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Case Report

A case report of post coronary artery bypass grafting Dressler's syndrome presenting with cardiac tamponade

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ABSTRACT

Dressler's syndrome is an inflammatory complication following myocardial injury or cardiac surgery that may rarely progress to clinically significant pericardial effusion and cardiac tamponade. We report a case of a patient who developed delayed symptomatic pericardial effusion following coronary artery bypass grafting, with echocardiography demonstrating tamponade physiology requiring urgent pericardiocentesis and drainage. The patient was subsequently managed with anti-inflammatory therapy with clinical and echocardiographic improvement, highlighting the importance of recognizing PPS as a potentially life-threatening cause of delayed post- coronary artery bypass grafting pericardial effusion.

Keywords: Dressler's syndrome, Post cardiotomy injury syndrome, Cardiac tamponade, Pericardiocentesis, Post CABG

INTRODUCTION

Dressler's syndrome, also known as post-cardiac injury syndrome (PCIS), is an immune-mediated inflammatory condition that develops following myocardial or pericardial injury. It has been described after myocardial infarction, cardiac surgery, percutaneous coronary intervention, and cardiac device implantation.[1] The syndrome typically presents with fever, pleuritic chest pain, pericardial or pleural effusion, and elevated inflammatory markers occurring days to several weeks after the initial cardiac insult.[2] Post-pericardiotomy syndrome following coronary artery bypass grafting (CABG) represents an important subset of PCIS and may occasionally manifest with significant pericardial effusion or cardiac tamponade requiring urgent intervention.[3] Although the incidence of Dressler's syndrome has declined in the modern reperfusion era, it remains an important differential diagnosis in patients presenting with pericarditis-like symptoms after cardiac surgery.[4] Early recognition is essential because most patients respond well to anti-inflammatory therapy, while some cases may require pericardial drainage when hemodynamically significant effusion develops.[5]

Here a case of a patient presenting several weeks after coronary artery bypass grafting (CABG) with progressive dyspnea, hypotension, and tachycardia is reported. Echocardiography demonstrated a large pericardial effusion with features consistent with cardiac tamponade. Emergency pericardiocentesis was performed, resulting in immediate hemodynamic improvement. Subsequent evaluation suggested Dressler's syndrome as the underlying etiology. The patient was treated with anti-inflammatory therapy and demonstrated clinical recovery without recurrence during follow-up.

CASE REPORT

A 66-year-old male with a history of coronary artery disease underwent elective coronary artery bypass grafting (CABG) for triple vessel coronary artery disease

at another institution. The postoperative period was initially uneventful, and the patient was discharged in stable condition on guideline-directed medical therapy including dual antiplatelet therapy, statin, beta-blocker, and angiotensin-converting enzyme inhibitor. He was also a known case of type II Diabetes mellitus, Hypertension and dyslipidemia.

Approximately four weeks after surgery, the patient presented to the emergency department of our hospital with progressively worsening dyspnea, generalized fatigue, palpitations and chest discomfort over several days. The patient also reported pain at surgical site and mild orthopnea. There was no history of fever, productive cough, or recent trauma.

On examination, the patient appeared uncomfortable and tachypneic. Vital signs revealed hypotension with a blood pressure of 80/60 mmHg, tachycardia with a heart rate 118/min, respiratory rate 25/min, and oxygen saturation 96 on room air. Jugular venous pressure was elevated, and heart sounds were muffled on auscultation. Peripheral pulses were weak but palpable. There was no significant peripheral edema. Surgical site showed healing wound with minimal discharge.

Table 1: Initial laboratory investigations.

Investigations	Results
Hemoglobin	12 g/dl
Total leukocyte count	15980 cells/ul
Platelet count	3.55 lakh/ul
Serum creatinine	1.2 mg/dl
Blood urea	34 mg/dl
Sodium	134 mEq/l
Potassium	4.4 mEq/l
C-reactive protein	152 mg/l
Erythrocyte sedimentation rate	92 mm/hr
High-sensitivity troponin	16 ng/l
D-dimer	6.7 ug/ml
Procalcitonin	9.68 ng/ml

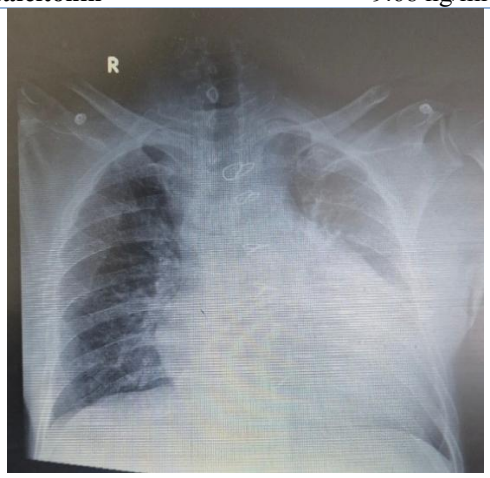

Figure 1: Chest X-ray AP view of patient on admission: cardiac silhouette is enlarged and sternotomy sutures noted.

Initial laboratory investigations showed elevated inflammatory markers including C-reactive protein, Procalcitonin and total count. Cardiac biomarkers were mildly elevated (Table 1). Electrocardiography demonstrated sinus tachycardia and nonspecific ST-T changes. Chest radiography revealed an enlarged cardiac silhouette Figure 1.

Patient was shifted to ICU and was initiated on ionotropic supports, non invasive ventilation and supportive care. Given the hemodynamic instability and clinical suspicion of pericardial effusion, urgent transthoracic echocardiography was performed. The echocardiogram (Table 2) demonstrated a large circumferential pericardial effusion with echocardiographic features suggestive of cardiac tamponade, including right atrial and right ventricular diastolic collapse and exaggerated respiratory variation in ventricular inflow velocities.

Table 2: Echo Parameters.

Echo parameters	Findings
LV ejection fraction	62%
Pericardial effusion size	Moderate to severe
Distribution of effusion	circumferential
Right atrial collapse	+
Right ventricular diastolic collapse	+
Respiratory variation in mitral inflow	>30%
IVC diameter	2.4 cm
IVC collapsibility	20%
Other structural abnormalities	LA dilated, Concentric LVH, no RWMA

In view of the hemodynamic compromise, emergent pericardiocentesis was performed under echocardiographic guidance. Approximately 500 ml of serous pericardial fluid was aspirated, resulting in immediate improvement in blood pressure and symptomatic relief. A pericardial drainage catheter was left in situ for continuous drainage.

Pericardial fluid analysis (Table 3) revealed inflammatory lymphocyticexudative fluid with negative bacterial cultures, and cytology was negative for malignant cells. Microbiological evaluation did not demonstrate evidence of bacterial or tuberculous infection. In the absence of alternative etiologies and given the temporal relationship with recent cardiac surgery, a diagnosis of post-pericardiotomy syndrome (Dressler's syndrome) was considered most likely.

Patient was intubated on day 2 due to respiratory distress and hypoxemia and put on mechanical ventilation. He was initiated on antibiotics with MRSA cover and later de-escalation done after clinical improvement and negative cultures. His inotropic supports were tapered off by day 3. Following stabilization; the patient was

initiated on anti-inflammatory therapy with aspirin 150 mg, steroids (prednisolone 30 mg/day) and colchicines (1 mg daily). Pericardial fluid was subsequently drained on following days through the catheter around 150 ml on day 2, 90 ml day 3 and 50 ml day 4. During ICU stay, he developed L sided hemiparesis on day 2-MRI brain showed multi infarct cardioembolic stroke in R MCA territory and he was conservatively managed in concordance with Neurology team with antiplatelets and anticoagulation.

Table 3: Pericardial fluid analysis.

Parameters	Results
Appearance	Serous
Total volume drained	500 ml
Total protein	4.1 g/dl
LDH	684U/l
Glucose	253 mg/dl
Total leukocyte count	710 cells/mm³
Differential count	(Neutrophils 4% / lymphocytes 96 %)
Red blood cell count	10-12cells/mm³
Gram stain	Negative
Bacterial culture	No growth
Acid-fast bacilli smear	No AFB seen
TB PCR / GeneXpert	Not detected
Fungal culture	No growth
Cytology for malignant cells	Not seen

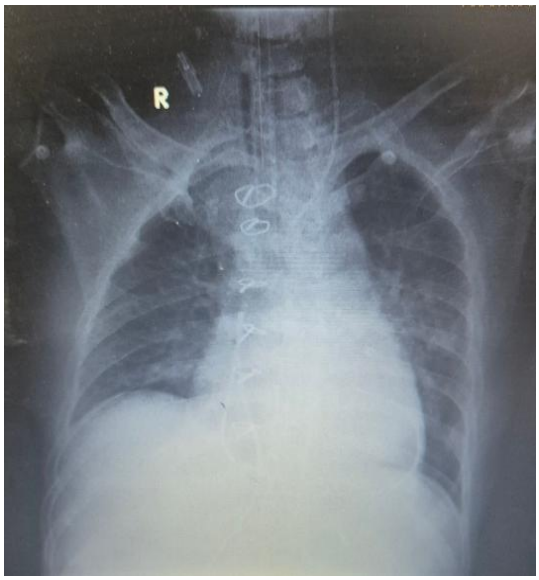

Figure 2: Chest X-ray AP view of the patient post pericardiocentesis and intubation: pericardial catheter *in situ*.

The patient demonstrated gradual clinical improvement, and repeat echocardiography showed minimal residual pericardial effusion. The pericardial drain was removed after 4 days once drainage decreased significantly. He was gradually weaned off ventilation and extubated on day 5. He improved clinically and radiological resolution also noted (Figure 2 and 3). Steroid dose tapering was done. He was later shifted out of ICU and supportive care including neuro rehabilitation continued.

The patient was discharged in stable condition with continuation of anti-inflammatory therapy and scheduled follow-up. At follow-up after 2 weeks and 1 month, the patient remained asymptomatic, and repeat echocardiography demonstrated no recurrence of significant pericardial effusion.

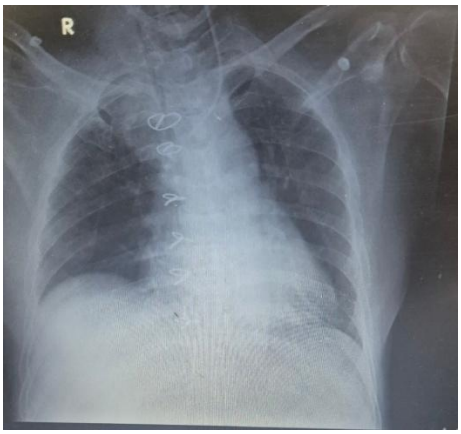

Figure 3: Chest X-ray AP view of patient showing complete resolution of pericardial effusion on day 7.

DISCUSSION

Dressler's syndrome, also known as post-cardiac injury syndrome (PCIS), represents an immune-mediated inflammatory response that occurs after myocardial or pericardial injury. The syndrome may develop following myocardial infarction, cardiac surgery, percutaneous coronary intervention, or cardiac device implantation.[1,8,9] The underlying pathophysiology is believed to involve autoimmune activation triggered by exposure of pericardial and myocardial antigens after cardiac injury, resulting in inflammation of the pericardium and pleura.[8,11]

The clinical presentation typically occurs several weeks after the inciting cardiac event and includes fever, pleuritic chest pain, malaise, and the development of pericardial or pleural effusion.[1,5] Post-pericardiotomy syndrome following coronary artery bypass grafting (CABG) represents a well-recognized manifestation of PCIS.[1] While most cases are mild and respond to anti-inflammatory therapy, a minority of patients may develop large pericardial effusions that progress to cardiac tamponade, a life-threatening complication requiring urgent intervention.[3,4]

Cardiac tamponade occurs when rapid or progressive accumulation of pericardial fluid leads to elevated intrapericardial pressure, impairing ventricular filling and

reducing cardiac output. Early recognition is essential because delayed treatment may result in obstructive shock and hemodynamic collapse. Echocardiography remains the diagnostic modality of choice for identifying pericardial effusion and assessing features of tamponade physiology, including right atrial or right ventricular collapse and exaggerated respiratory variation in ventricular filling.[11]

Several case reports have documented cardiac tamponade as a manifestation of Dressler's syndrome. Hertzeanu et al described a patient who developed tamponade secondary to Dressler's syndrome requiring pericardial drainage.[4] Similarly, Lee et al reported cardiac tamponade occurring after CABG in the context of post-pericardiotomy syndrome, in which urgent pericardiocentesis resulted in rapid clinical improvement.[3] These cases highlight that although PCIS is generally considered benign, it can occasionally present with severe hemodynamic compromise.

Management of PCIS primarily involves anti-inflammatory therapy. Current recommendations from the European Society of Cardiology guidelines on pericardial disease advocate the use of nonsteroidal anti-inflammatory drugs or high-dose aspirin as first-line therapy, with colchicine as an adjunct to reduce recurrence and improve symptom resolution.[11,12] However, when a large pericardial effusion results in tamponade physiology, urgent pericardiocentesis is indicated to relieve intrapericardial pressure and restore cardiac output.[3,4] Following drainage, continued anti-inflammatory therapy is recommended to control the underlying inflammatory process and prevent recurrence.[8,10]

The present case highlights an important but uncommon presentation of Dressler's syndrome manifesting as cardiac tamponade requiring urgent pericardiocentesis and subsequent anti inflammatory therapy. Our patient also had further complications including respiratory failure requiring mechanical ventilatory support and cardioembolic CVA. He recovered with prompt treatment.

Awareness of Dresslers syndrome is essential for clinicians managing patients in the postoperative period following CABG or other cardiac procedures. Early diagnosis using echocardiography and prompt intervention can be lifesaving and may prevent progression to hemodynamic collapse. Recognition of PCIS as the underlying etiology is also important to guide subsequent anti-inflammatory therapy and reduce the risk of recurrence.[1,3,8]

CONCLUSION

Dressler's syndrome should be considered in patients presenting with pericardial effusion weeks after CABG. Prompt echocardiographic evaluation and timely pericardiocentesis can be lifesaving when cardiac tamponade develops.

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