Severe vitamin C deficiency in a critically ill adult: a case report

Doll, Sébastien; Ricou, Bara

How to cite

DOLL, Sébastien, RICOU, Bara. Severe vitamin C deficiency in a critically ill adult: a case report. In: European journal of clinical nutrition, 2013, vol. 67, n° 8, p. 881–882. doi: 10.1038/ejcn.2013.42

This publication URL: https://archive-ouverte.unige.ch/unige:42349
Publication DOI: 10.1038/ejcn.2013.42

European Journal of Clinical Nutrition (2013) **67**, 881–882

www.nature.com/ejcn

CLINICAL CASE REPORT

Severe vitamin C deficiency in a critically ill adult: a case report

S Doll and B Ricou

Scurvy, a severe form of vitamin C deficiency, killed scores of people until its cause and treatment were firmly established at the end of the eighteenth century. Since then, cases have surged periodically around the world, mostly in developing countries and during times of war and famine. In developed countries, scurvy is still endemic and evidence is growing that vitamin C deficiency might affect up to 30 percent of the population. Low socio-economic status, alcoholism, severe psychiatric illness leading to poor nutrition and critical illness are significant risk factors. We hereby report the case of a patient admitted in a Swiss intensive care unit of a tertiary teaching hospital and presenting with clinical signs and symptoms of severe vitamin C deficiency.

European Journal of Clinical Nutrition (2013) **67,** 881–882; doi:10.1038/ejcn.2013.42; published online 3 April 2013

Keywords: scurvy; vitamin C deficiency; intensive care; multiple organ dysfunction syndrome

CASE

A 79-year-old man was brought to the emergency department because of a progressive onset of confusion associated with marked lethargy. His only complaint consisted of diffuse bone pains. His relatives reported significant weight loss over the past 3 months, associated with anorexia and non-bloody diarrhea. A treatment of levofloxacine had been started 10 days before admission because of suspected pneumonia.

Upon clinical examination, the patient was lethargic. Hemodynamic parameters showed mild hypotension (96/66 mm Hg) associated with tachycardia (97/min), despite a cumulated fluid challenge of 3 l. Oral inspection revealed diffuse and spontaneous bleeding of the gums, which appeared blackened. Many teeth were missing, with the few remaining ones rotten and ready to fall. Subcutaneous edema was noted, involving predominantly the lower limbs, and was associated with purpuric lesions over the legs and forearms. Abdominal auscultation was normal and palpation was painless.

Initial lab results (including normal values in brackets) showed a mild leukocytosis at 14 G/l (4–11), without any band shift, and serum C reactive protein level was slightly elevated, at 58 mg/l (0–10). Moderate anemia was present, at 111 g/l (140–180), with associated macrocytosis (107 fl (82–98)) and few schistocytes. Coagulation tests were abnormal, with a prothrombin ratio at 26% (>70%), factor VII–X at 16% (>70%) and factor V at 68% (>70%). Renal function tests indicated renal failure, with creatininemia elevated at 196 μmol/l (62–106), while urea was normal, at 4 mmol/l (2.8–7.1). Ferritine was slightly elevated at 758 μg/l (26–417). Glucose, electrolytes and liver tests were within normal range, as were blood levels of cyanocobalamine, folate, cortisol, TSH thiamine-pyro-phosphate and pyridoxal-5-phosphate.

A thoraco-abdominal CT scan was performed, which showed mild and bilateral lung effusions, a partial left lung atelectasis, and free and diffuse abdominal liquid in moderate quantity without signs of bowel inflammation or perforation.

Suspecting a septic shock of either abdominal or pulmonary origin, hemocultures were drawn and intravenous ceftriaxone plus metronidazole were started. The patient was then admitted to the ICU on low doses of noradrenaline. Oral vitamin C supplements at 300 mg/day were also started, along with vitamin B1 and enteral feeding.

On the second night and after initial stabilization, the patient presented with a coma of rapid onset, which required emergent oro-tracheal intubation. Over that same night, he developed multiple-organ dysfunction syndrome manifested by a major hemodynamic instability requiring high doses of noradrenaline, acute respiratory failure with severe and refractory hypoxemia, and anuric acute kidney failure. A moderate pericardic effusion was noted on trans-thoracic echocardiography, without signs of tamponade. In spite of sustained reanimation maneuvers, he passed away the following morning.

While none of the bacteriological cultures ever returned any positive results, admission vitamin C S-ascorbate dosage eventually showed severely lowered values, at levels of 3.0 μmol/l (17–85).

DISCUSSION

Many characteristic symptoms and signs of severe vitamin C deficiency, upon which the diagnosis of scurvy is established,[1] were present in our patient: general symptoms (fatigue and lethargy), oral findings (spontaneous bleeding, gum retraction and loss of dentition), cutaneous abnormalities (including hemorrhagic skin lesions predominantly in the lower limbs) and musculoskeletal findings (severe pain upon mobilization). In addition, plasma vitamin C levels were well below <11.4 μmol/l, a lower value associated in the literature with significant vitamin C depletion.[2]

Vitamin C acts as a unique cofactor for at least eight enzymes involved in the synthesis of collagen, carnitine and norepinephrine. It may also take part in the body anti-oxidant system through its redox potential, buffering harmful oxygen-free radicals produced during pathological conditions such as sustained anaerobic respiration and lasting inflammation.[3] Its critical role in the development of scurvy, through deficient hydroxylation of proline and lysine residues leading to collagen structural

Adult Intensive Care, Department of Anesthesiology, Pharmacology and Intensive Care, University Hospital of Geneva (HUG), Geneva, Switzerland. Correspondence: Dr S Doll, Adult Intensive Care, Department of Anesthesiology, Pharmacology and Intensive Care, University Hospital of Geneva (HUG), Rue Gabrielle-Perret-Gentil 4, 1205 Geneva, Switzerland.
E-mail: Sébastien.doll@hcuge.ch
Received 21 January 2013; accepted 24 January 2013; published online 3 April 2013

instability, has been thoroughly described.[4] It is hypothesized that in our patient's case severe vitamin C depletion induced pulmonary alveolar hemorrhage, lung effusions, progressive myocardial hemorrhage with hemopericardium and impaired vasoconstriction following adrenergic stimuli, all leading to multiple organ failure, refractory hemodynamic collapse and, eventually, death.

The cause of our patient's vitamin C deficiency was attributed to a diet consisting almost exclusively of canned pasta, as reported by the patient's relatives. While vitamin C content is significantly reduced upon cooking and boiling, its preservation also depends on the food-processing methods. It is lowest in canned food when compared to frozen or fresh-cooked foods. A sepsis of either digestive or pulmonary origin, leading to sustained metabolic demand, might have acted as a precipitating factor. No other potential contributing factors to vitamin C depletion such as smoking, chronic diseases, malabsorbtion or excessive elimination such as in chronic hemodialysis could be identified.

Vitamin C supplementation criteria and recommendation for intake have been proposed for the general population.[5] For critically ill patients on parenteral nutrition, the recommendation for daily vitamin C supplements ranges from 100 to 3000 mg per day, depending on renal function and the severity of the underlying disease.[6] In severe cases of scurvy associated with multiple organ dysfunction syndrome, high-dose supplementation has been shown to be associated with rapid improvement of the clinical situation without any significant side effects.[7] In our case, supplementation did not improve the outcome, although a dosage of 300 mg per day was possibly suboptimal.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

REFERENCES

1 Hirschmann JV, Raugi GJ. Adult scurvy. *J Am Acad Dermatol.* 1999; **41**: 895–906.
2 Levine M, Eck P. Vitamin C: working on the x axis. *Am J Clin Nutr* 2009; **90**: 1121–1123.
3 McGregor GP, Biesalsky HK. Rationale and impact of vitamin C in clinical nutrition. *Curr Opin Clin Nutr Metab Care* 2006; **6**: 697–703.
4 Peterkofsky B. Ascorbate requirement for hydroxylation and secretion of procollagen: relationship to inhibition of collagen synthesis in scurvy. *Am J Clin Nutr* 1991; **54**: S1135–S1140.
5 Levine M, Rumsey SC, Rushad D, Park JB, Wang Y. Criteria and recommendations for vitamin C intake. *JAMA* 1999; **281**: 1415–1423.
6 Berger MM. Vitamin C requirements in parenteral nutrition. *Gastroenterology* 2009; **137**: S70–S78.
7 Kiefer P, Thannberger P, Wilhelm JM, Kieffer C, Schneider F. Multiple organ dysfunction dramatically improving with the infusion of vitamin C: more support for the persistence of scurvy in our 'welfare' society. *Intensive Care Med* 2001; **27**: 448.