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Case Report
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A Case of Acute *Streptococcus pneumoniae* Pericarditis Leading to Fatal Cardiac Tamponade: A Case Report and Literature Review

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Abstract

Background: This case report highlights the initial ambiguous presentation of a patient with acute purulent pericarditis. The etiological agent was streptococcus pneumonia, a rare cause in the age of pneumococcal vaccination.

Case Presentation: An elderly gentleman presented with non-specific chest pain. He was suspected to have had a myocardial infarction, treated as such, but was then found to have pyopericardium. There was rapid progression of the disease which led to cardiac tamponade and obstructive shock. Following emergency drainage, refractory septic shock ensued.

Conclusion: The case report highlights key differences and similarities in history, clinical examination and investigations between pericarditis and myocardial infarction. Finally, we conclude with a discussion based on a literature review focused on pyopericardium.

Introduction

Acute pericarditis is defined as inflammation of the pericardium which resolves within 6 weeks. Pyopericardium is the presence of pus within the pericardial sac. Pyopericarditis is invariably fatal without administration of antibiotics. Broad spectrum antibiotics have decreased mortality rates to 40% [1,2]. The most common cause of bacterial pericarditis is Gram-positive cocci from direct intra-thoracic spread. The case report was approved by the Human Research Ethics Committee (HREC) (Certificate No. M180581).

Case Presentation

A 54 year old gentleman presented at 09:00 to an Emergency Department (ED) with acute-onset central chest pain and non-radiating epigastric pain (grade 10/10); associated with dyspnea. He had a 30 pack year history of smoking and lived a sedentary lifestyle. He had no fever, syncope or any preceding events leading up to presentation to the ED. He had no medical (hypertension, hyperlipidemia or diabetes) or family history of cardiovascular disease.

The initial clinical examination showed an overweight man. He was fully conscious but distressed. He was apyrexial, hypotensive (96 mmHg/58 mmHg) and maintained oxygen saturation above 96% on 4 L/min nasal prongs.

Further examination revealed no jugular vein distension, no muffled heart sounds and clear lung field auscultation. The first Electrocardiogram (EKG) is reported to have a regular pulse rate of 91/min with ST-segment elevation of leads V2-V6, limb leads I, AVL, III and reciprocal ST-depression of AVR. At this point, an anterolateral Myocardial Infarction (MI) was suspected. An initial transthoracic 2-dimensional echocardiogram (echo) was unobtainable. Chest radiography showed no abnormalities of the lung fields but an increased cardiothoracic ratio. Given that Percutaneous Coronary Intervention (PCI) would not be achieved within 90 min from presentation (door-to-balloon time), it was decided that the patient would benefit from thrombolysis. Serial EKG's were performed and the patient reported relief of the chest pain.

Once stable, the patient was transferred to a facility capable of PCI. Upon arrival he was hemodynamically stable and not in cardiac failure, however, his chest pain was assessed as 7/10. The first echo performed revealed an ejection fraction >50%, hypokinesia of the anterolateral wall, as well as a small pericardial effusion. Subsequently a coronary angiogram (5 h after initial

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presentation) discovered a distal segment filling defect of the right coronary artery which was recanalized for suspicion of a thrombus. However, on retrospective analysis the EKG changes were discordant to the angiogram findings.

At 15:00, post-procedure, he was transferred to the cardiac care unit. Six hours after transfer his Acute Kidney Injury (AKI) worsened with an increase in serum creatinine from 239 umol/l to 472 umol/l (no premorbid creatinine available). During this period the high sensitivity Troponin-T level increased from 45 ng/L to 82 ng/L.

At 24:00, a rapid deterioration to shock. A repeat EKG showed ST-segment elevation in leads V2-V6, I and AVL with electrical alternans. The cardiologist performed an echo which showed a significant pericardial effusion with cardiac tamponade. An emergency echo-guided needle pericardiocentesis aspirated 360 ml of foul-smelling, thick, green, purulent fluid which was sent for Gram stain and culture (MC&S). No drain was left in-situ. Post-pericardiocentesis the patient's hemodynamics improved. The gram-stain revealed gram positive diplococci. Antibiotic therapy (amoxicillin/clavulanic acid) and colchicine were commenced.

Over the following 24 h the patient developed multi-organ failure. His level of consciousness deteriorated and he developed type 1 respiratory failure. He was intubated, mechanically ventilated, vasopressor support initiated and transferred to Intensive Care Unit (ICU).

He was in septic shock requiring high-dose vasopressor support (epinephrine at 0.8 μg/kg/min). Investigations were performed to isolate a primary source of infection; including chest X-ray, urine studies and blood cultures. Steroid therapy was initiated and antibiotic regimen changed to tazobactam-piperacillin (loading dose followed by continuous infusion). The choice of antibiotic was empirically based on a community-acquired gram positive organism and to optimize antibiotic concentrations above Minimum Inhibitory Concentrations (MIC) as per ICU protocol. Due to the hemodynamic instability, severe metabolic acidosis and hyperkalemia that persisted despite medical therapy; sodium bicarbonate infusion was initiated. Hemodynamic management was guided by passive leg raise testing and stroke volume variation using continuous invasive hemodynamic monitoring. A repeat echo, in light of continued deterioration, showed recurrent tamponade. Due to the viscosity of the purulent fluid; repeat echo-guided needle pericardiocentesis was unsuccessful. An emergency pericardial window was performed by the surgical team. A further 1000 ml of purulent fluid was drained and sent for MC&S. Marginal hemodynamic improvement enabled initiation of continuous renal replacement therapy for refractory metabolic acidosis, hyperkalemia and anuria. At this stage, the creatinine had increased to 696 μmol/l while the procalcitonin was 909 μg/l. Despite these interventions; vasopressor requirements increased over 24 h. A calcium infusion was started for hypocalcemia and positive inotropic effects. Thiamine given for increasing lactate levels. The pericardial drain output was approximately 900 mL/24 h. An echo showed very poor cardiac contractility. Without access to Extracorporeal Membrane Oxygenation (ECMO); the patient ultimately succumbed to multi-organ failure.

The culture result received 24 h after his demise reported a sensitive *Streptococcus pneumoniae*, with a MIC<0.03 ug/ml to Cefotaxime, thus indicating that tazobactam-piperacillin was appropriate empiric treatment.

The post-mortem results indicated the presence of an occult pneumonia and thick purulent and hemorrhagic exudate on the epicardium.

Discussion

Pyopericardium has an incidence rate of approximately 1/18000 cases of pericarditis. It remains a fatal condition due to tamponade and septic shock. The most common bacterial causes of pyopericarditis are *Staphylococcus aureus*, *Streptococcus pneumoniae*, *Haemophilus influenza* and anaerobic organisms. The incidence of *Streptococcus pneumoniae* has decreased (51% to 9%) over the past 2 decades, following the roll out of the pneumococcal vaccine [2]. Pericarditis secondary to *Streptococcus pneumoniae* occurs predominantly in children and young adults with a likely source of infection i.e. pneumonia (93%). However, recent studies have shown the average affected age has increased to 49 years of age [2]. It has been claimed that it is not a true primary infection but due to hematogenous or direct spread from a non-radiologically significant pneumonia or other occult infection.

Acute pericarditis presents with increasing severity of chest pain that is worse in the recumbent position, fever and other non-specific inflammatory symptoms (tachycardia, altered level of consciousness and dyspnea). Clinical examination can reveal a pericardial friction rub, pulsus paradoxus and the classical Beck's triad of systemic hypotension, Kussmaul's sign (paradoxical rise in jugular venous pressure during inspiration) and muffled heart sounds on auscultation [3,4].

EKG changes are revealing; typical changes including sinus tachycardia, widespread ST-elevation (saddle shaped ST-segment), reciprocal ST depression in lead AVR (+- V1), Spodick's sign (downward sloping TP segment), PR depression, flattened or inverted T waves and/or electrical alternans (Table 1). It is, however, important to note 10% to 35% of patients may have a normal EKG.

Ultimately an echocardiogram revealing a pericardial effusion confirms the diagnosis and while diastolic chamber collapse (right atrial and/or ventricular) indicates tamponade [5]. Troponin concentrations may rise in many forms of cardiac injury including pericarditis. Other relevant investigations are a full blood count, renal functions, inflammatory markers (C-reactive protein, procalcitonin) and blood cultures. An angiogram assists to exclude a myocardial infarction, if suspected [4]. Further investigation based on the suspected primary source of the acute pyopericarditis may be appropriate.

To confirm the diagnosis of acute pericarditis the patient must meet 2 of the following 4 criteria: Typical chest pain, pericardial friction rub, typical EKG changes and new or worsening pericardial effusion.

The treatment of pyopericardium incorporates medical and surgical interventions and the urgency of intervention determined

Table 1: Steps to distinguish pericarditis from STEMI [16].

	Relevant electrocardiogram feature	Presence	Likely diagnosis
1	Is there ST depression in a lead other than AVR or V1?	Yes	STEMI
2	Is there convex up or horizontal ST elevation?	Yes	STEMI
3	Is the ST elevation greater in III than II?	Yes	STEMI
4	Is there PR depression in multiple leads	Yes	Pericarditis

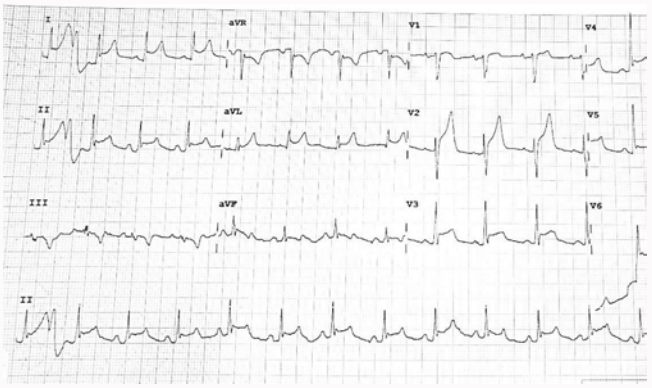

Figure 1: Electrocardiogram at presentation (25 mm/s, 10 mV, 50 Hz).

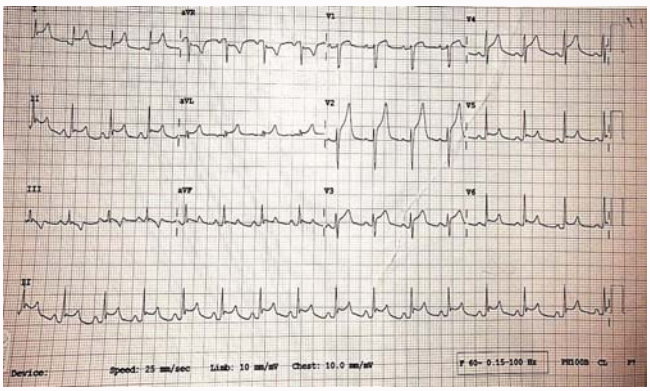

Figure 2: Electrocardiogram: 1 hour post-fibrinolysis (25 mm/s, 10 mV, 50 Hz).

by the presence of tamponade. The medical management includes broad spectrum antibiotics for 2 to 4 weeks. Antibiotic choice guided by the most commonly associated pathogens. Provided there are no contra-indications, these of colchicine and ibuprofen are advocated for analgesia. The anti-inflammatory effects of colchicine have been proven to expedite resolution of primary pericarditis as well as prevent recurrences by up to 50%, but use in pyopericarditis not yet studied. It is recommended as first line therapy for up to 12 weeks [6].

Early corticosteroid use may actually increase the risk of relapse [6]. Low dose corticosteroids are suggested in septic shock; if fluid and vasopressor therapies are not able to restore adequate hemodynamics. The presence of sepsis and tamponade result in a complex hemodynamic state with a combination of obstructive and distributive shock. The sepsis induced myocardial dysfunction will include poor systolic function, diastolic dysfunction and vasoplegia. Hemodynamic management is not trivial and requires advanced continuous hemodynamic monitoring. Veno-arterial ECMO may be useful for hemodynamic failure in septic shock.

Urgent source control is a priority and would include ultrasound guided emergency pericardiocentesis for tamponade and definitive surgical pericardiotomy or a pericardial window if thick purulent fluid proves difficult to drain percutaneously.

Important complications of pyopericardium are septic shock, cardiac tamponade with obstructive shock, recurrent pericarditis and constrictive pericarditis (Figures 1-4).

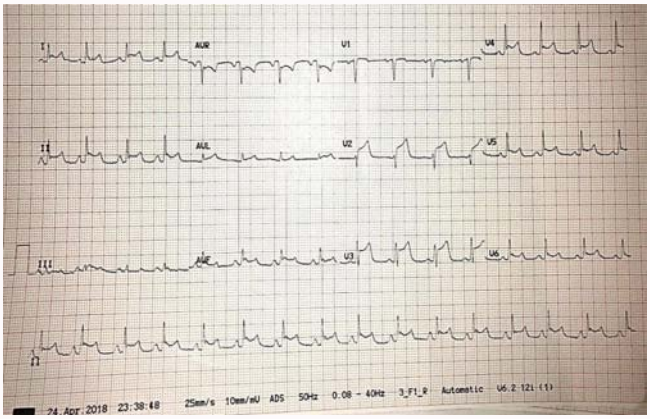

Figure 3: Electrocardiogram prior to pericardiocentesis (25 mm/s, 10 mV, 50 Hz).

Figure 4: Purulent fluid aspirated by echo-guided needle pericardiocentesis.

Conclusion

Acute bacterial pericarditis is a diagnosis that requires a high-index of suspicion and is often accompanied by an associated primary infection. EKG changes need to be carefully scrutinized and the echo performed timorously to differentiate between acute myocardial infarction (cardiogenic shock) and acute pericarditis with cardiac tamponade (obstructive shock).

Despite adequate drainage, cardiac tamponade from pyopericardium may well progress to refractory vasodilatory septic shock requiring ECMO.

Permissions

1. Permission granted by Chris Hani Baragwanath Academic Hospital

2. Permission granted to collect patient data by Chris Hani Baragwanath Hospital Intensive Care Unit

3. Permission granted and consent obtained by Human Research Ethics Committee (Medical) Clearance Certificate No. M180581

Declarations

Author contributions

1. Wezley Laney. This author helped with case report design, patient data retrieval and analysis, article drafting, final approval and submission.

2. Edward Buga. This author helped with case report design, analysis and interpretation, article review, and final approval.

3. Shahed Omar. This author helped with case report design, analysis and interpretation, article review, final approval, hospital and ethics permission and submission.

Ethics approval and consent to participate

1. Written consent obtained from next-of-kin

2. Ethics approval obtained from Human Research Ethics Committee (Medical) Clearance Certificate No. M180581.

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