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HAEMORRHAGIC PERICARDIAL EFFUSION AS THE PRESENTING SYMPTOM OF SCURVY

Hajar Joulal, Jaouad Yousfi, Laïla Benjilali, Mouna Zahlane, Lamiaa Essaadouni

Department of Internal Medicine, University Hospital Center of Mohammed VI, Marrakech, Morocco

Corresponding author: Hajar Joulal **e-mail:** hjoulal@gmail.com

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ABSTRACT

Introduction: Vitamin C deficiency (or scurvy) usually takes weeks to become apparent as cutaneous signs and impaired wound healing. Haemorrhagic pericarditis remains a rare complication of scurvy, which has never been reported as an isolated condition. We report the case of a haemorrhagic pericarditis revealing a vitamin C deficiency in a 56-year-old patient.

Case description: A 56-year-old woman presented with a 2-week history of worsening chest pain and dyspnoea, with no significant medical history. Upon admission, the patient exhibited tachycardia, tachypnoea, low blood pressure, elevated jugular venous pressure, muffled heart sounds and multiple petechiae on her lower limbs. An ultrasound revealed a large pericardial effusion, and an emergency pericardiocentesis was performed, which yielded haemorrhagic fluid without atypical cells. An initial workup including haemoculture, PT and PTT, tuberculosis workup, autoantibodies, tumour markers and infectious disease was negative. A whole-body CT scan showed no evidence of tuberculosis or lymphoma. Additional testing showed that her vitamin C level was <3 umol/L. Following stabilisation, high-dose vitamin C therapy was initiated. Subsequently, she showed continued clinical improvement and remained asymptomatic upon her discharge.

Discussion: While uncommon, it is crucial to investigate vitamin C deficiency when confronted with an unexplained haemorrhagic pericardial effusion, particularly in patients with risk factors.

Conclusion: Our case highlights the significance of early detection of this condition in promptly addressing the diverse complications of scurvy, thereby enhancing the prognosis of a potentially fatal condition.

KEYWORDS

Haemorrhagic pericarditis, vitamin C, scurvy

LEARNING POINTS

- Haemorrhagic pericarditis could be an initial indication of scurvy.
- Vitamin C deficiency must be included in the differential diagnostic of haemorrhagic tamponade, even in the absence of a typical signs and symptoms of scurvy.

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INTRODUCTION

Ascorbic acid or vitamin C is a water-soluble vitamin and an essential dietary nutrient in all primates, known for its roles in wound healing, collagen formation, iron absorption and immune function. Vitamin C deficiency (also known as scurvy) usually takes weeks to become apparent as cutaneous signs and impaired wound healing become more apparent with gingivitis, petechiae, perifollicular haemorrhage and bruising, and occasional systemic manifestations. While haemorrhagic pericarditis has been previously described, it remains a rare complication of vitamin C deficiency and has never been reported in isolation. We report a case of a 56-year-old patient with haemorrhagic pericarditis revealing a vitamin C deficiency.

CASE DESCRIPTION

A 56-year-old woman presented to the emergency department with a 2-week history of worsening chest pain and dyspnoea. She denied any history of other pulmonary or cardiac symptoms or conditions, autoimmune or infectious disease, as well as any recent travel or immobilisation. Furthermore, she denied any fever, cough or haemoptysis, and was not taking any over-the-counter medicine.

The patient was tachycardic at 110 beats per min, tachypneic at 26 breath per min and hypotensive at 95/60 mmHg with no fever. The physical exam showed an elevated jugular venous pressure, muffled heart sounds and multiple petechiae on the lower limb. An ultrasound revealed a large pericardial effusion and an emergency pericardiocentesis was performed, which revealed haemorrhagic fluid with no atypical cells. The red blood cell count was 4,383,000 cells/mm^3, and the white blood cell count was 2,826 cells/mm^3 with lymphocytes predominant. An initial workup including haemoculture, adenosine deaminase level and PCR for Mycobacterium tuberculosis yielded negative results. A whole-body CT scan showed no evidence of tuberculosis or lymphoma. The initial workup was also negative for autoantibodies (including antinuclear and antineutrophil cytoplasmic antibodies), tumour markers and infectious diseases. The complete blood count showed a normal white blood cell count and an elevated C-reactive protein at 23.4 mg/L. The liver, kidney and thyroid tests as well as prothrombin time and partial thromboplastin time were within normal range.

After the initial negative workup, an extensive literature review revealed scurvy as a potential cause for this presentation, given the presence of cutaneous and mucous signs.

Currently, there is no available data on the prevalence of scurvy in the region of Marrakech. Consequently, a systematic vitamin C dosage was not included in the early investigations. Her vitamin C level was found to be <3 umol/L (reference range: 26–85 umol/L). After stabilising, she was started on high-dose vitamin C plus maintenance treatment. The patient was not an alcoholic nor was she on a fad diet. Further assessment revealed no other signs of malabsorption, so we concluded it could be only due to a poor diet in vitamin C. After receiving substitution therapy alone, she continued to improve clinically and remained asymptomatic after her discharge.

DISCUSSION

Scurvy, a disease caused by vitamin C deficiency, has been described as far back as 1500 BC in historic voyagers' reports. Nowadays, reported cases of scurvy are relatively rare, possibly due to easier access to vitamin C sources, but also due the vague symptoms of scurvy which often lead to misdiagnosis.

There are many risk factors for vitamin C deficiency including low socio-economic status, alcoholism, senile age, restrictive eating habits, poor dentition, gastrointestinal diseases and psychiatric disorders[1].

Vitamin C plays a crucial role in the synthesis of collagen fibres, which are essential for connective tissue function and vascular integrity. Without enough vitamin C, the disruption of these functions can lead to various cutaneous and mucous symptoms, as well as vasculitis-like symptoms.

The typical symptoms of scurvy usually include malaise and anorexia in the early stages, followed by mucocutaneous manifestations including gingival bleeding, periodontitis and dental caries, and vasculitis-like symptoms[2]. It can evolve in later stages to more severe and life-threatening conditions such as oedema, neuropathy, convulsions, severe jaundice, haemolysis, acute spontaneous bleeding and even death[3].

Therefore, the diagnosis of scurvy is typically established by interrogating the patient to identify the principal risk factors (as outlined above), evaluating the presence of associated characteristic signs, conducting a thorough clinical examination and in certain cases, assessing the response to vitamin C therapy.

Despite being a rare manifestation of scurvy, a few cases of haemorrhagic pericarditis have been reported in the literature[2,4-6]. However, there is currently a limited amount of information available regarding this condition. To the best of our knowledge, this is the first reported case of an isolated haemorrhagic pericardial effusion in the context of vitamin C deficiency. In four out of five cases, the diagnosis was made in a setting of tamponade. With the exception of the one case in which the diagnosis was made post-mortem, vitamin C supplementation led to a complete recovery.

As with most nutritional deficiencies, treatment for scurvy involves daily supplementation with vitamin C along with the management of the underlying cause of deficiency. We usually prescribe ascorbic acid 1 to 2 g/day for 3 days followed by 500 mg/day for 1 week, followed by 100 mg/day for 1 to 3 months[1].

CONCLUSION

Although rare, when faced with a haemorrhagic pericardial effusion of unexplained cause it is important to look for vitamin C deficiency, especially in patients with risk factors. Our case illustrates the importance of an early

diagnosis of this disease in the rapid management of various complications of scurvy allowing the improvement of the prognosis of a pathology which, undiagnosed, remains potentially fatal.

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