



Case Report

A pneumococcal purulent pericarditis revealing a pneumonia and complicated by an acute cardiac tamponade

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KEYWORDS

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Summary Purulent pericarditis secondary to pneumococcal pneumonia is a rare entity, and often underestimated despite being associated with a high mortality rate. We report a case of a man who developed a cardiac tamponade related to a previously unknown pneumococcal pneumonia. In conclusion, we emphasize the need of repeated clinical and echocardiographic exams.

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Introduction

Streptococcus pneumoniae is the most common bacterium responsible for community-acquired pneumonia in immunocompetent individuals. Since the widespread use of antibiotics, complications secondary to invasive *S. pneumoniae* infections such as purulent pericarditis have become rare [1–3]. Purulent pericarditis occurs chiefly in patients who have other co-morbidities such as diabetes, immunosuppression, previous cardiac surgery, or chronic alcoholism [4]. Specific serotypes associated with a high level of antibiotic resistance may be selectively involved in purulent pericarditis [5].

The diagnosis of purulent pericarditis is challenging, because of both the low incidence of this event and the nonspecific presenting symptoms [1,4–7]. Nevertheless, achieving the diagnosis is crucial, as mortality remains high and surgical drainage is often required [8]. We report the case of a middle-aged man who had purulent pericarditis which revealed pneumococcal pneumonia.

Case report

A 45-year-old man from Senegal presented at the emergency department of our hospital for chest pain. He had heterozygous sickle-cell disease (A/S, 65%/35%) and had been living in France for 2 years without returning to Africa.

His body temperature was normal but he reported chills 24h earlier. He reported no cough, headache, or other symptoms consistent with a viral illness. His chest pain was very severe in all positions and increased during

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inspiration. His systolic blood pressure was 120 mmHg and his heart rate 90/min. No heart murmurs or pericardial rub were heard. There was no polypnea, neck vein congestion, or hepatomegaly. Crackles were heard over the lung bases, being more intense on the right. The only abnormal electrocardiographic finding was PQ segment depression seen in lead II (Fig. 1A).

The chest radiograph showed a normal-sized cardiac shadow and moderate bilateral opacities in the lung fields. Computed tomography of the chest did not visualize any evidence of pulmonary embolism or aortic dissection; ground-glass opacities and alveolar condensations were seen in the two lower lung lobes.

Laboratory test results were as follows: C-reactive protein (CRP), 118 mg/L; leukocyte count, 8600/mm³;

procalcitonin, 0.41 ng/mL; creatine phosphokinase, 584 U/L (*N*, 20–215); troponin Ic, normal; creatinine, 142 μmol/L (*N*, 50–90); and DDimers, 1572 ng/mL (*N*, <500).

He was given a diagnosis of pneumonia and a prescription for clarithromycin and returned to his home. Just before he left, an echocardiogram was performed to look for a pericardial reaction. The only abnormality was a 2-mm pericardial effusion beside the left chambers associated with a pericardial thickening.

The following day, he came back to the hospital because of persistent chest pain. He still had no fever but his CRP level was 309 mg/L, his procalcitonin level was 4.5 ng/ml, and his leukocyte count was 23,700/mm³. A repeat electrocardiogram showed ST elevation in all the leads (Fig. 1B). A fever of 39 °C developed.

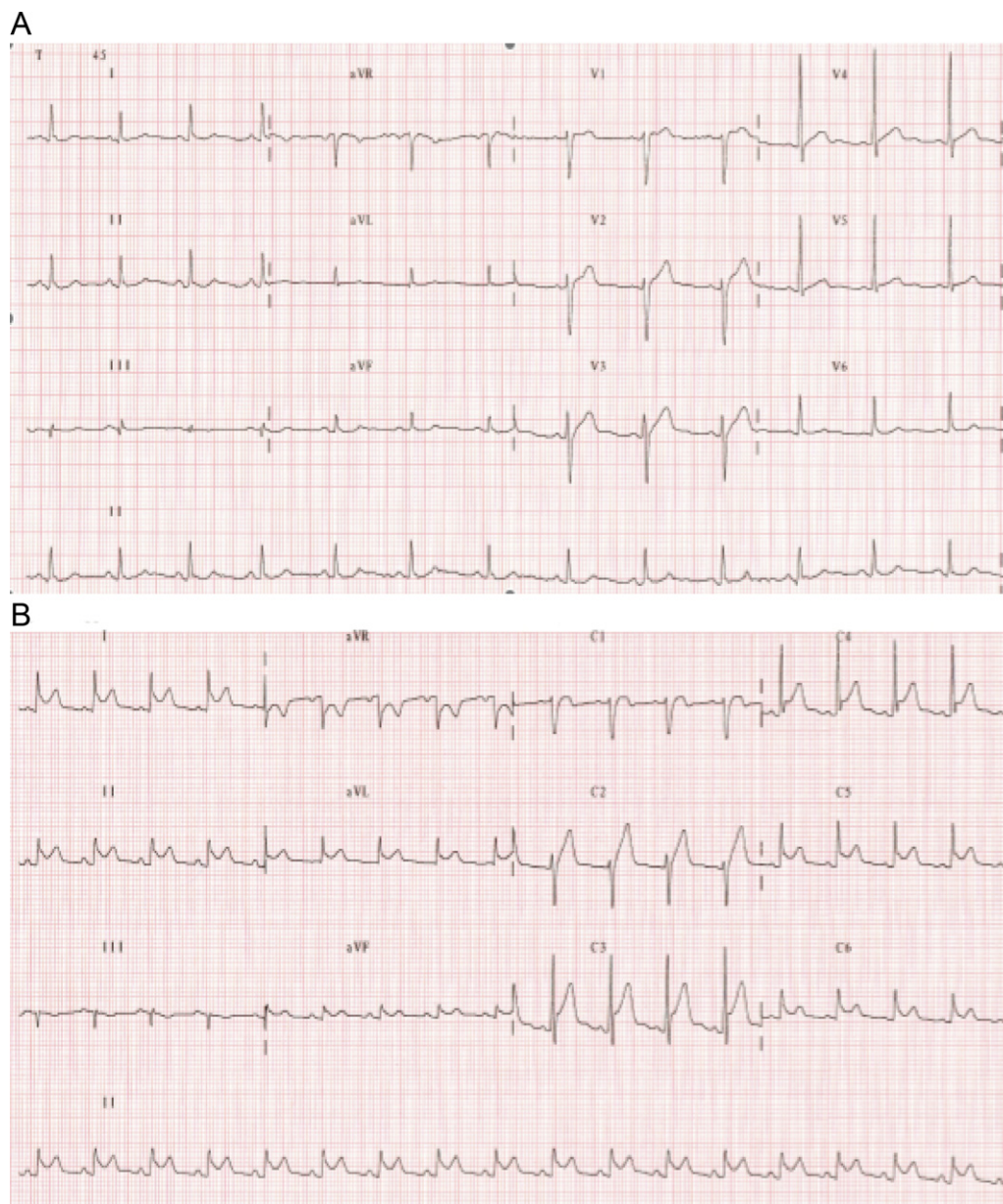


Figure 1 (A) First electrocardiogram (ECG) at time of emergency department consultation, PQ segment depression seen in the D2 lead without other abnormality. (B) ECG during the second consultation on day 2 with wide ST segment elevation.

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