S pneumoniae purulent pericarditis in the setting of community-acquired pneumonia

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A 47-year-old Tongan man presented with four days of fatigue, pleurisy and dyspnoea. Clinically, he was normotensive with no jugular venous distension, Kussmaul’s sign or pulsus paradoxus, but was febrile (39°C Celsius) with a raised white cell count 20x10⁹/L (normal 4–11) and CRP 500mg/L (normal <3). Electrocardiogram (ECG) demonstrated atrial fibrillation and widespread ST elevation (Figure 1A). Chest x-ray (CXR) showed a widened mediastinum (Figure 1B). Echocardiogram showed a 3cm pericardial effusion with echocardiographic signs of tamponade (Figures 1C and 1D). 700ml of frank pus was drained sub-costally (Figure 2). Blood cultures and aspirate grew Streptococcus pneumoniae sensitive to penicillin. Despite treatment, he had ongoing fever and re-accumulation of pericardial effusion found on transoesophageal echocardiogram. He underwent surgical subxiphoid pericardial washout with 1.8L pus drained; pericardial drain was left in-situ for 14 days with further 800ml drained (Figures 3A and 3B). A follow-up CXR five days after admission revealed left lower lobe consolidation. It was postulated that the pericardial effusion was a complication of pneumonia. Fourteen days after presentation he was febrile again. CXR showed a loculated left pleural effusion requiring drainage—500ml was drained (no growth). He completed 28 days IV benzyl-penicillin and was well at discharge.

Streptococcus pneumoniae is the most common cause of community-acquired pneumonia (CAP) in New Zealand, being responsible for 20–40% of cases.¹² Pacific peoples are at increased risk from CAP, compared to European.³ Age standardised rates of invasive pneumococcal disease (IPD) in Māori and Pacific peoples are consistently three times higher than that of Europeans.⁴ In the modern era, purulent pericarditis is uncommon, but high mortality persists (40%) even with early diagnosis and prompt treatment.⁵ Death is most often from cardiac tamponade, with or without septic shock and constrictive pericarditis.⁶ In the pre-antibiotic era, direct spread from an intrathoracic focus was the most common cause; however, in modern practice, only 22% of patients with purulent pericarditis had a recognised primary source.⁷ Penicillin remains the drug of choice for invasive pneumococcal infections in New Zealand, as 98.1% of isolates remain penicillin sensitive.⁷
**Figure 1A:** Electrocardiogram (ECG) showed atrial fibrillation with widespread ST elevation in most leads.

![Electrocardiogram (ECG)](image)

**Figure 1B:** Chest x-ray (postero-anterior [PA] view) showed enlarged heart and widened mediastinum.

![Chest x-ray (postero-anterior [PA] view)](image)
**Figure 1C:** Transthoracic echocardiogram apical four-chamber view showing 2.5cm collection lateral to the left ventricle (red arrow).

**Figure 1D:** Transthoracic echocardiogram Doppler across mitral valve showing marked respiratory variation in mitral E velocity, suggesting early haemodynamic compromise.
Figure 2: 700ml frank pus drained subcostally on day of admission.
**Figure 3A:** Subxiphoid incision and pericardial window with pus freely draining (black arrow).

**Figure 3B:** 1,800ml of frank pus drained with free suction at time of pericardial window.
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Nil.

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