Pericarditis due to Campylobacter fetus subsp fetus – A Case Report of an Uncommon Infection

Marília Andreia Fernandes, Francisco Gonçalves, Lino Gonçalves

1. Department of Internal Medicine, Hospital Curry Cabral, Centro Hospitalar Universitário de Lisboa Central, Lisbon, PRT
2. Cardiac Intensive Care Unit, Department of Cardiology, Coimbra University Hospital Centre, ICBR-Faculty of Medicine, University of Coimbra, Coimbra, PRT

Corresponding author: Marília Andreia Fernandes, mariliandreia@sapo.pt

Abstract

Pericarditis is a common condition with numerous aetiologies. Bacteria other than Mycobacterium tuberculosis complex are an exceptional cause. We present a case of a subacute pericarditis due to Campylobacter fetus subsp fetus in an immunosuppressed patient undergoing biologic therapy in relation with systemic lupus erythematosus (SLE). On admission the patient presented chest pain, dyspnoea and diaphoresis, and lately developed fever and a large pericardial effusion (PE) with concomitant increase in the inflammatory parameters. The clinical presentation along with the exclusion of a flare of the autoimmune disease together with the isolation of Campylobacter fetus subsp fetus on blood samples permitted the diagnosis. After therapy with antibiotics and colchicine the patient showed full recovery.

Categories: Cardiology, Infectious Disease, Internal Medicine
Keywords: pericarditis, pericardial effusion, campylobacter fetus, campylobacter fetus subsp fetus, immunosuppression

Introduction

Pericarditis is the most common disease affecting the pericardium. Infections, autoimmune diseases, malignancy, metabolic, iatrogenic and drug-related conditions may cause the inflammation of the pericardial sac which has unknown aetiology in most cases. In developed countries bacteria are hardly ever implicated as a cause, except in those where tuberculosis has a high prevalence [1]. Campylobacter infections typically involve the gastrointestinal tract. However, invasive disease may sporadically occur, with Campylobacter fetus being the most common implied species (up to 53% of infections). Immunosuppression and regular contact with natural reservoirs are risk factors to bacteraemia due to Campylobacter fetus [2]. We report a case of pericarditis due to Campylobacter fetus subsp fetus, a rare infection with a dozen cases previously described in literature [3].

Case Presentation

Female, aged 50-years-old, living in a rural area, smoker, with arterial hypertension, dyslipidaemia, grade 1 obesity, peripheral arterial disease and systemic lupus erythematosus (SLE) with nephritis, diagnosed 14 years ago and in that context medicated with prednisone 5 mg daily, hydroxychloroquine 400 mg daily and belimumab 10 mg/Kg monthly. Without known allergies nor other relevant medical history, including recent history of infection.

The patient has presented to the emergency department due to oppressive chest pain with several days of evolution and worsening in the previous four hours associated with dyspnoea. At admission her blood pressure was 85/60 mmHg, heart rate 100 beats per minute, temperature 35.4 °C, and oxygen saturation 96% on room air. She was diaphoretic and tachypneic with no other relevant findings on physical examination. The 12-lead electrocardiogram (ECG) showed a diffuse concave ST-segment elevation (Figure 1). The blood analysis denoted an elevation of leucocytes (10.95 x10⁹/L (reference range 4.0-10.0) and C-reactive protein (CRP, 113.1 mg/L, <5.0) without alteration on haemoglobin, platelets, troponin I, creatine kinase (CK), urea and creatinine serum levels. Chest radiograph was also normal. Even so, suspecting an ST-elevation acute coronary syndrome (ACS) in a centre where percutaneous coronary intervention (PCI) is not available and predicting an absolute time from diagnosis to PCI-mediated reperfusion greater than 120 minutes, the patient underwent systemic fibrinolysis with tenecteplase 10,000 units. Afterwards, she was transported by helicopter to the closest centre able to perform an urgent PCI.
Besides minor irregularities in the left anterior descending and circumflex arteries, the coronary angiogram revealed an ostial stenosis of 91-99% in the right coronary artery (dominant). A drug-coated stent was placed in the culprit artery with a good angiographic result. Nonetheless, during the first 48 hours of the in-patient period the patient was still complaining about strong thoracalgia, especially when lying down, and high fever. By that time, inflammatory parameters had increased (leucocytes 12.4 x10^9/L, CRP 241.4 mg/L, and erythrocyte sedimentation rate (ESR) 99 mm/h, 1-20), keeping myocardial injury and renal function markers within normal range. The patient had performed the first transthoracic echocardiogram which revealed a mild PE. These findings corroborated the results of recently made thoracic computed tomography angiography which incidentally showed a spontaneous dense, non-loculated fluid surrounding the left ventricle. After discussion with her rheumatologist, the study was also completed by dosing complement, anti-double stranded DNA (anti-dsDNA), and anti-ribonucleoprotein (anti-RNP), all within the normal range. So, the patient started colchicine 0.5 mg twice daily and increased aspirin to 750 mg every 8 hours, keeping the usual corticosteroid dose, considering disease flare unlikely according to the SLE Disease Activity Index 2000 (score increasing of 3 points compared with the previous assessment and counting with fever, whose infectious aetiology hasn’t been ruled out yet). One week later the echocardiogram pointed out an increased PE with 15 mm, circumferential, and no hemodynamic compromise (Figure 2). Afterwards, serial echocardiograms were performed until documentation of improvement of PE (maximum 23 mm with no signs of cardiac tamponade; at discharge, just a small lamina, mainly posterior). In the meantime, *Campylobacter fetus* subsp *fetus* was isolated in blood cultures and the patient started gentamicin 5 mg/Kg/day and ceftriaxone 2 g/day. After 10 and 15 days, in this order, she stopped the aforementioned antibiotics, given the good clinical and laboratorial evolution with demonstration of clear blood cultures. The patient was discharged after 26 days of hospitalisation with indication to maintain colchicine and avoid vigorous physical activity for three months. Three months after leaving the hospital, the patient was asymptomatic, with echocardiographic evidence of total resolution of PE.

**Discussion**

Acute pericarditis (AP) usually manifests as a sharp and pleuritic chest pain, which is relieved by sitting upright and leaning forward, and a widespread ST elevation (hallmark sign) or PR depression on ECG. Pericardial rub may be audible and a new PE may occur in up to one-third and 60% of patients, respectively. Elevation of inflammation markers as well as the imaging evidence of pericardial inflammation support the

---

**FIGURE 1:** The 12-lead electrocardiogram performed at admission showing a concave ST-segment elevation in leads II, III, aVF, and V2-V4.

**FIGURE 2:** Transthoracic echocardiogram demonstrates a moderate circumferential PE (asterisks), measuring 15 mm.

Panel a, parasternal short axis; panel b, apical 4-chamber view; panel c, subcostal view. LV, left ventricle; RV, right ventricle; LA, left atrium; RA, right atrium; IVS, interventricular septum.
diagnosis of AP [1]. AP imposes differential diagnosis with ACS [1], which may not be straightforward in patients who have multiple cardiovascular risk factors and present the so-called angina equivalents [4] namely dyspnoea and diaphoresis, as in this case. Indeed, some hospitals do not have the availability to perform urgent PCI (like the one where the patient had been initially admitted), which diminishes the time to make a decision and meet the recommended time to implement a reperfusion strategy.

Since the diagnosis of AP is established, the presence of high-risk features should be looked for, as the presence of any determines the need for hospitalisation. Aetiologic study is mandatory in those cases [1]. Viral infections are the most common cause of AP [1] counting for around 90% of cases together with those which have unexplained aetiology [5]. Systemic autoimmune diseases could be responsible for 15% of acute or recurrent pericarditis [1]. In fact, serositis is a usual manifestation of extra renal flare of SLE [6] and pericarditis could reach up to 54% of the patients [7]. Among others, nortropic syndrome with renal insufficiency, interstitial lung disease, pulmonary hypertension and cardiomyopathy have been associated with pericarditis in patients with SLE, but none of these features were evident in our case. Furthermore, and excluding fever and high ESR (that in fact could also be attributable to other causes), the absence of clinical and serological signs of disease flare (namely, haemolytic anaemia, thrombocytopenia, elevation of anti-dsDNA and/or anti-RNP, and low complement) [6,7] makes lupus PE less probable. On the other hand, immunosuppressive treatment related to this disease may make the patient prone to opportunistic microorganisms [1], like Campylobacter fetus subsp fetus [2,5] lately identified. Of note, despite the fact that bacterial AP is fairly uncommon, tuberculosis is the most frequent form of pericardial disease in developing countries, and the most prevalent cause of AP in developed countries with a high prevalence of this infection [1] such as Portugal [8]. Actually, the suspicion of purulent pericarditis should promptly motivate the performance of a pericardiocentesis, which permits the drainage essential for the treatment, and the biochemical and cultural analysis of pericardial fluid samples collected. Nonetheless, this procedure wasn't performed, considering the lower estimated content of pericardial fluid in a patient under dual antiplatelet therapy. Indeed, non-steroidal anti-inflammatory drugs (NSAIDs) are used as first line treatment for AP with ibuprofen being the drug of choice, unless ischemic heart disease or other indication for antiplatelet treatment is present when aspirin ranks first. This class of drugs should be kept accordingly until evidence of an inactive disease, determined by full symptom remission and CRP normalisation. In turn, colchicine, which is an adjuvant drug that avoids recurrence, should be maintained for three months. Corticosteroids should be just used as an alternative option in patients with contraindications or failure of NSAIDs or when a systemic disease is present, as they are linked to a chronic evolution and recurrence of disease. Moreover, specific therapy to the underlying cause is indicated. Regarding non-pharmacological measures, physical activity should be restricted to less than usual until disease remission in non-athletes [1].

AP does usually have a benign course. However, there are some factors which predict an ominous prognosis. Subacute onset, high fever, large PE, failure to respond within seven days to NSAIDs, and immunosuppression, all present in the case reported, are related to a poorer prognosis. Additionally, bacterial pericarditis has an increased risk of cardiac tamponade, recurrence and constriction, which confer a greater mortality and morbidity [1].

Conclusions
Pericarditis is a clinical diagnosis supported by laboratory and imaging data. Personal history may drive to the most likely aetiology, which is useful to search for as it influences the accurate management. Immunosuppressive conditions should lower the threshold of suspicion for less common infections, especially when they impose a non-neglected risk of mortality and/or morbidity.

Additional Information
Author Contributions
All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Marília Andreia Fernandes, Francisco Gonçalves, Lino Gonçalves

Acquisition, analysis, or interpretation of data: Marília Andreia Fernandes

Drafting of the manuscript: Marília Andreia Fernandes

Critical review of the manuscript for important intellectual content: Francisco Gonçalves, Lino Gonçalves

Disclosures
Human subjects: Consent was obtained or waived by all participants in this study. Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: Payment/services info: All authors have declared that no financial support was received from any organization for the
Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. Other relationships: All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

Acknowledgements

Authors’ contributions: MAF wrote the initial draft and most significant edits of the manuscript. FG and LG reviewed it. All authors read and approved the final submission. Acknowledgements: The authors would like to thank all colleagues at Unidade de Cuidados Intensivos Cardiacos, Hospital da Universidade de Coimbra, who actively participated in the management of the patient.

References