Case Report

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Post-Myocardial Infarction Complications: A Case of Cardiac Tamponade Secondary to Dressler's Syndrome

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Abstract

Cardiac tamponade, a rare but life-threatening complication following myocardial infarction (MI), can manifest acutely and necessitate urgent intervention. In this case report, we present the clinical course of a 47-year-old male who developed cardiac tamponade secondary to Dressler's syndrome three weeks after a non-ST-elevation myocardial infarction (NSTEMI). The patient presented with epigastric pain, a syncopal episode, and hypotension. Bedside echocardiography revealed a massive pericardial effusion with signs of tamponade physiology. We promptly performed emergency pericardiocentesis, which led to significant clinical improvement. Early recognition and management are crucial to preventing life-threatening complications.

Keywords: Cardiac tamponade, dressler syndrome, pericardiocentesis

Introduction

Dressler syndrome, also known as post-myocardial infarction syndrome, is characterized by aseptic pericarditis that develops following a documented cardiac injury (1). Historically, the occurrence of Dressler syndrome with acute myocardial infarction (AMI) varied between 3% and 4% before the widespread use of coronary revascularization. However, with advancements in early reperfusion therapy and immune-modulatory drugs, the incidence of Dressler syndrome has decreased significantly to less than 0.1% (1, 2).

Cardiac tamponade, although uncommon in the context of Dressler syndrome, can have lethal consequences if not promptly recognized and treated. The accumulation of pericardial fluid can lead to obstructive shock and cardiovascular collapse. Therefore, maintaining a high index of suspicion, conducting thorough clinical assessment, and utilizing ultrasound in emergency settings are essential for timely diagnosis. Emergent pericardiocentesis remains a life-saving intervention.

Only a few case studies of Dressler syndrome complicated with cardiac tamponade have appeared in recent years. This case report highlights the clinical presentation, diagnostic challenges, and therapeutic approach for a patient who developed cardiac tamponade due to Dressler syndrome post-NSTEMI, emphasizing the importance of vigilance in post-MI care.

Case

A 47-year-old Malay male with pre-existing hypertension and ischemic heart disease was transferred from a private hospital to our emergency department for acute myocardial infarction (AMI) at midnight. He presented to the ED after three hours of epigastric pain, accompanied by a syncopal attack. Three weeks prior, he had a history of NSTEMI. During the previous admission, an echocardiogram revealed a left ventricle ejection fraction of 45% with apical septal and anterior hypokinesia but no left ventricle thrombus or pericardial effusion. A subsequent coronary angiography conducted two weeks ago revealed single-vessel disease with chronic total occlusion of the mid-left anterior descending artery.

During the presentation, the patient experienced severe, crushing epigastric pain while at rest, radiating to both shoulders and accompanied by diaphoresis and dyspnoea. His wife discovered his syncopal episode while he was using the toilet and promptly took him to a private hospital. He regained consciousness after 20 minutes. Aside from these

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symptoms, he reported no palpitations, lower limb swelling, headaches, dizziness, fever, upper respiratory tract infection symptoms, or gastroenteritis symptoms. He was treated with antiplatelets for AMI because of ST elevation in ECG leads V2-V6 and was referred to our ED for further management.

Upon arrival at our ED, the patient was alert and conscious, experiencing moderate pain, rated at four on the pain scale, and had no signs of tachypnoea. Physical examination revealed a blood pressure of 100/60 mmHg without inter-arm discrepancy, a heart rate of 150 bpm characterized by moderate pulse volume and irregularly irregular pulse, SPo2 of 95% on room air, and a temperature of 37 °C. The cardiovascular examination revealed a muffled heart sound with a dual rhythm, no murmurs, pericardial rubs, or prominently distended neck veins. Other systemic examinations were unremarkable.

The cardiac monitor detected paroxysmal atrial fibrillation, with an initial ECG showing a fast ventricular response and aberrant conduction or ventricular premature complexes. Subsequent ECGs revealed sinus tachycardia, low voltage limb leads, Q wave and ST elevation over anterior leads (V1–5), and T wave inversion over precordial leads (V2–6) (Figure-1). Bedside cardiac ultrasound identified an aortic root measuring 3.3 cm with no intimal flap seen, massive pericardial effusion (maximum thickness of 3 cm), and right ventricular collapse during diastole (Figure-2). Due to persistently low blood pressure and evidence of cardiac tamponade, an emergency bedside ultrasound-guided subxiphoid pericardiocentesis was performed, draining 100 cc of haemorrhagic pericardial fluid.

Post-pericardiocentesis, vital signs improved, with blood pressure reaching 111/63 mmHg and the heart rate reducing to 83 bpm. A chest X-ray revealed cardiomegaly and an urgent chest CT angiography (CTA) confirmed the presence of a large pericardial effusion measuring 2.8 cm in maximum thickness, along with the collapse of the right ventricle relative to the left ventricle, suggestive of the tamponade effect (Figure 3). Laboratory tests showed a borderline high white blood cell count, thrombocytosis, elevated infective and cardiac markers, transaminitis, and acute kidney injury. However, pericardial fluid analysis ruled out infection and malignancy.

The cardiology team admitted him to the CCU with a diagnosis of pericarditis-induced pericardial effusion post-NSTEMI, likely Dressler syndrome. A formal inpatient echocardiogram revealed global pericardial effusion with clot formation seen over the left ventricular apex, hypokinesia over the apical wall, and a moderate ejection fraction (40–50%). Treatment included anti-inflammatory drugs (Aspirin 750 mg TID, Colchicine 0.5 mg BID) and oral anticoagulants (Warfarin). The patient remained hemodynamically stable during the two-week hospitalization with no bleeding tendencies or other active complaints. Serial bedside echocardiograms demonstrated a reduction in the size of the pericardial effusion. The patient was discharged with nine months of dual therapy (Aspirin plus Warfarin) and scheduled for follow-up in the cardiac clinic as an outpatient. Written informed consent was taken from the patient.

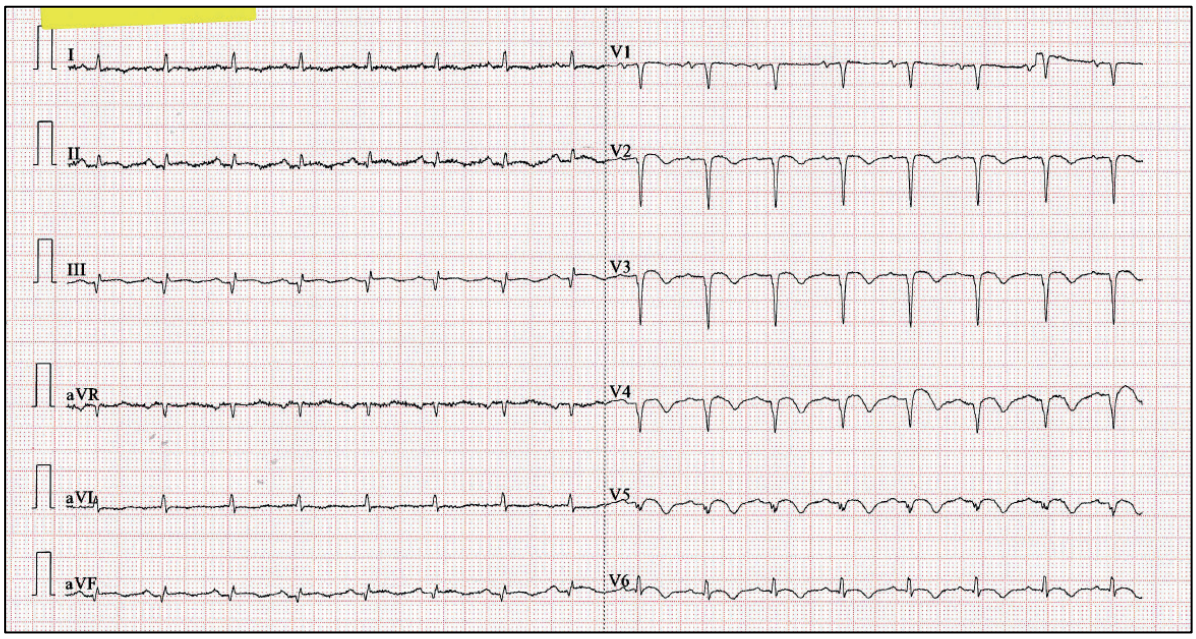

Figure 1. 12 leads ECGs revealed sinus tachycardia, low voltage limb leads, Q wave and ST elevation over anterior leads (V1-5), and T wave inversion over precordial leads (V2-6).

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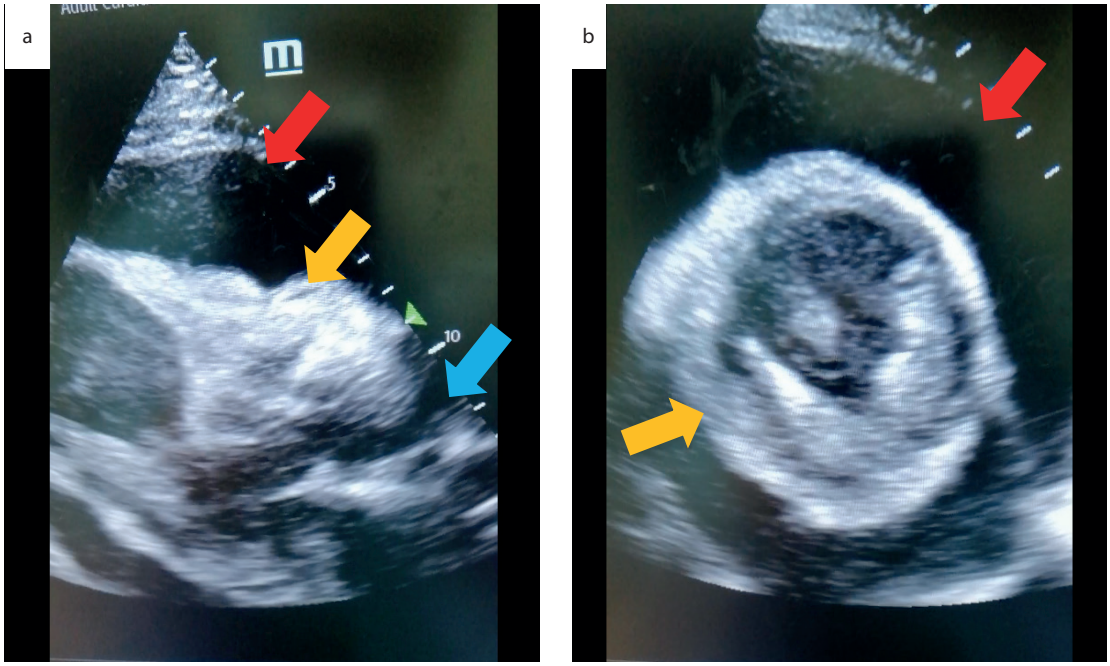

Figure 2. Bedside cardiac echocardiography: (a) Parasternal long-axis view and (b) Parasternal short-axis view during diastole phase identified an aortic root measuring 3.3cm with no intimal flap seen (blue arrow), massive pericardial effusion (maximum thickness of 3cm) (red arrow), and right ventricular collapse (orange arrow).

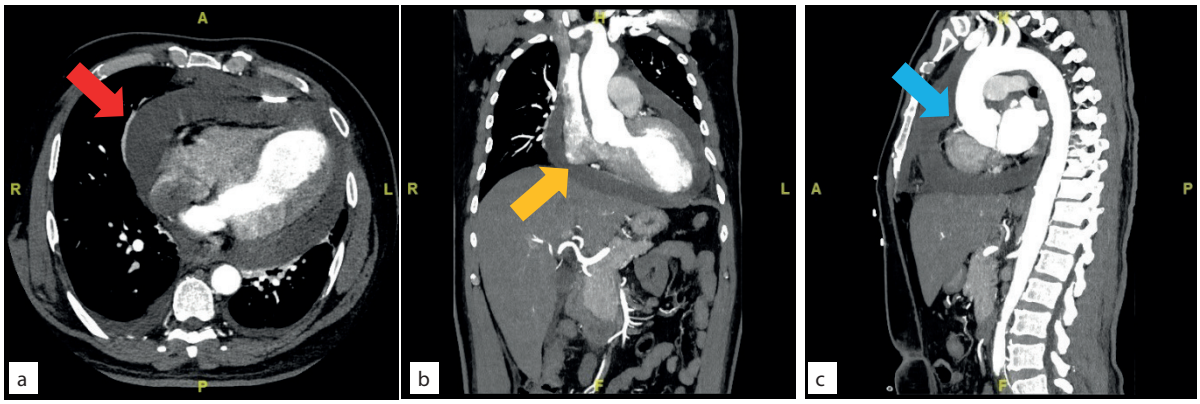

Figure 3. Chest computed tomography angiography (CTA): (a) Axial view, (b) Coronal view, and (c) Sagittal view confirmed the presence of a large pericardial effusion measuring 2.8 cm in maximum thickness (red arrow), along with the collapse of the right ventricle relative to the left ventricle (orange arrow), suggestive of the tamponade effect. There is no evidence of aortic aneurysm or dissection (blue arrow).

Discussion

Dressler syndrome is a form of pericarditis thought to be auto-immune-associated, triggered by cardiac tissue injury following myocardial infarction (1, 3).It typically occurs 2–6 weeks after the infarction(1). Other conditions linked to cardiac injury, such as trauma, post-pericardiotomy, and iatrogenic causes from cardiac procedures, can also drive autoimmune-mediated pericardial inflammation (3). In this case, the patient's recent NSTEMI and angioplasty procedure resulted in Dressler syndrome.

Dressler syndrome is characterized by pericarditis, pleuritis, and pneumonitis(1). On the other hand, the classic triad of chest pain, pericardial effusion, and ECG changes (diffuse ST elevation and PR depression) is not very sensitive, which is present in a minority of patients(4). The patient presented with severe epigastric pain radiating to both shoulders and a syncopal episode, underscoring the variability in myocardial infarction symptoms, which can sometimes mimic gastrointestinal or other non-cardiac conditions, complicating the diagnosis.

Sinus tachycardia, old ischemic changes (Q wave, ST elevation, and T inversion), and low voltage in limb lead on the ECG, suggesting cardiac tamponade. Autonomic sympathetic arousal, secondary to reducing cardiac output, is the cause of tachycardia. ST-segment elevation could be a non-specific finding in cardiac tamponade (5). Despite the initial ED referral for an acute myocardial infarction, the absence of a significant rise in troponin levels and the absence of new left ventricle wall motion abnormalities excluded this diagnosis. Low QRS voltage is due to heart position changes, increased distance from

the current generator to the recording electrodes, and reduced cardiac chamber size and volume (5). Classic pericarditis ECG changes are absent in this case, a finding not uncommon and present in only 20–24% of cases (4).

Dressler syndrome can cause pericardial effusion. Accumulation of pericardial fluid can lead to cardiac tamponade, characterized by hemodynamic instability due to impaired diastolic filling and reduced cardiac output. If the accumulation occurs rapidly, even small amounts (200–300 ml) of pericardial fluid can cause compressive effects(6). In contrast, if the accumulation is slow, these symptoms might not be evident, and the heart may compensate until 1-2 litres of fluid are present before becoming clinically symptomatic (6).The classic triad of hypotension, tachycardia, and muffled heart sounds, along with bedside cardiac ultrasound findings of massive pericardial effusion and right ventricular diastolic collapse, suggest cardiac tamponade. These classic symptoms may be absent in subacute or chronic pericardial effusions(6).

Cardiac tamponade is a clinical diagnosis based on a combination of a suggestive history, unstable vital signs, and physical examinations. The patient's blood pressure was relatively hypotensive (in obstructive shock), considering he was chronically hypertensive, which emphasizes the significance of bedside ultrasound in the rapid assessment and diagnosis of hemodynamically unstable patients in an emergency setting. In cardiac tamponade, echocardiography shows pericardial effusion, right ventricular compression during diastole, abnormal respiratory variation in both ventricular dimensions and tricuspid and mitral valve flow velocities with a plethoric inferior vena cava (6, 7). In this case, the patient exhibited hemodynamic instability, and echocardiography showed massive pericardial effusion with right ventricular collapse during the diastolic phase, confirming the diagnosis of cardiac tamponade.

The primary treatment for cardiac tamponade is pericardiocentesis, which involves draining the accumulated pericardial fluid to decompress the tamponade effect. Advances in medical technology now perform pericardiocentesis under real-time echocardiographic guidance, enabling precise detection of the largest pericardial effusion area, optimal needle insertion location, and prevention of accidental puncture of vital structures(8). In this case, drainage of 100 cc of fluid successfully decompressed the tamponade effect of the surrounding pericardial fluid, as evidenced by the stabilization of the patient's vital signs. The haemorrhagic nature of the fluid and the exclusion of infection and malignancy from pericardial fluid analysis pointed towards an inflammatory aetiology consistent with Dressler syndrome. Additionally, chest CTA is particularly useful to determine the causes of pericardial effusion and cardiac tamponade. The absence of CT evidence of aortic aneurysm or dissection, along with recent myocardial infarction evident by coronary angiography, confirmed the diagnosis and assured further optimization of management.

Following stabilization, the patient was managed with anti-inflammatory therapy to address the underlying autoimmune pericarditis. Aspirin, preferred for its dual anti-inflammatory and anti-platelet role, was administered, and colchicine was added for its synergistic effect and to reduce the risk of recurrent pericarditis (9). The patient's stable course over a two-week hospitalization and gradual reduction in pericardial effusion reflected the effectiveness of this treatment approach. Warfarin was also initiated, given the evidence of a left ventricular thrombus in the apex, as recommended by the American Heart Association (10).The patient's outpatient follow-up with aspirin and warfarin aims to prevent recurrent pericardial effusion and thromboembolic events.

Conclusion

Clinicians should maintain a high index of suspicion for cardiac tamponade in post-MI patients presenting with hypotension and other signs of hemodynamic instability. Rapid bedside echocardiography can be a crucial tool in diagnosing this condition. Prompt recognition and emergent pericardiocentesis are life-saving.

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Conflict of Interest statement
None declared.

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