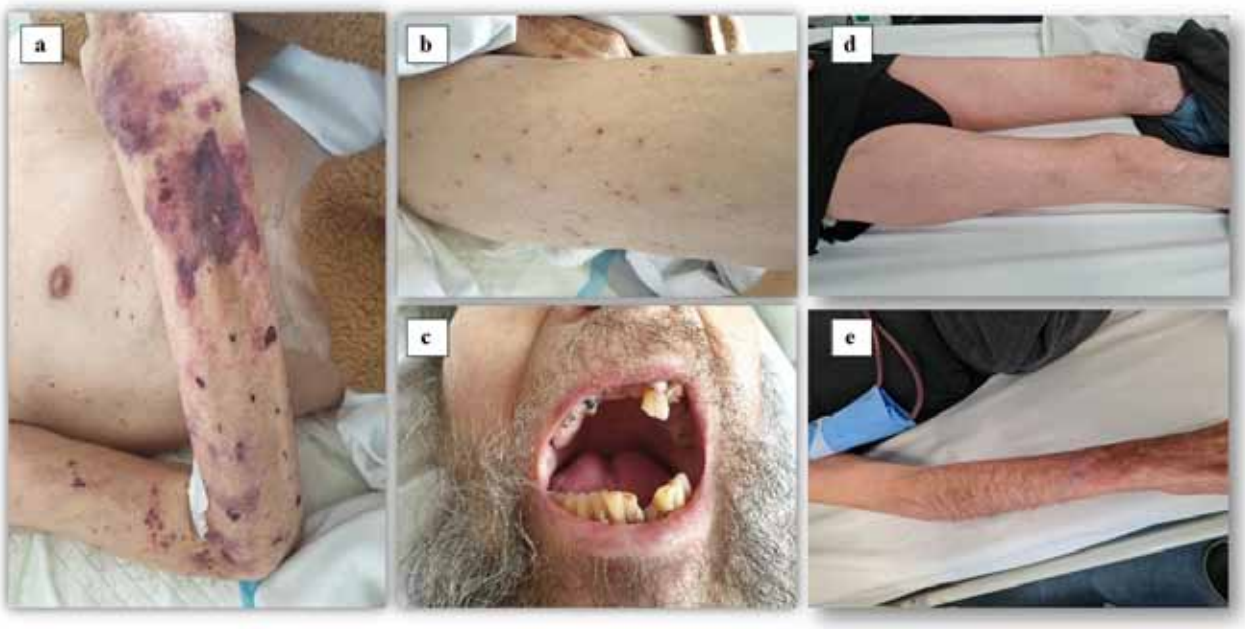

Figure 1. Typical mucocutaneous findings in scurvy and the rapid clinical response to treatment with vitamin C: (a) ecchymoses and poor wound healing, (b) perifollicular bleedings, (c) "corkscrew" facial hair and defective tooth, (d) resolution of perifollicular bleedings, (e) resolution of ecchymoses.

hypoalbuminemia (serum albumin 19 g/L). There were multiple ecchymoses (Figure 1, a) and perifollicular bleedings (Figure 1, b) in the skin. The teeth were defective, and the patient's facial hair had a "corkscrew" appearance (Figure 1, c). The platelet count was normal, as was the serum fibrinogen level and the prothrombin- and activated partial thromboplastin times. Vancomycin-resistant *Enterococcus faecium* and multi-drug-resistant *Acinetobacter baumanii* were isolated from the urine. Therefore, hemodialysis, linezolid, and colistin were started. However, the patient continued to be lethargic, immobile, and with prominent skin bleeding. Medical workup excluded the possibility of an underlying malignancy or an autoimmune disorder. Finally, scurvy was suspected and 500 mg daily of oral vitamin C was introduced into therapy. In the following two weeks, the general condition of the patient significantly improved and he was discharged from the hospital in good condition – mobile and with complete resolution of skin lesions (Figure 1, d and e). Three months later, the patient was still under maintenance hemodialysis and had mild anemia (hemoglobin 123 g/L).

Interestingly, scurvy was the first disease in the history of medicine for which a randomized trial found a cure (4). The differential diagnosis of scurvy includes skin infections, hematologic disorders, collagen vascular disorders, and anticoagulant/antiplatelet side-effects (1). Pathognomonic skin findings which may help raise suspicion of scurvy are perifollicular bleedings and "corkscrew" hair. Notably, laboratory testing for vitamin C concentration is not necessary to confirm scurvy as it tends to reflect recent dietary intake of vitamin C (2). Nevertheless, it may be useful to identify less typical cases (2). In our case, rapid clinical improvement with the resolution of skin lesions and joint contractures after the introduction of vitamin C confirmed the clinical diagnosis of scurvy. Additionally, vitamin C deficiency could be, at least partly (besides kidney failure and acute infection), responsible for severe anemia at disease presentation (5). This case serves to remind clinicians not to forget scurvy when treating patients at risk for vitamin C deficiency who present with fatigue, anemia, bone pains, and unexplained mucocutaneous bleedings. In suspected cases, vitamin C should be administered without hesitation.

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Ivan Krečak[1*], Gordan Babić[1], Marko Skelin[2,3]

[1]Department of Internal Medicine, General Hospital of Šibenik-Knin County, Šibenik, Croatia
Faculty of Medicine, University of Rijeka, Rijeka, Croatia

[2]Pharmacy Department, General Hospital of Šibenik-Knin County, Šibenik, Croatia

[3]Department of Basic and Clinical Pharmacology with Toxicology, Faculty of Medicine, University of Rijeka, Rijeka, Croatia

***Corresponding author:**
Ivan Krečak, MD, PhD
Department of Internal Medicine, General Hospital of Šibenik-Knin County
Stjepana Radića 83
22000 Šibenik
Croatia
krecak.ivan@gmail.com

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