Acute Pharyngitis with Synovial and Extra-Synovial Complications

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Abstract

Suppurative pericardial effusion (SPE) is characterized by infection of the pericardium that produces a purulent effusion that is either seen macroscopically or on tissue microscopy. Bacterial pericardial infections are rare but typically manifest as a serious febrile disease with tachycardia, hypotension and altered mental state. Prompt diagnosis is essential as SPE will inevitably proceed to pericardial constriction and cardiac tamponade without surgical and antimicrobial therapy. This article describes a rare case of SPE with \textit{Streptococcus pyogenes} in a 47-year-old man who presented with acute pharyngitis with synovial and extra-synovial complications.

\textbf{Keywords:} Suppurative Pericardial Effusion; Cardiac Tamponade; Group A Streptococcus; \textit{Streptococcus pyogenes}; Constrictive Pericarditis; Pericardiocentesis; Pericardiectomy; Echocardiography; Cardiothoracic Surgery; Intensive Care Unit; Septic Shock; Invasive haematological infection

Abbreviations

SPE: Suppurative Pericardial Effusion; GAS: Group A \textit{Streptococcus}; MAP: Mean Arterial Pressure

Learning Objective

Early diagnosis of SPE is challenging in patients due to its non-specific presentation and often occurring in the setting of concurrent systemic disease. However it is critical that we consider it as a differential cause of persistent septic shock in patients who do not respond to initial therapy due to its high associated mortality.

Introduction

\textit{Streptococcus pyogenes} is a \(\beta\)-haemolytic bacterium that commonly causes a wide range of clinical diseases in humans. Whilst it is well known for causing both superficial and invasive disease, it has rarely been isolated from suppurative pericardial effusions (SPE). In an antibiotic rich era, \textit{Staphylococcus aureus} predominates presentations of SPE and the clinical syndrome is characterized by fevers, tachycardia, hypotension and altered mental state. Mortality is high with rates of 30 - 50\% despite initiation of targeted therapy due to cardiac tamponade [1]. Prompt diagnosis and management is critical however recognizing SPE remains clinically challenging particularly in patients presenting with more peripherally visible foci of infection. We report a rare case of SPE caused by \textit{Streptococcus pyogenes} in a 47-year-old man who presented with synovial and extra-synovial complications.

Case Report

A 47-year-old gentleman with a background of alcohol abuse presented to the emergency department with left knee pain, fevers, rigors and altered mental status. He had a week of untreated acute pharyngitis, which progressed to systemic deterioration including gradually worsening left knee pain. He reported no recent travel history. His past medical history included alcohol abuse and attention deficit hyperactivity disorder.

On assessment, he was febrile (38.9°C), tachycardic (130/minute) with maintained hemodynamics (125/70 mmHg). On auscultation, dual heart sounds, no cardiac murmurs and clear lung fields were recorded. Left popliteal fullness with extreme pain on passive movement was noted. The left knee aspirate was purulent and immediately sent to the laboratory. His laboratory testing showed raised white cell count (14.8 x 10^9/L) and C-reactive protein (116 mg/L). He was empirically commenced on flucloxacillin 2g intravenous q6hourly and gentamicin 320 mg intravenous STAT as per local guidelines.

On day one of admission, he had a surgical washout. Due to persistent hypotension (90/55 mmHg), he was transferred to the intensive care unit for inotropic support. A metaraminol infusion was commenced with maximal requirements of 0.55 microg/kg/min needed to attain a mean arterial pressure (MAP) greater than 65 mmHg. On day two, his microbiology was positive for *Streptococcus pyogenes*, sensitive to penicillin. Targeted antimicrobial therapy was initiated with benzylpenicillin 1.2g intravenous q4hourly. His haemodynamics began to stabilize at this time and the metaraminol infusion was weaned to 0.06microg/kg/min then ceased. Unfortunately, the patient further deteriorated overnight with ongoing hypotension, tachycardia with global ST-T changes and high-grade fevers (Figure 1). A noradrenaline infusion was titrated to maintain MAP > 65 mmHg and required peak concentrations of 0.22 microg/kg/min. The ECG changes were not associated with an elevated troponin.

**Figure 1:** ECG of patient in the intensive care unit.
By day four of admission the patient had received two washouts of his left knee with minimal improvement of his systemic inflammatory response syndrome and a secondary focus of infection was further investigated. A small fluctuant swelling was found in his right shoulder and aspirated purulent material that further isolated *S. pyogenes*. Despite this intervention the patients’ hypotension remained refractory to fluids with persistent tachycardia and intermittent fevers. We were unable to successfully wean his noradrenaline infusion with requirements fluctuating between 0.04 - 0.1 microg/kg/min. Single antibiotic therapy was continued with Infectious Disease guidance, however dosing at this time was increased to benzylpenicillin 1.8g intravenous q4hourly.

On day seven, a limited bedside echocardiogram revealed evidence of pericardial effusion and right ventricular (RV) hypokinesis suggestive of early cardiac tamponade. Urgent cardiology review including a formal echocardiogram was requested. Formal echocardiogram confirmed a large pericardial effusion with RV hypokinesis. Sterile pericardiocentesis was performed and 100 mL of purulent material was aspirated (Figure 2). Again, *S. pyogenes* was cultured. Post-diagnostic pericardiocentesis, significant improvement in RV function was noted and he no longer had inotropic requirements. Cardiothoracic surgery was consulted and the patient was transferred for urgent pericardial washout. Post-procedure, the patient rapidly improved with a prolonged course of benzylpenicillin 1.8g intravenous q4hourly for a further six weeks and then step-down amoxicillin 1gram orally q8hourly for four weeks with repeat echocardiography.

**Figure 2:** Suppurative pericardial effusion aspiration.

**Discussion**

The widespread use of systemic antibiotics has greatly reduced the incidence of Suppurative pericardial effusion (SPE) in modern practice. In developed countries, bacteria only represent 5% of causative organisms with the majority contributed to by viral and autoimmune aetiologies [2]. SPE was not infrequently a complication of pneumococcal pneumonia however in the post-antibiotic era, most cases are related to haematological spread of septic emboli from nosocomial infections and not from direct extension of respiratory illness’ [1,3,4].

Several studies suggest that 78% of reported cases have one or more predisposing factors, including pre-existing pericardial diseases (i.e. due to uraemia, tumor, collagen vascular disease), immunosuppression, alcohol abuse (as in our highlighted case), thoracic surgery and/or chest wall trauma [5,6].
SPE is a life-threatening diagnosis with mortality rates as high as 40% despite appropriate management [3]. Suspicion and early diagnosis in the setting of haematological infections are important. Once identified, SPE must be aggressively treated before complications including cardiac tamponade, systemic toxicity, cardiac decompensation and constriction progresses [3,7]. Literature review supported the infrequency of \textit{S. pyogenes} as a causative organism, especially in adults, with only nine clearly reported cases of \textit{S. pyogenes} SPE. In these reviews, patients commonly presented with fevers, tachycardia and altered mental state [3]. All patients developed cardiac tamponade with one fatality despite pericardial drainage and antibacterial therapy. The authors universally concluded that septic shock in the setting of synovial infection should prompt a referral for urgent transthoracic echocardiography [8].

Our patient was initially diagnosed with septic arthritis further complicated by septic shock. Despite inotropic support, fluid resuscitation and surgical debridement of infected joints, the patient remained persistently hypotensive. Ongoing haemodynamic instability with persistent inotropic demand and global ST changes (Figure 2) prompted bedside echocardiography. Formal echocardiography for infective endocarditis found a large pericardial effusion, which led to diagnostic pericardiocentesis. It is an important learning point that SPE may be initially disguised by more tangible diagnosis that can potentially delay pericardiocentesis and we reiterate that a high index of suspicion is critical.

Conclusion

Bacterial pericarditis has a tendency to re-accumulate and loculate and therefore initial treatment should aim to decompress the pericardial sac with pericardiocentesis that is both diagnostic and therapeutic [8]. Targeted antimicrobial therapy must be continued, and once haemodynamically stable pericardiectomy is implicated as the gold standard of care [9]. Current guidelines suggest four-eight weeks of targeted antimicrobial therapy to continue thereafter with frequent repeat transthoracic echocardiograms to review for complete clinical recovery [5]. Although \textit{S. pyogenes} is rarely implicated in purulent pericarditis, it is imperative that we still exercise vigilance and consider multifocal haematological spread in patients who present with fevers and shock. This case highlights the importance of bedside echocardiogram as an inexpensive life-saving tool that enriches diagnostic proficiency and enhances management of our critically unwell patients.

Declaration of Conflicting Interests

The authors declare no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

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