

CASE REPORT

Purulent pneumococcal pericarditis, a vaccine-preventable illness

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Abstract

A 74-year-old gentleman presented to hospital with a 1-day history of acute onset pleuritic chest pain and fever. He was found to have widespread ST segment elevation on electrocardiogram, and blood cultures taken were positive for *Streptococcus pneumoniae*. Two days following admission the patient developed dyspnea, and a large pericardial effusion and right middle lobe consolidation were demonstrated on a computed tomography scan of the chest. A transthoracic echocardiogram confirmed the presence of a large circumferential pericardial effusion with multiple prominent adhesions and marked heterogenous thickening of the pericardium, without evidence of tamponade. Pericardiocentesis drained a purulent exudate positive for pneumococcal antigen. The occurrence of purulent pericarditis secondary to pneumococcal community-acquired pneumonia is rare in the modern antibiotic era and represents an often-lethal manifestation of invasive pneumococcal disease (IPD). IPD is a vaccine-preventable illness for which adult vaccination rates are low despite high morbidity. Healthcare professionals need to vaccinate older patients opportunistically.

INTRODUCTION

Purulent pneumococcal pericarditis is a rare but often fatal manifestation of invasive pneumococcal disease (IPD). The 23-valent pneumococcal polysaccharide vaccine (PPV) is available free of cost on national immunization programs in Australia and the United Kingdom and is effective at reducing IPD. However, there is widespread under-vaccination of adults, and clinicians need to opportunistically vaccinate older patients at risk.

CASE REPORT

A 74-year-old gentleman with a 15-pack-year smoking history presented to a rural hospital with a 1-day history of acute onset left-sided pleuritic chest pain, fever and wide-spread ST eleva-

tion on electrocardiogram, Fig. 1. The patient had not received pneumococcal or influenza vaccinations. The patient was tachycardic at presentation with a heart rate of 103 bpm, a blood pressure of 103/75 mmHg and tympanic temperature of 38.6°C. Clinical examination did not identify any murmurs, friction rub, signs of fluid overload or jugular venous distension. Initial investigation revealed an elevated white cell count ($13.6 \times 10^9/l$) and a markedly elevated C-reactive protein (366 mg/l). High-sensitivity troponin, HIV testing and chest radiograph were normal at presentation.

Blood cultures were taken, and empirical antibiotic therapy for community-acquired pneumonia (CAP) commenced with ampicillin, in addition to prednisolone 25 mg daily for a presumed exacerbation of chronic obstructive airways disease. The patient continued to experience fevers following admission and subsequently had four of four blood culture sets isolate

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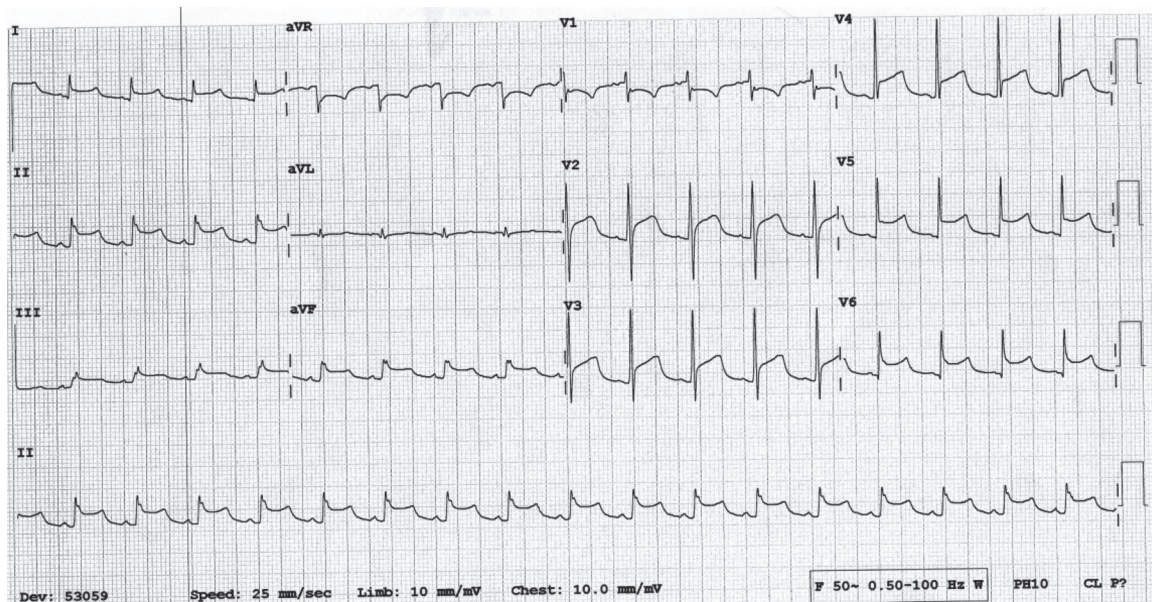


Figure 1: Twelve-lead electrocardiogram at presentation demonstrating sinus rhythm associated with widespread concave ST-segment elevation, 'PR interval elevation in lead aVR and PR depression in lead II'.

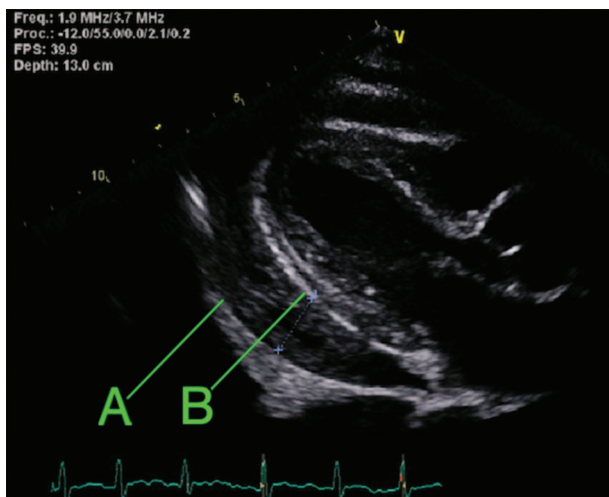


Figure 2: Parasternal transthoracic echocardiogram demonstrating a heterogeneous pericardial effusion and thickening of the parietal (A) and visceral pericardium (B).

Streptococcus pneumoniae (pneumococcus) sensitive to penicillin at <1-day incubation.

A computed tomography (CT) chest performed on Day 2 of admission to evaluate progressive dyspnea detected a significant pericardial effusion (width 27 mm) and bilateral pleural effusions with consolidation in the right middle lobe. The patient was subsequently transferred to a tertiary referral hospital for further management, and a transthoracic echocardiogram at this time confirmed the presence of a large circumferential pericardial effusion (width 22 mm) with multiple prominent adhesions and marked thickening of the parietal and visceral pericardium, Fig. 2. There was no echocardiographic evidence of tamponade. Pericardiocentesis was performed, and a pericardial drain placed; 900 ml of purulent pericardial fluid was drained, Fig. 3. Subsequent analysis of the pericardial

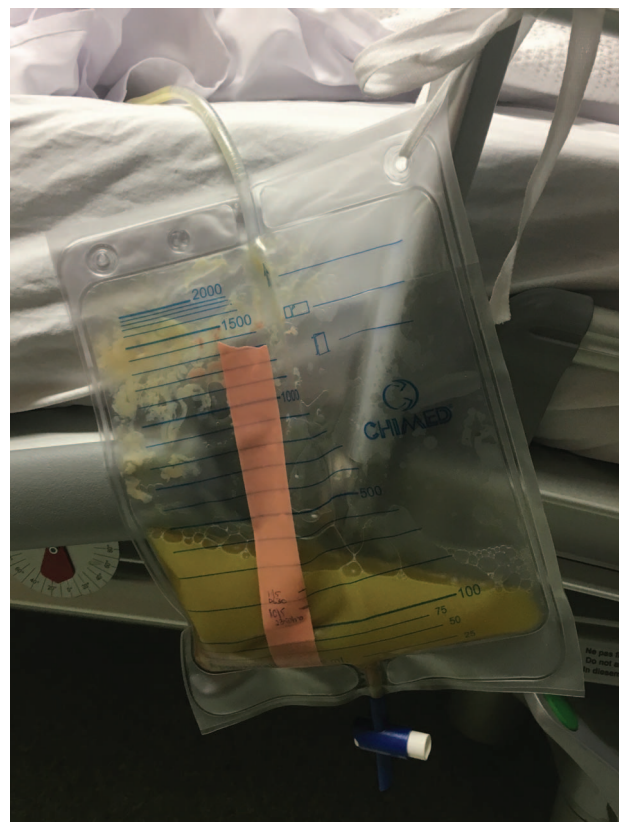


Figure 3: 300 ml of frank pus drained from a subxiphoid approach.

fluid identified the presence of pneumococcal antigen, innumerable leukocytes, markedly elevated lactate dehydrogenase (LDH) (>200 000 U/l) and elevated protein (56 g/l); no organism was isolated on culture of the pericardial fluid. After drainage, the patient was treated with ceftriaxone and moxifloxacin.

Following management of his pericarditis, the patient was investigated for lymphadenopathy and splenomegaly noted incidentally on CT of his chest. Core biopsy of an abdominal lymph node led to a diagnosis of mantle cell lymphoma. During his diagnostic work-up and prior to the commencement of chemotherapy, the patient had recurrent dyspnea with large bilateral pleural effusions. Follow-up echocardiogram demonstrated constrictive pericarditis with ventricular interdependence and segmental wall abnormalities secondary to tethering of the myocardium. The patient subsequently experienced a precipitous decline with profound hypoxia and died suddenly in hospital secondary to a respiratory arrest.

DISCUSSION

We report a case of purulent pericarditis secondary to *S. pneumoniae*, an often-fatal manifestation of IPD, rare in the modern antibiotic era and preventable with appropriate vaccination. This case highlights a serious albeit infrequent consequence of low vaccination coverage in adults and the need for clinicians caring for older adults to opportunistically vaccinate those at risk of IPD.

Recognized since the time of Galen, purulent pericarditis was a frequent complication of pneumococcal pneumonia before the advent of antibiotics [1]. In addition to direct spread from a pulmonary source, bacterial pericarditis may also develop from the extension of a subdiaphragmatic abscess or haematogenous seeding [2]. Risk factors include a pre-existing pericardial effusion, immunosuppression, poorly controlled diabetes, alcoholism and cardiac surgery [3]. *Staphylococcus aureus* is the most common microbiological cause overall, accounting for ~22% of cases, while *S. pneumoniae* is the most common cause from an intrathoracic focus [2].

Clinically, purulent pericarditis is characterized by a fulminant course, with a mean time to hospitalization of 3 days and a mortality rate from 42% to 77% [1, 4]. Other clinical findings include the presence of fever (96%) and cardiorespiratory symptoms, dyspnea (54%), cough (42%) and chest pain (27%). A pericardial friction rub is present in around half of patients [1]. Laboratory and imaging studies are nonspecific, and while echocardiography is useful in detecting the presence of tamponade, it cannot reliably predict the cause of exudative effusions (bacterial, inflammatory and malignant). Pericardiocentesis is the essential diagnostic test, with pericardial fluid exhibiting high protein, low glucose (<2 mmol/l) and high leukocyte count.

Effective treatment relies on timely drainage and appropriate antimicrobial therapy. Drainage may be achieved via pericardiocentesis, which is fast and straightforward but less useful for loculated effusions, or via subxiphoid pericardiotomy, which achieves more complete drainage and has lower rates of subsequent constrictive pericarditis [3].

The incidence of IPD is bimodal, primarily occurring in adults >65 years and children <2 years of age. Pneumococcus remains one of the most commonly identified causes of CAP in older adults; however, its incidence is declining partly in response to the herd effect of childhood pneumococcal vaccine programs [5–7].

The polysaccharide capsule of *S. pneumoniae* determines both its virulence and serotype, and capsular polysaccharides are key to all available pneumococcal vaccines. The 23-PPV vaccinates against 23 of the 90 distinct pneumococcal serotypes that exist. A recent meta-analysis on the 23-PPV in adults ≥ 60 years demonstrated vaccine effectiveness of 45–73% against IPD and

48–64% against pneumococcal pneumonia [8]. In the United Kingdom and Australia, the 23-PPV is provided free of cost on national immunization programs for all adults aged ≥65 years and for younger patients with risk factors for IPD. Despite this, there is widespread under-vaccination of adults; in England, 30.5% of adults aged ≥65 years have not been vaccinated with the 23-PPV vaccine [9]. Similarly in Australia, of the 3.5 million adults aged ≥65 years, only 51% have received both influenza and pneumococcal vaccines for which they were eligible [10], compared to a 93% pneumococcal vaccination rate in children. This discrepancy underscores the need for health providers to opportunistically vaccinate older patients at risk of IPD with the same resolve that childhood immunization is taken. Health professional recommendation is the most important factor influencing vaccination uptake in older patients [10].

CONFLICT OF INTEREST STATEMENT

No conflicts of interest.

FUNDING

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ETHICAL APPROVAL

No specific ethical approval was required.

CONSENT

Written informed consent was obtained from the patient for the submission of this manuscript.

GUARANTOR

Matthew James Rees is the guarantor of this article.

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