

# Revista Portuguesa de **Cardiologia**Portuguese Journal of **Cardiology**

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#### CASE REPORT

### Purulent pericarditis: A rare diagnosis\*



Luís Ferreira dos Santos<sup>a,\*</sup>, Davide Moreira<sup>a</sup>, Pedro Ribeiro<sup>b</sup>, Bruno Rodrigues<sup>a</sup>, Emanuel Correia<sup>a</sup>, Luís Nunes<sup>a</sup>, Miguel Sequeira<sup>b</sup>, Ana Albuquerque<sup>b</sup>, Inês Barros<sup>b</sup>, José Pedro Saraiva<sup>b</sup>, Oliveira Santos<sup>a</sup>

Received 25 July 2011; accepted 11 April 2013 Available online 23 November 2013

#### **KEYWORDS**

Bacterial pericarditis; Purulent pericarditis; Pericardial effusion; Tamponade; Pneumonia **Abstract** The authors present two cases of purulent pericarditis secondary to pneumococcus pneumonia, a rare entity in the antibiotic era, one of them in an apparently healthy person. A systematized diagnostic approach to moderate pericardial effusion is presented, together with a review of purulent pericarditis. The presence of pericardial effusion with persistent fever with or without known etiology, particularly in the immunocompromised but also in the apparently healthy patient, should always raise the possibility of purulent pericarditis.

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#### PALAVRAS-CHAVE

Pericardite bacteriana; Pericardite purulenta; Derrame pericárdico; Tamponamento; Pneumonia

#### Pericardite purulenta: um diagnóstico raro

Resumo Os autores apresentam dois casos de pericardite purulenta secundária a pneumonia por *pneumococos*, um deles num doente sem antecedentes patológicos conhecidos. É feita uma sistematização da abordagem diagnóstica ao derrame pericárdico de moderadas dimensões e uma revisão da pericardite purulenta, uma entidade muito rara na era da antibioterapia. A constatação de derrame pericárdico com quadro de febre persistente, com ou sem origem conhecida, fundamentalmente no doente com compromisso imune, mas também no aparentemente saudável, deve levantar-se sempre a possibilidade de pericardite purulenta.

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E-mail address: luisferreirasantos@gmail.com (L. Ferreira dos Santos).

<sup>&</sup>lt;sup>a</sup> Serviço de Cardiologia, Centro Hospital Tondela-Viseu, Viseu, Portugal

<sup>&</sup>lt;sup>b</sup> Unidade de Cuidados Intensivos Polivalente, Centro Hospital-Tondela, Viseu, Portugal

<sup>\*</sup> Please cite this article as: Ferreira dos Santos L, Moreira D, Ribeiro P, et al. Pericardite purulenta: um diagnóstico raro. Rev Port Cardiol. 2013;32:721–727.

<sup>\*</sup> Corresponding author.

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#### Introduction

Pericardial disease mainly manifests as pericarditis and/or pericardial effusion (PE) and is caused by a variety of etiologies, including infection, inflammation and neoplasms, or iatrogenic, traumatic or metabolic origin, or unknown (idiopathic).

It may occur in isolation, or be one component, of lesser or greater clinical importance, in the presentation of entities with systemic involvement.<sup>1</sup>

#### Case reports

#### Case 1

A 39-year-old woman, with no history of disease and not taking regular medication, came to the emergency department for right lower back pain and worsening dyspnea on moderate exertion of one week's duration. She reported no fever, cough or expectoration, and was normotensive, with tachyarrhythmia and mild hypoxemic respiratory failure. Laboratory tests showed signs of inflammation and infection, the ECG revealed atrial fibrillation with rapid ventricular rate ( $\approx$ 115 bpm), and the chest X-ray showed increased cardiothoracic index and segmental infiltrate in the mid third of the right hemithorax (Figure 1).

The patient was admitted with a diagnosis of community-acquired pneumonia, and empirical antibiotic therapy was begun with ceftriaxone and azithromycin. Thoracic computed tomography (CT) on the second day of hospitalization showed bilateral lung condensations, bilateral pleural effusion and PE, which on transthoracic echocardiography (TTE) measured 10 mm at the posterior wall in diastole, with no signs of cardiac tamponade. On the same day, there was a sudden deterioration of the clinical setting, evolving to septic shock, and the patient was transferred to the intensive care unit. Inotropic support was required for three days, but chemical cardioversion to sinus rhythm was achieved on the second day. On the third day, multisusceptible *Streptococcus pneumoniae* was identified in blood samples collected at



**Figure 1** Chest X-ray at admission.



Figure 2 Chest X-ray on the 10th day.

admission and therapy was begun with penicillin G. Respiratory and hemodynamic stabilization was achieved from the fourth day, although the patient still required mechanical ventilation and remained febrile, with no resolution of the infection on laboratory tests. The cardiac silhouette was still enlarged and serial TTE showed progressive worsening of the PE, which measured 20 mm at the posterior wall in diastole on the 10th day, with signs of cardiac tamponade (Figures 2 and 3A). Pericardiocentesis was therefore performed for diagnostic purposes and drainage, 800 cc of fibrinous purulent fluid being drained (Figure 3B), which was negative on bacteriological study, both direct and in culture. Screening for acid-alcohol resistant bacilli (AARB) was also negative. Subsequent TTE revealed resolution of the effusion, accompanied by clinical, radiological and laboratory improvement (Figure 4). Screening for immune deficiency diseases was negative. The patient presented no constrictive pattern on TTE at 12-month follow-up.

#### Case 2

A 33-year-old man had a history of congenital hypogammaglobulinemia (diagnosed in 2001), chronic alcoholism and recurrent pneumonia, the last episode (in 2005) having been complicated by pleural effusion requiring decortication. He had taken no medication since 2005. He came to the emergency department for fever and worsening dyspnea over the previous five days. At admission, he was febrile, prostrate, tachycardic and hypotensive. Laboratory tests showed marked elevation of inflammatory and infectious parameters and arterial blood gas analysis revealed hypoxemic respiratory failure. A chest X-ray showed a markedly enlarged cardiac silhouette, with no parenchymal lung lesions; TTE was therefore performed, which confirmed the presence of a PE, measuring 23 mm at the posterior wall in diastole, with abnormal motion and diastolic collapse of the right atrial free wall (Figure 5A and B).

In view of the setting of septic/obstructive shock, the patient was transferred to the intensive care unit,

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