CASE REPORT

Purulent pericarditis as a complication of bacteraemic *Enterococcus faecalis* urinary tract infection

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SUMMARY

Purulent pericarditis is a rare clinical entity in the modern antibiotic era. The most common portal of entry is thought to be direct extension from a primary lung source and is usually caused by *Staphylococcus aureus*, *Streptococcus pneumoniae* or *Haemophilus influenzae*. We report the case of a man aged 69 years who presented with purulent pericarditis due to *Enterococcus faecalis* likely caused by haematogenous spread from a urinary tract source. Urgent pericardiocentesis was vital and restored his haemodynamic stability. He was treated for a total duration of 4 weeks with susceptible antibiotics. Echocardiography 3 weeks later showed persistent resolution of the pericardial effusion. This case shows that prompt diagnosis and drainage of the pericardial effusion are vital to achieve a positive outcome in purulent pericarditis. To the best of our knowledge, this is the first reported case of purulent pericarditis caused by *E. faecalis* from a urinary tract source.

BACKGROUND

Infectious pericarditis is caused by a wide range of organisms, with most cases due to possible or confirmed viral infection.¹ Purulent pericarditis, defined as an infection localised in the pericardial space producing purulent fluid, is a rare clinical entity in the modern antibiotic era. Mortality rates are estimated at 30–50% despite treatment, mostly due to cardiac tamponade, especially when the diagnosis is delayed.² Risk factors include immunosuppression, previous pericardial effusion, diabetes, uraemia, cardiac surgery and chest wall trauma.³

CASE PRESENTATION

A man aged 69 years with a medical history significant for type 2 diabetes mellitus, coronary artery disease and ischaemic cardiomyopathy presented with symptoms of fatigue and chills of several days duration. Review of systems was positive for dysuria. He denied chest pain or shortness of breath. Medications included lisinopril, aspirin, metoprolol, atorvastatin and metformin. On evaluation, he had a temperature of 99°F, a pulse rate of 96 bpm and a blood pressure of 124/97 mm Hg. Physical examination revealed lungs clear to auscultation, no jugular venous distention and no murmurs or rubs on cardiac auscultation. Laboratory evaluation was significant for leucocytosis at 17 400 cells/ μ L with a left shift and acute kidney injury with a creatinine of 1.95 mg/dL. Urine analysis was consistent with infection. Urine and blood cultures grew *Enterococcus faecalis* susceptible to ampicillin and his initial broad antibiotic coverage was narrowed to intravenous ampicillin. Given his bacteraemia, a transthoracic echocardiogram was obtained which showed no evidence of endocarditis. Repeat blood cultures and urine cultures 48 hours later revealed no growth.

The patient did reasonably well over the next few days with improved leucocytosis and kidney function. However, he progressively deteriorated afterwards. He became disoriented and hypotensive with increased leucocytosis and worsening kidney function necessitating haemodialysis and vasopressor support. The antibiotic coverage was broadened to piperacillin–tazobactam and vancomycin and investigations ensued for another source of infection.

Figure 1 Transoesophageal echocardiography revealing moderate pericardial effusion (arrows).

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INVESTIGATIONS

Repeat blood cultures remained negative. Given the patient's altered mentation, CT scan of the head, neck and sinuses was performed which was negative for a source of infection and CT scan of the abdomen was unremarkable. ECG showed atrial fibrillation with no signs of acute ischaemia. Troponin I was elevated up to 1.39 ng/mL. A repeat transoesophageal echocardiogram to rule out endocarditis revealed a moderate pericardial effusion measuring 1.2 cm associated with significant mitral valve inflow variability with no signs of tamponade and no vegetation (figure 1). CT scan of the chest confirmed a 1.8 cm pericardial effusion (figure 2).

TREATMENT

Emergent pericardiectomy was performed within 24 hours and 500 mL of purulent material was drained. Interestingly, tamponade parameters were evident only on the third echocardiogram obtained during pericardiectomy. Gram stain revealed Gram-positive cocci and culture grew *E. faecalis* with the same sensitivities as the urinary organism.

OUTCOME AND FOLLOW-UP

The patient experienced rapid clinical improvement with cleared mentation, improvement of renal function and leucocytosis. Troponin I levels trended back to normal.

The patient was discharged on parenteral antibiotics for a total duration of 4 weeks. Repeat transthoracic echocardiography 3 weeks later showed persistent resolution of the pericardial fluid.

DISCUSSION

Since the presenting symptoms are often vague in purulent pericarditis, a high degree of clinical suspicion is necessary. Often, the classic signs of pericarditis may be absent.⁴ In a septic patient with an identifiable source and organism on appropriate antibiotics, as in the present case, prompt diagnosis of purulent pericarditis is particularly challenging.⁵ The differential diagnosis for pericardial effusion in our patient included uraemia, myocardial infarction and infection. The diagnosis could only be confirmed by examination of the pericardial fluid.

Our patient experienced worsening infection with multiple organ failure despite broad-spectrum antibiotic therapy. Detection of the pericardial fluid with emergent drainage was vital and led to rapid clinical improvement.

Purulent pericarditis is most often caused by *Staphylococcus aureus* and *Streptococcus pneumoniae*.⁶ *Enterococcus faecalis* causing purulent pericarditis has been rarely reported in the

literature. The most common portal for infection of the pericardial space is thought to be direct extension from a primary lung source. Other modes of infection include local extension from the myocardium itself and perforating injury to the chest wall. Haematogenous dissemination is rare.⁷ In our case, the most probable source is urinary with haematogenous spread to the pericardial space.

The above patient demonstrated signs of tamponade with significant variation of the mitral and tricuspid inflow velocities and invagination of the left atrium on the third echocardiogram. Purulent pericarditis results in cardiac tamponade in 80% of cases and requires emergent treatment.

Management of bacterial pericarditis includes aggressive antibiotic therapy initially targeting Gram-negative and Gram-positive species along with surgical drainage of the pericardial fluid followed by culture-guided antibiotics for 4–6 weeks. The European Society of Cardiology recommends obtaining Gram stain, acid-fast smear, fungal smear, pericardial culture and body fluid cultures of the drained pericardial fluid.⁸

This case is interesting in terms of the microorganism involved and its unusual presentation. To the best of our knowledge, this is the first reported case of *E. faecalis* purulent pericarditis from a urinary source. The patient's only risk factor was diabetes which may have resulted in immunosuppression. Prompt diagnosis and management of purulent pericarditis with associated tamponade which resulted in a favourable outcome. Therefore, purulent pericarditis should be considered and investigated early in patients with otherwise unexplained clinical deterioration, despite appropriate antibiotics.

Learning points

- ▶ Purulent pericarditis should be considered in patients who continue to deteriorate, despite appropriate conventional management.
- ▶ Emergent pericardiectomy and drainage of the pericardial fluid is vital in purulent pericarditis with signs of tamponade.
- ▶ Despite appropriate therapy, mortality remains high partly due to a delay in diagnosis.

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Figure 2 Axial CT of the chest revealing a 1.8 cm pericardial effusion (arrows).

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