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CASE REPORT PEER REVIEWED | OPEN ACCESS

A case of non-ST elevation MI with revascularization by PCI complicated by bacterial pericarditis

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ABSTRACT

Introduction: Pericarditis, an inflammation of the pericardial sac, can stem from various causes, including idiopathic, viral, and infectious origins. While viral and idiopathic cases are common in developed countries, tuberculosis is a leading cause in developing regions. Purulent bacterial pericarditis usually follows surgery or bacteremia. Post-myocardial infarction (MI) pericarditis occurs in early and late phases, the latter known as Dressler's syndrome. This case report presents a rare occurrence of purulent bacterial pericarditis after percutaneous coronary intervention (PCI).

Case Report: Our case describes a 57-year-old male with coronary artery disease (CAD), hypertension, and a recent upper respiratory tract infection (URTI) who presented with severe left shoulder pain. He was diagnosed with an acute MI and underwent PCI. Post-procedure, he developed increasing leukocytosis, fever, and respiratory distress. Despite initial broad-spectrum antibiotics, his condition necessitated further imaging and eventually pericardiocentesis, which revealed Methicillin-sensitive Staphylococcus aureus (MSSA) infection. Targeted antibiotic therapy led to his clinical improvement.

Conclusion: This case highlights the necessity for vigilant differential diagnosis and prompt treatment of rare but serious complications such as bacterial pericarditis following PCI.

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INTRODUCTION

Pericarditis is a condition characterized by inflammation of the visceral and parietal pericardium, usually accompanied by pericardial effusion [1]. The pericardium surrounds the heart as a barrier to prevent the spread of infection into the heart, but also provides a structural component to maintain ventricular compliance by preventing cardiac dilation [2]. In developed countries, most cases of pericarditis are of an idiopathic or viral origin, while in developing countries, the most common etiology is disseminated tuberculosis [3, 4]. Purulent bacterial pericarditis usually occurs following surgical manipulation such as in open heart surgery or through bacteremia with dissemination of systemic infection [5].

Pericarditis following myocardial infarction commonly occurs in two phases, an early phase peri-infarction pericarditis due to ischemic injury to the underlying myocardium and a late phase immune-mediated post-cardiac injury pericarditis (Dressler's syndrome). The incidence of Dressler syndrome has decreased to 6% since the advent of thrombolytic agents [6]. Diagnosis of pericarditis is made through a combination of clinical presentation and diagnostic data. The most common presenting symptom is chest pain, and auscultation can reveal a pericardial friction rub which is highly specific for

the condition [1]. The electrocardiogram (ECG) findings in pericarditis involve diffuse ST elevations with or without PR depression. Echocardiogram most often shows pericardial effusions. Cardiac computed tomography (CT) can also be used to confirm the diagnosis. Idiopathic and viral cases are usually treated with colchicine and nonsteroidal anti-inflammatory drugs (NSAIDs) while treatment of bacterial pericarditis requires antibacterial therapy based on culture data.

Patients suspected of having an ST elevated myocardial infarction (STEMI) are recommended to undergo revascularization, with either percutaneous coronary intervention (PCI) or fibrinolysis.

Infectious complications following PCI-mediated revascularization are relatively rare with an incidence of 2.4%, although it can be assumed that with improvement in medical knowledge and technology, the incidence may be much lower [7]. Detailed literature review revealed multiple cases of bacteremia following PCI, but cases of purulent bacterial pericarditis were very rare. Bacterial pathogens include *S. aureus* [8], *Haemophilus influenza*e, *Neisseria meningitidis*, and *Streptococcus pneumoniae* and rarely fungal pathogens such as Candida and Aspergillus [9, 10]. In 2012, a case reported a patient with *S. aureus* as the most common source of bacteremia post-elective PCI intervention, and when correlated to a previous study, concluded that the possibility of infection was related to instrumentation as the incidence of developing bacteremia post-PCI intervention for an MI is uncommon [7].

Our case describes a gentleman presenting with an ST-elevation MI with revascularization achieved by PCI with his hospital course complicated by purulent bacterial pericarditis.

CASE REPORT

A 57-year-old male with past medical history (PMH) of coronary artery disease (CAD) s/p coronary artery bypass graft surgery (CABG) on 9/7/2012 (at age 45), mild mitral insufficiency, hyperlipidemia (HLD), active tobacco use disorder 1PPD, alcohol use disorder, hypertension (HTN), recent URTI presenting to the emergency department (ED) with left-sided 8/10, non-radiating shoulder pain. In the ED, he was hemodynamically stable: T98.4, BP 134/85, HR 95, and RR 18. Lab work most significant for mild leukocytosis of 16.2 10^9/L with neutrophil predominance. The ECG showed subtle and non-significant ST changes in leads II, III, aVF (Figure 1). The ECG was reviewed by interventional cardiology and deemed not a ST-elevation myocardial infarction (STEMI).

Initial troponins were within normal limits (6–>8 ng/L) eventually peaking at 9 ng/L. The patient was subsequently admitted under the interventional cardiology service for management of non-ST elevation myocardial infarction (NSTEMI) and taken to Cath Lab for urgent left heart catheterization (LHC). Left heart catheterization was successfully done with finding of right dominant circulation with patent saphenous vein graft to right coronary artery (SVG-RCA), saphenous vein graft to 2nd Obtuse Marginal (SVG-2nd OM), and left internal mammary artery to left anterior descending artery (LIMA-LAD) grafts. The left main and circumflex arteries had no angiographically significant disease while the proximal LAD had diffuse 70% distal disease with discrete 100% and 90% proximal lesions which was stented to 0%. Left heart catheterization was also significant for discrete 100% lesions in the right coronary artery (RCA) and 2nd obtuse marginal arteries (Figure 2). The procedure was done without any reported complications including perforations.

Echocardiogram on the same day showed preserved systolic and diastolic function with a moderate-sized anterior pericardial effusion (Figure 3).

Following LHC, the patient developed worsening leukocytosis (16.2–>21.4–>26.4 × 10^9/L) with low grade fevers (99.5°F and 100°F). Blood cultures were obtained which subsequently had no growth on completion.

Methods (Differential Diagnosis, Investigations, and Treatment)

Differentials at this time included:

1. Post-MI pericarditis
2. Post-procedure atelectasis and pneumonia
3. URTI
4. Urinary tract infection (UTI)
5. Less likely, bacterial pericarditis (based on its frequency as a complication).

Infectious workup including detailed history, chest X-ray (CXR), urinalysis (UA), and infectious respiratory panel were unrevealing for a source such as pneumonia, URTI, UTI, or intravenous (IV) drug related infection.

On the second day of his hospitalization, he developed low grade fevers with diaphoresis, and difficulty breathing. Computed tomography angiography (CTA) chest showed small to moderate pericardial effusions with trace bilateral pleural effusions and no evidence of pulmonary embolus. Infectious disease was consulted, and he was started empirically on broad spectrum antibiotics namely Vancomycin and Zosyn. C-reactive protein (CRP) was 45.5 mg/L with erythrocyte sedimentation rate (ESR) 102 mm/h. Repeat echo showed right ventricular (RV) diastolic collapse consistent with a hemodynamically significant pericardial effusion (Figure 4).

Although he had no clinical signs of tamponade such as hypotension, given echo findings consistent with worsening effusion and impending tamponade physiology, he was also started on colchicine and aspirin for post-MI pericarditis. Two days later, he underwent ultrasound (US)-guided pericardiocentesis with drainage of 300 cc of serous straw-colored fluid (Figure 5). Initial pericardial pressure during pericardiocentesis was 15 mmHg with post-pericardiocentesis pressure 2 mmHg.

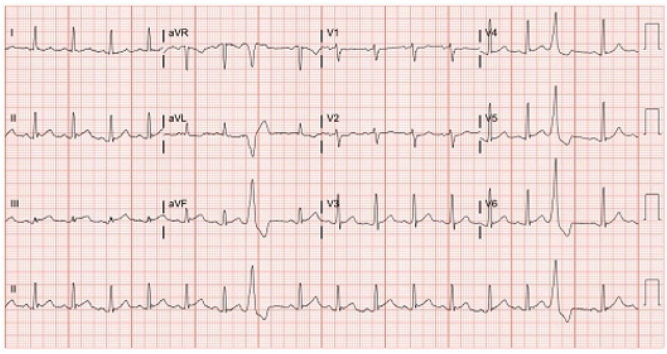

Figure 1: ECG. Normal sinus rhythm with subtle non-significant ST-segment changes in the inferior leads.

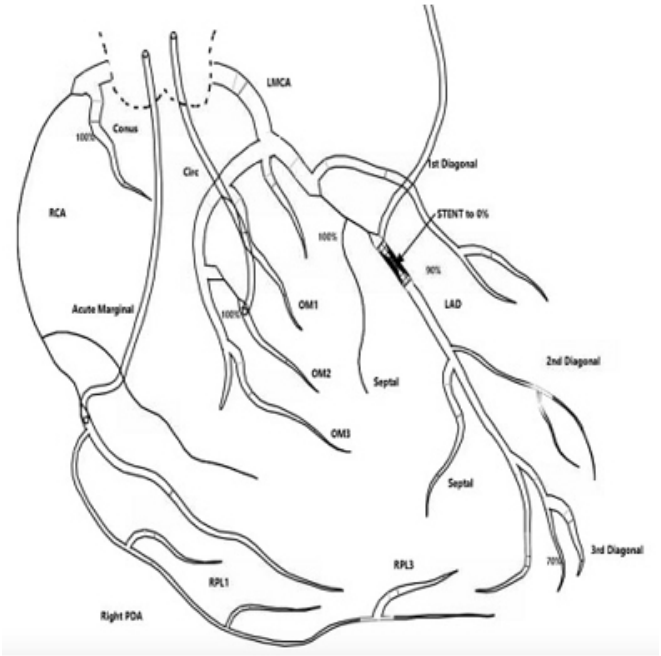

Figure 2: LHC findings with patent grafts with 90% LAD lesion stented to 0%.

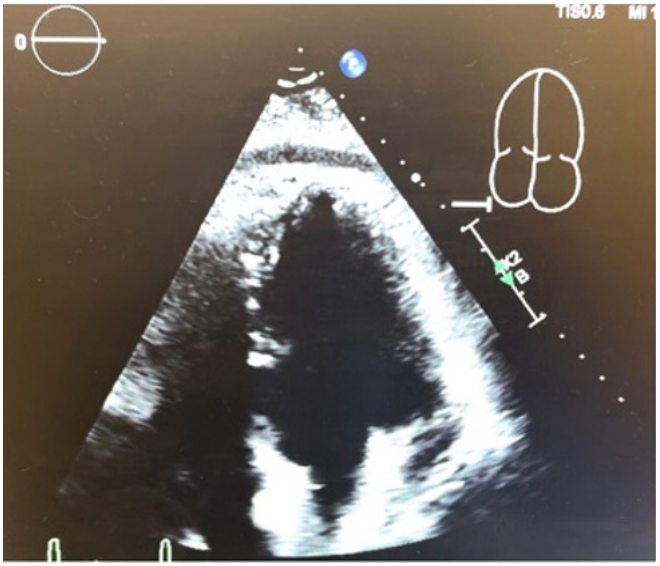

Figure 3: Initial echocardiogram. Apical 4-chamber view showing mild–moderate pericardial effusion.

Cytology was negative for malignancy but showed rare mesothelial cells with severe acute inflammation. Blood cultures showed no growth, pericardial fluid cultures eventually grew Methicillin-sensitive *Staphylococcus aureus* (MSSA) and antibiotics were de-escalated to cefazolin.

Follow-up CT chest showed pericardial drain in place with decreased fluid collection. Follow-up echo after drain removal revealed left ventricular ejection fraction (LVEF) of 40–49% with small appearing RV with restricted filling with a hemodynamically significant thrombus and loculated effusion within the pericardium anteriorly and apically causing right heart compromise and respiratory variations to both the left and right heart (Figure 6) consistent with effusive constrictive pericarditis. Despite this echocardiographic finding, given his stable clinical presentation and stable echocardiographic findings, a decision was made to manage conservatively and follow up in the outpatient setting with repeat imaging and further intervention.

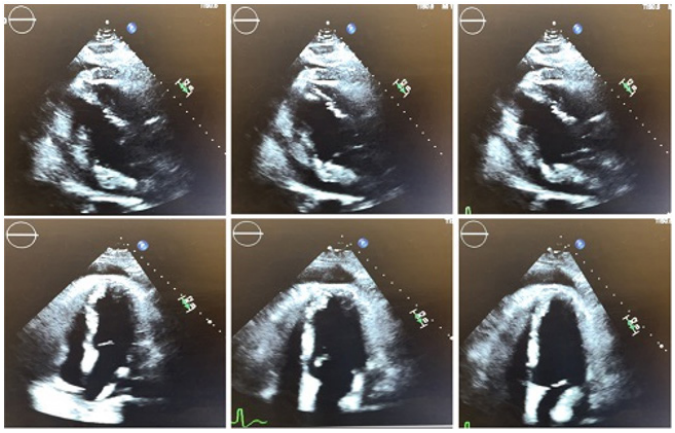

Figure 4: Repeat echocardiogram showing hemodynamically significant pericardial effusion with diastolic collapse. Top images: Parasternal long; Bottom images: Apical 4-chamber.

Figure 5: Fluid sample from pericardial drain.

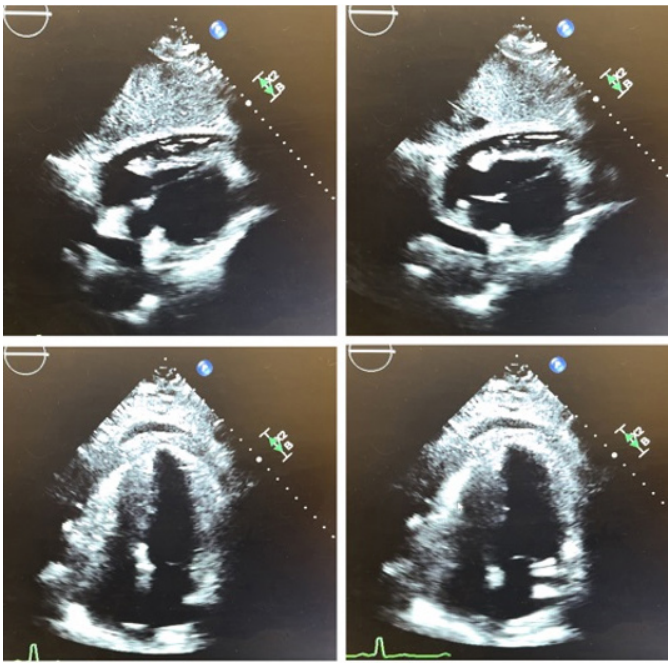

Figure 6: Final echo prior to discharge showing pericardial thrombus. Top images: Subxiphoid view; Bottom images: Apical 4-chamber view.

DISCUSSION

Pericarditis following revascularization is usually post-infarction pericarditis or a delayed autoimmune pericarditis (Dressler's syndrome) which can be managed with colchicine and full dose Aspirin. Given that there is no entry into the pericardial space, PCI should not be associated with infections in the pericardial service. Here, we present a rare case of bacterial pericarditis complicating the hospital course of a patient following revascularization.

CONCLUSION

The patient improved clinically, remained hemodynamically stable and was subsequently discharged home to complete a 6-week course of Cefazolin with close outpatient follow-up with cardiology, cardiothoracic surgery, and infectious disease. Following discharge, the patient was evaluated in the outpatient setting by his primary care physician, infectious disease and cardiology. He was symptom-free with CRP <1 mg/L and ESR 11 mm/h.

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Author Contributions

Anderson Ariaga – Conception of the work, Design of the work, Drafting the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Felicia Zhang – Design of the work, Drafting the work, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Pradnya Brijmohan Bhattad – Design of the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

Nili Gujadhur – Conception of the work, Design of the work, Revising the work critically for important intellectual content, Final approval of the version to be published, Agree to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved

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Conflict of Interest
Authors declare no conflict of interest.

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All relevant data are within the paper and its Supporting Information files.