CASE REPORT

Scurvy in an alcoholic patient treated with intravenous vitamins

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SUMMARY

Vitamin C deficiency is rare in developed countries but there is an increased prevalence in chronic alcohol abusers. In the UK, it is common practice to treat patients with chronic alcoholism who are admitted to hospital with intravenous vitamins B₁, B₂, B₃, B₆ and C for 2–3 days, followed by oral thiamine and vitamin B-compound tablets. This is a case of a 57-year-old man with a history of chronic alcoholism and chronic obstructive lung disease who was admitted to the intensive care unit for pneumonia requiring ventilatory support. He was given high doses of intravenous vitamins B₁, B₂, B₃, B₆ and C for 3 days then oral thiamine and vitamin B compound tablets but developed scurvy 4 days later. He was restarted on oral vitamin C supplementation and showed signs of improvement within 3 days of treatment.

BACKGROUND

Vitamin C is an essential vitamin that is involved in important physiological cellular processes and tissue development. At a molecular level, vitamin C is needed in the Krebs's cycle for the formation of ATP. At a cellular level, vitamin C is involved in the synthesis of collagen and tissue healing. Humans are incapable of synthesising vitamin C. Vitamin C deficiency can lead to multisystemic disease and death if left untreated.

Scurvy is the clinical manifestation of severe vitamin C deficiency and is rarely seen in developed countries though certain patient groups have a higher prevalence. The prevalence of vitamin C deficiency within high risk groups in the UK have been reported to be as high as 25% in men and 16% in women.¹ Chronic alcoholism plays an important role in the development of vitamin C deficiency. Intravenous replacement of vitamins B and C in hospitalised patients with chronic alcoholism is standard practice. To the best of our knowledge, this is the first case report in current literature to report the inadequacy of a common intravenous vitamin replacement regimen to correct vitamin C deficiency in an alcoholic patient.

On examination he was found to be unkempt but alert and oriented. He had bilateral expiratory wheeze and right basal crepitations. Cardiovascular examination was normal and abdominal examination revealed a palpable liver edge. Arterial blood gas analysis was normal and chest X-ray demonstrated opacity in the right-middle zone.

He was diagnosed with community-acquired pneumonia, started on intravenous antibiotics, oral steroids, nebulised bronchodilators and transferred to the ward. Within 24 h, he became hypotensive, developed type 2 respiratory failure and was transferred to the intensive care unit (ICU) for blood pressure and respiratory support.

Low-molecular-weight heparin was started for thromboprophylaxis, intravenous vitamins B₁, B₂, B₃, B₆ and C were administered for 3 days in a preparation known as 'Pabrinex'. This was then changed to thiamine and vitamin B compound tablets (vitamin B costrong) which was administered via a nasogastric tube. The patient was receiving standard nasogastric feed while sedated and ventilated in the ICU.

The patient showed good clinical response to treatment; however on day 7 of the ICU stay, nursing staff on turning the patient noted a worsening rash on the patient's back ([figure 1](#)). The rash

Figure 1 Photograph of patient's back on day 7 of intensive care unit stay.

CrossMark

CASE PRESENTATION

A 57-year-old man with a history of chronic alcohol excess and chronic obstructive pulmonary disease presented to the accident and emergency department with signs and symptoms of a right-sided pneumonia. Routine observations noted fever of 38.3°C, normal blood pressure and heart rate, respiration rate of 25 bpm and oxygen saturations of 93% on room air.

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Figure 2 Petechial and purpuric rash on patient's lower back.

had both petechial and purpuric elements (figure 2), was distributed on his upper limbs and back but was gradually spreading to the anterior chest wall. The consultant microbiologist at this point substituted the intravenous piperacillin and tazobactam for meropenem due to concerns the rash could be a penicillin-related drug reaction. A dermatology opinion was also requested. Given the nature of the non-blanching rash and persistent neutrophilia, the consultant intensivists requested for a lumbar puncture to exclude infective meningitis that could have seeded from the pneumonia.

Cardiovascular, respiratory and abdominal re-examination was unremarkable but it was noted that the patient had thin brittle hair, very poor dentition and mild gingival bleeding without any obvious trauma that could be caused by contact with the endotracheal tube in situ. A provisional diagnosis of scurvy was made and a serum vitamin C level was sent for analysis. The patient was started on nasogastric vitamin C supplements and showed signs of clinical improvement within 2–3 days of treatment.

INVESTIGATIONS

A summary of the investigations is as follows:

Investigation	On admission	Day 7 ICU (onset of rash)	Normal range
Leucocyte count ($\times 10^9/L$)	14.9	15.5	3.7–11.1
Eosinophil count ($\times 10^9/L$)	0.2	0.2	0.2–0.5
C reactive protein (mg/L)	412	97	0–6
Platelet count ($\times 10^9/L$)	159	116	150–450
Albumin (g/L)	16	19	35–50
Bilirubin ($\mu\text{mol/L}$)	25	19	3–21
Alkaline phosphatase (IU/L)	43	239	30–130
Aspartate transaminase (IU/L)	53	100	16–50
γ Glutamyl transpeptidase (IU/L)	187	930	12–62
Clotting studies	Normal	Normal	
Renal function and electrolytes	Normal	Normal	

No organisms were grown in peripheral and central blood cultures. Sputum cultures from admission grew *Haemophilus influenza* and *Enterobacter*. Analysis of the cerebrospinal fluid (CSF) was unremarkable.

A CT scan demonstrated small cavitating lesions in both lungs with characteristics of *Klebsiella* infection and there was fatty infiltration within the liver consistent with the diagnoses of community-acquired pneumonia and alcoholic liver disease, respectively.

The vitamin C level was undetectable at <5 ($\mu\text{mol/L}$) which confirmed the diagnosis of scurvy.

DIFFERENTIAL DIAGNOSIS IF RELEVANT

The differentials for the rash in this case were

- **Drug-related rash/reaction:** The sudden onset of a rash in a hospitalised patient receiving new medication always raises the suspicion of a drug-induced reaction. However, the normal eosinophil count of $0.2 \times 10^9/L$ (normal 0.2 – $0.5 \times 10^9/L$), the nature of the rash, along with the clinical picture did not support the diagnosis of a drug-related reaction.
- **Heparin-induced thrombocytopenia (HIT):** The fall in platelet count to $116 \times 10^9/L$ (normal 150 – $450 \times 10^9/L$) which coincided with the onset of the petechial-purpuric rash made HIT a possible differential diagnosis. However on repeat blood tests 12 h later, the platelet count had normalised despite the patient still being on heparin. This made the diagnosis of HIT unlikely.
- **Infective meningitis:** With evidence of a community-acquired pneumonia which could have been pneumococcal in origin and the onset of a non-blanching petechial rash, meningococcal meningitis was an important differential diagnosis to be excluded. History and neurological examination was of limited value as the patient was sedated; however, the normal CSF analysis ruled out the possibility of meningitis or encephalitis.
- **Vasculitis:** Undiagnosed vasculitis causing pulmonary infiltrates, pneumonia and the petechial-purpuric rash was the last and least likely of the differential diagnoses on our list. A screen for serum autoantibodies was performed and results were unremarkable.

TREATMENT

The patient had been given conventional treatment of high-dose vitamins B₁, B₂, B₃, B₆ and C intravenously in a preparation known as Pabrinex—two doses a day for 3 days which was then changed to nasogastric thiamine (100 mg 3 times a day) and vitamin B compounds (vitamin B costrong tablets, 1 tablet twice a day). This however was clearly inadequate at correcting vitamin C deficiency as evidenced by the clinical manifestation of scurvy and the undetectable levels of serum vitamin C on day 7 of the ICU stay.

Oral vitamin C supplementation (100 mg 3 times a day) was started alongside the thiamine and vitamin B compound tablets that were given to the patient via a nasogastric tube. The patient received meropenem and clarithromycin for his pneumonia and was discharged home with oral vitamin supplementations.

OUTCOME AND FOLLOW-UP

After 2 days of further vitamin C treatment, the rash was visibly less florid and the gingival bleeding had stopped. The patient spent another week in the ICU and was discharged home from a medical ward after spending a total of 25 days in hospital.

Unfortunately this gentleman did not attend his outpatient follow-up appointment. A recent home visit 3-month post-discharge confirmed complete resolution of the rash (figure 3) and gingival bleeding.

Figure 3 Resolution of rash 3 months after hospital discharge.

DISCUSSION

Severe vitamin C deficiency causes the clinical features of scurvy. Scurvy is rarely seen in the UK, and so it is important for clinicians to be able to recognise its clinical features when it presents in high-risk individuals. The table below illustrates common (in *italics*) and uncommon signs and symptoms that have been reported in vitamin C deficient states.

Chronic alcoholism can lead to vitamin C deficiency in several ways: (1) malnutrition through self-neglect or poverty, (2) malabsorption from chronic diarrhoea secondary to alcohol or chronic pancreatitis and (3) increased urinary excretion of vitamin C caused by alcohol.² Features of vitamin C deficiency such as malaise, loss of appetite, diarrhoea, a propensity towards bleeding and jaundice could be easily masked and attributed to coexisting liver disease. Symptoms and complications of alcoholic liver disease could also be worsened by untreated vitamin C deficiency.

Within the UK, it is common practice to give hospitalised chronic alcohol abusers high doses of intravenous vitamins, often over 2–3 days to treat vitamin deficiencies and prevent complications such as Wernicke-Korsakoffs' syndrome. Vitamins B₁, B₂, B₃, B₆ and C are given intravenously in the form of a commercial preparation commonly known as 'high-dose Pabrinex'. Pabrinex is the only licensed intravenous preparation containing vitamin C and the various vitamin Bs in the British National Formulary. It comes in a set of either two 5 mL ampoules or two 10 mL ampoules. 'High-dose Pabrinex' refers to the 10 mL ampoules—it is ampoule 2 which contains 1000 mg of vitamin C. Usually 4–6 doses are administered over 2–3 days. Patients are then given oral thiamine (100–300 mg daily) and vitamin B compound tablets (1–2 tablets twice daily).

The normal body store of vitamin C is estimated to be 1500 mg in an average healthy man and the clinical features of scurvy usually occurs after 3 months of vitamin C deficiency when levels fall below 0.1 mg/dL.³ Therefore, many physicians would assume the commonly used regimen described above would be more than adequate to correct vitamin C deficiency.

This case report however highlights the inadequacy of this regimen and demonstrates chronic alcohol abusers who are at risk of longstanding vitamin C deficiency may need a prolonged course of vitamin C supplementation either intravenously or orally, especially in those who are acutely ill or critically unwell. This is possibly because vitamin C is a negative acute phase reactant and there is increased use of vitamin C in the cellular processes involved in inflammation and tissue repair, particularly in acute critical illness and infectious states. Further GI and renal losses of vitamin C (eg, from intestinal failure or emergency renal replacement therapy) in the critically ill chronic alcoholic patient may also occur and contribute to a vitamin C-deficient state.

In specialist liver centres such as King's College Hospital, intravenous vitamin C supplementation is often given to the acutely unwell or critically ill liver patient for 1 week before switching to oral supplementation. We should therefore consider either a prolonged course of intravenous vitamin replacement or the addition of oral/nasogastric vitamin C supplementation (100 mg once to three times a day) in patients with chronic alcoholism and alcoholic liver disease.

Learning points

- ▶ Vitamin C deficiency is rare in the general population but common in high-risk groups such as chronic alcohol abusers.
- ▶ The cause of vitamin C deficiency in chronic alcoholism is multifactorial and poor nutrition as well as extragastrointestinal losses need to be considered.
- ▶ The commonly used regimen of intravenous vitamin C supplementation (two to three times a day for 3 days) may not be sufficient to correct long standing vitamin C deficiency in chronic alcohol abusers, especially in the acutely unwell or critically ill.
- ▶ Features of vitamin C deficiency could be attributed and masked by features of liver disease and we should be vigilant of vitamin C deficiency in patients with chronic alcohol excess.

Organ/system	Signs or associations	Symptoms
Skin	<i>Purpura, petechia</i> , ecchymosis, <i>perifollicular hyperkeratosis</i> , alopecia, splinter haemorrhages	Dry, brittle skin, bruises
Eyes	Conjunctival haemorrhage, <i>intraocular haemorrhage</i>	Dry eyes, blurred vision
Mouth	<i>Gingivitis</i> , loss of teeth	<i>Gingival bleeding</i>
Respiratory system	Recurrent chest infections in smokers	Dyspnoea
Cardiovascular system	Cardiac hypertrophy, cardiac failure, haemopericardium, coronary artery disease, <i>anaemia</i>	Dyspnoea
Gastrointestinal (GI) system	Jaundice, upper GI bleeds	Loss of appetite, weight loss, diarrhoea
Genitourinary system	Urinary tract infections	Haematuria
Neurological system	Seizures, <i>pseudoparalysis</i> , <i>neuropathy</i>	—
Musculoskeletal system	Haemarthrosis, scorbutic rosary (chest wall deformity seen in children), pathological fractures, dislocations, <i>osteopenia</i>	<i>Myalgia (especially legs)</i> , arthralgia
General	<i>Irritability (in children)</i> , fever	Lethargy, malaise

Contributors JO treated the patient in the intensive care unit, gained consent for case report and photographs and followed up with the patient 3 months postpresentation. RR was involved in ward-based care after discharge from the intensive care unit and helped in editing the manuscript before submission.

Competing interests None.

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