Early Diagnosis and Management of Acute Pericarditis in a patient with G6PD deficiency: A Case Report

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Abstract

Chest pain is a common presentation in clinical practice and needs immediate intervention. It can have several differentials, and it is vital to recognise the signs and symptoms early, differentiate it from other important causes and initiate appropriate management in a timely manner.

Here we present a case of acute chest pain in a young male, following a viral upper respiratory tract infection, which was later diagnosed as acute myopericarditis. Prompt investigations, early referral and treatment allowed the patient to make a full uneventful recovery and prevent adverse complications.

Keywords: Acute pericarditis, myopericarditis, pleuritic chest pain, G6PD deficiency, colchicine therapy.

Case presentation

A 30-year-old male patient with a pre-existing history of G6PD deficiency and asthma presented to a general practitioner with a two-day history of acute, central, pleuritic chest pain. The patient had recently completed a four-day course of antibiotics for an upper respiratory tract infection. He denied experiencing haemoptysis or fever. The chest pain was exacerbated by supine positioning and alleviated by leaning forward. Vital signs were stable, including temperature. Physical examination revealed muffled heart sounds. Chest X-ray was unremarkable excluding consolidation or other abnormalities (Figure 1). ECG demonstrated global ST elevation, suggesting acute pericarditis (Figure 2). Given the clinical presentation and ECG findings, the patient was promptly referred to the emergency department for further evaluation. Laboratory investigations revealed elevated C-reactive protein at 119mg/L and troponin T at 445 ng/L. The echocardiogram demonstrated preserved left ventricular systolic function (Figure 3). Subsequent cardiac magnetic resonance imaging (MRI) confirmed findings consistent with acute myopericarditis (Figure 4). The patient was initiated on non-steroidal anti-inflammatory drugs and colchicine therapy and discharged from the hospital. However, colchicine treatment was discontinued after six weeks due to adverse effects on liver function tests. Following up in the cardiology clinic, the patient made a full recovery.

Background

Acute pericarditis is the predominant inflammatory heart condition over acute myocarditis and infective endocarditis (1). Acute pericarditis occurs in about 27.7 cases per 100,000 people annually in the general population, while hospital admissions for pericarditis are estimated at 3.32 cases per 100,000 person-years. It is more common in young males (1). Pericardium is a double layered fibro-elastic sac providing a cover around the heart. It consists of 2 layers: a visceral layer around the epicardium and a richly innervated parietal layer. These are separated by a potential space containing around 15-50ml of serous fluid. Pericarditis is the inflammation of the pericardial sac. It is the most common diseases involving the pericardium. It is classified as acute, subacute, chronic or recurrent. Recurrent cases can occur in up to 30% of the cases (2). Inflammation in the pericardium can lead to accumulation of fluid leading to pericardial effusion. The fluid can be serous, haemorrhagic or purulent depending on the cause. If this pericardial effusion becomes large, it can cause pressure on the heart affecting diastolic filling, leading to cardiac tamponade, which is a life-threatening emergency. Pericardial thickening later can present as constrictive pericarditis (2).

Acute pericarditis has a multifactorial aetiology. It can be classified as infectious (attributed to around 80-85% cases) and non-infectious (attributed to around 15-20% of cases). Infectious causes include viruses like- Coxsackie virus, echovirus, herpes, influenza, adeno, HIV, HCV, parvo viruses, SARS-Co-V2 etc. Bacterial causes among infectious aetiology can include tuberculosis and rarely fungal or parasitic. Non-infectious causes can include neoplastic, metabolic, drug related- including vaccines (covid-19) (1,2).

Acute pericarditis often presents with sharp, pleuritic chest pain that worsens when lying down and improves with sitting forward, as supine position increases the pressure on the heart while sitting up relieves the pressure. Other nonspecific symptoms like fever, cough, shortness of breath maybe present indicating underlying cause, like in our case. On auscultation there is a pericardial friction rub, which can be explained by friction of the 2 inflamed layers of pericardium. This can be heard as a high-pitched scratchy or squeaky sound at the left sternal border. Basic investigations include ECG-which may show diffuse ST elevation, PR depression, bloods including inflammatory markers and cardiac troponins, a chest x-ray, echocardiogram and other cardiac imaging modalities like CT or cardiac MRI to assess myocardial inflammation in some cases. Other tests to investigate underlying diseases to find the secondary cause like QuantiFERON test for tuberculosis, viral serology for HIV, and autoimmune screening etc should be done (1,3).

According to ESC guidelines 2015, the diagnosis of acute pericarditis is made when 2 of the 4 criteria are met:

1) typical pleuritic chest pain
2) pericardial friction rub
3) widespread ST elevation or PR depression on ECG
4) pericardial effusion (4).

Most cases of pericarditis usually resolve within 4 weeks, but in cases where the symptoms persist longer than 4-6 weeks, it is called incessant pericarditis, whereas if the symptoms last more than 3 months, it is called chronic pericarditis. If there is a period of remission lasting more than 4-6 weeks in between episodes, it is called recurrent pericarditis (5).

Exercise avoidance is advised for patients, especially in athletes for a period of 3 months. Treatment involves using first line drugs like non-steroidal anti-inflammatory drugs and colchicine. Aspirin is given in doses of 750- 1000mg every 8 hours, while ibuprofen is given as 600mg every 8 hours for a period of 1-2 weeks. Colchicine is given as 0.5mg twice a day for 3 months. Colchicine use not only helps in remission but also reduces the risks of recurrence by more than half. Corticosteroids should be avoided, due to risk of viral replication and should be reserved for cases of idiopathic pericarditis, where there might be underlying autoimmune disorder. If patients do not respond to above medications, azathioprine, intravenous immunoglobulin and anakinra (an interleukin-1β antagonist) can be used. Surgical pericardiectomy is used when all above treatments fail, as a last resort, in those with history of cardiac surgery or with features of constriction (4,5).

Figure 1: Unremarkable chest X-ray findings

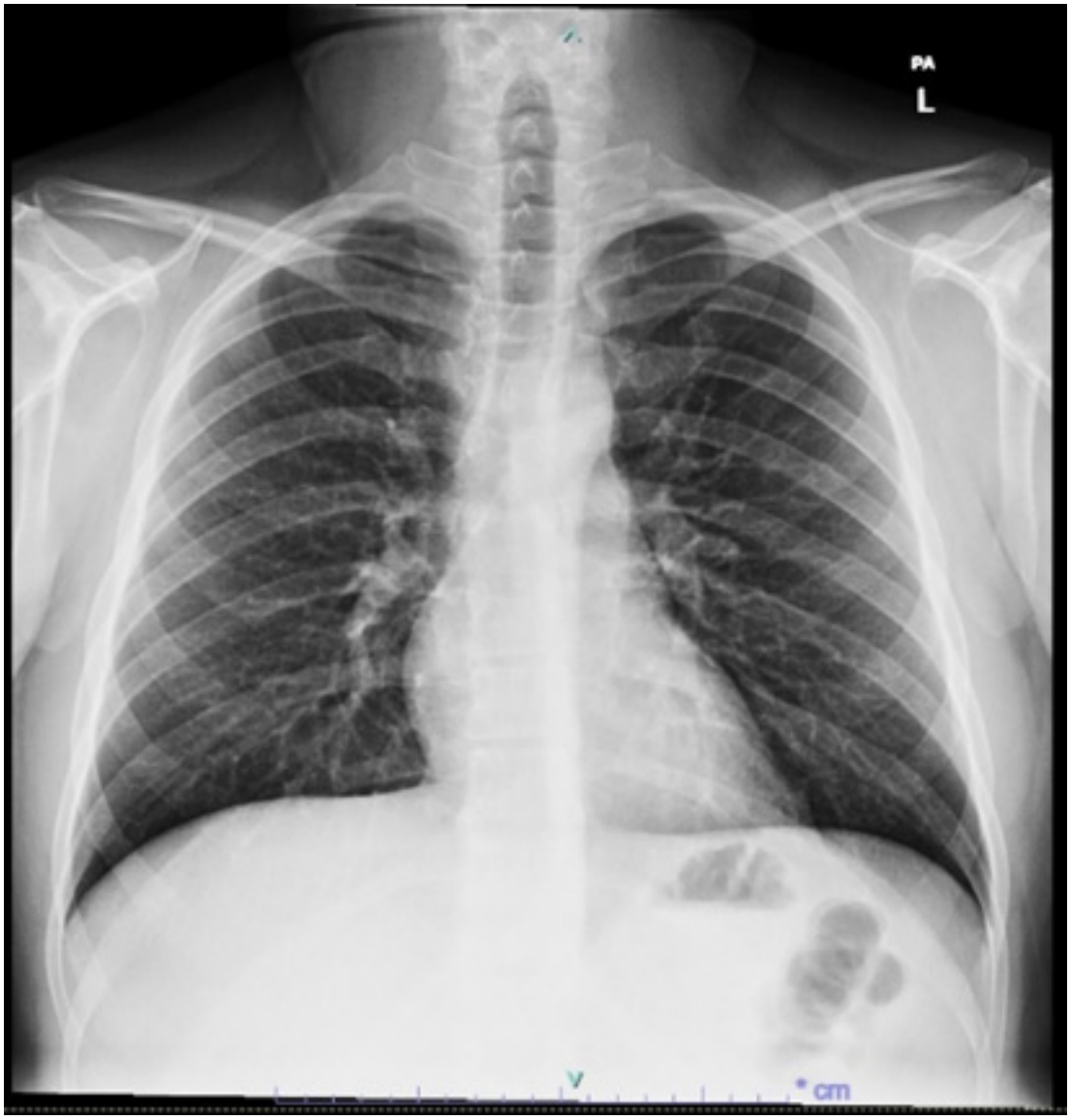


Figure 2: Global ST-segment elevation

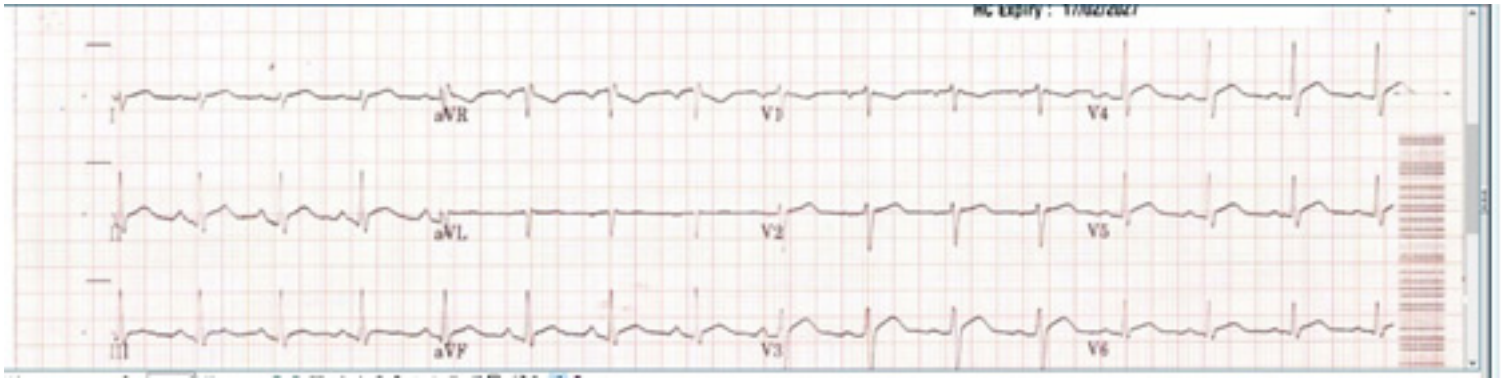


Figure 3: Unremarkable findings on ECHO

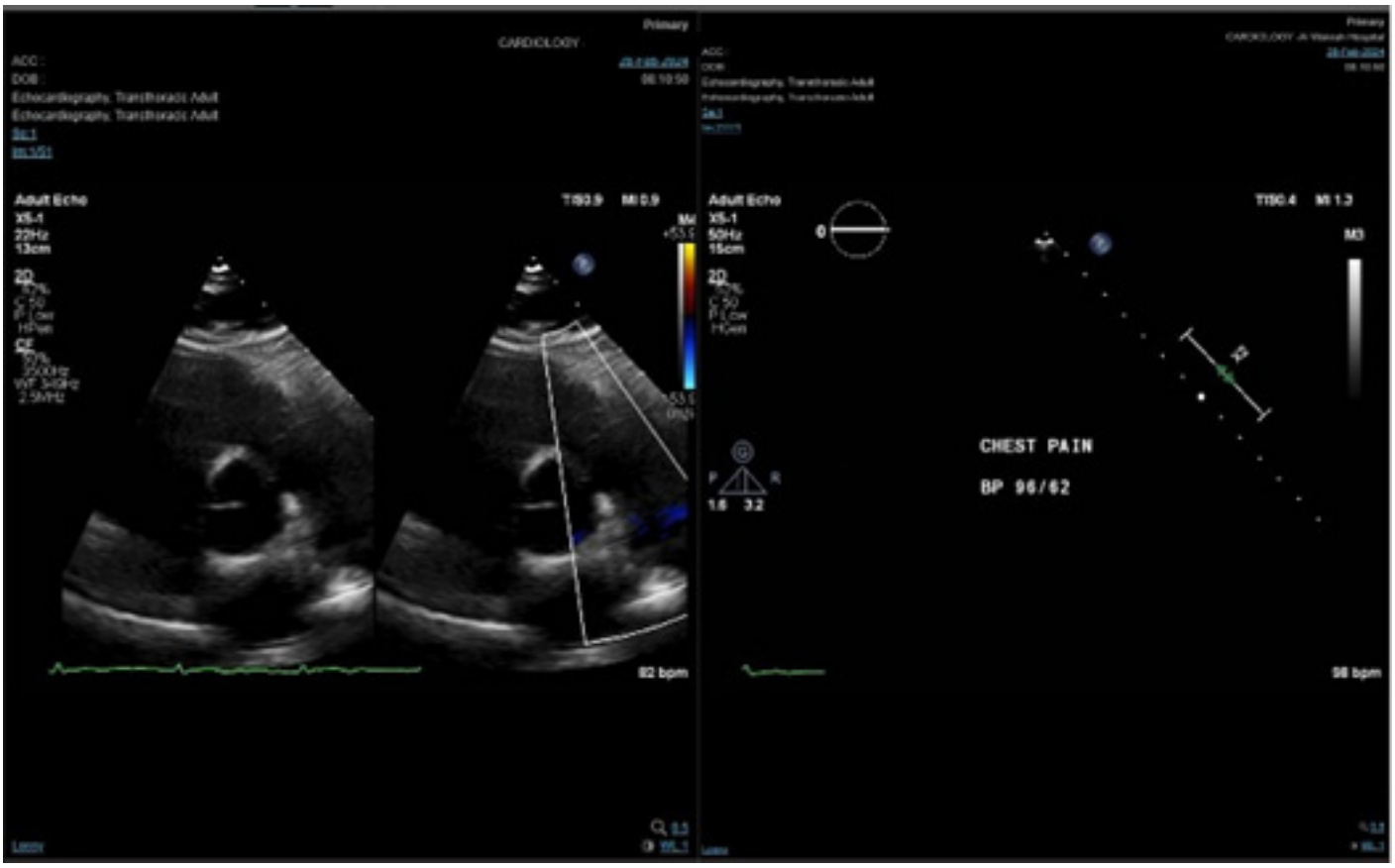
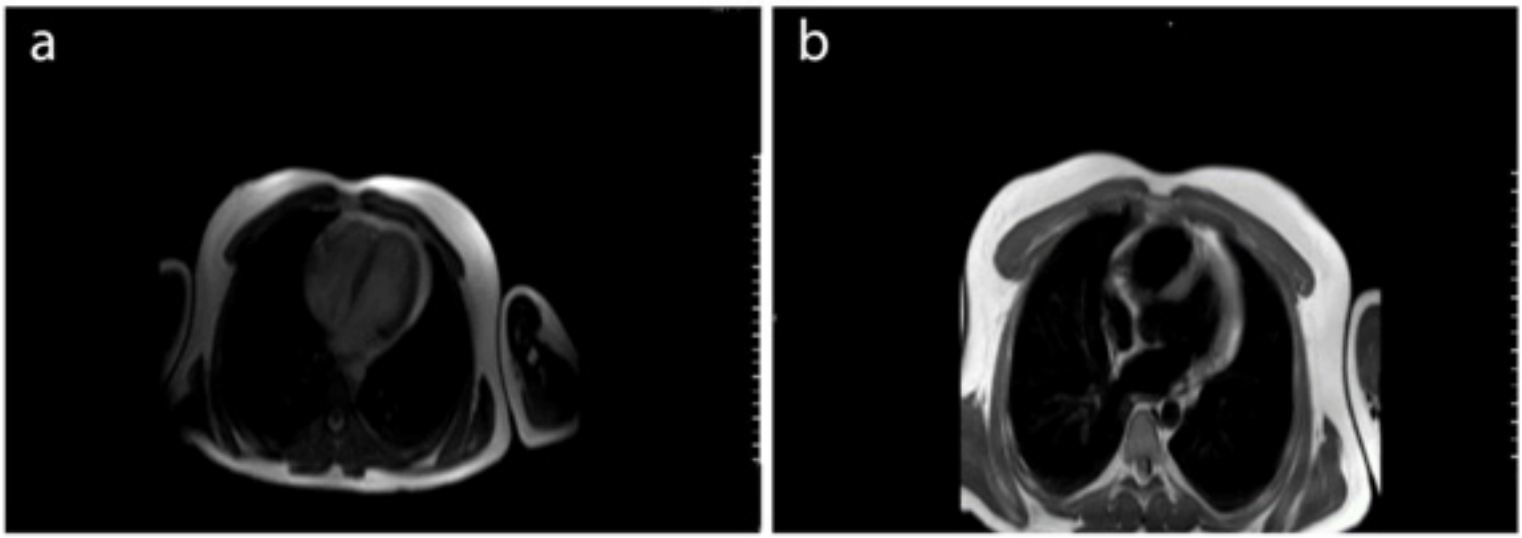


Figure 4 (a and b): CMR findings on MRI consistent with myopericarditis



Discussion

This case presents a classic scenario of acute pericarditis following an upper respiratory tract infection in a young male with underlying comorbidities of G6PD deficiency and asthma. While the exact aetiology remains elusive, the temporal relationship to the infection suggests a viral trigger, a common antecedent in acute pericarditis. The patient's presentation with pleuritic chest pain, ECG changes and elevated inflammatory markers is consistent with the typical clinical picture.

The exact pathophysiology of acute pericarditis is complex and often multifactorial. Aligning with the presented case it typically involves an initial insult, which triggers an inflammatory response. This insult can be viral, bacterial, autoimmune or even traumatic (6). The inflammatory process leads to the release of inflammatory mediators such as cytokines and chemokines, which recruit immune cells to the pericardium (7). The accumulation of these cells and the release of inflammatory substances results in pericardium thickening, pericardial effusion and potentially fibrin deposition (6). The increased pressure within the pericardial sac due to fluid accumulation can impair ventricular filling, leading to hemodynamic compromise in severe cases (8). Diagnosis of myopericarditis is made when there is myocardial involvement in cases of acute pericarditis, as evidenced by raised cardiac enzymes. It has presentation ranging from mild self-limiting disease to symptoms of heart failure, arrhythmias, syncope and even cardiac arrest. Chest x-ray and echocardiogram will point to any underlying heart failure or ventricular dysfunction. Cardiac MRI is used to assess degree of myocardial or pericardial involvement (4,9).

The patient in our case study, had minor myocardial involvement, with raised cardiac troponins. His chest x-ray was unremarkable, and echocardiogram showed preserved left ventricular systolic function. Cardiac MRI showed LV apical subepicardial oedema, hyperaemia and late enhancement, consistent with myocarditis thus pointing to a diagnosis of myopericarditis.

Excluding acute coronary syndrome can be a bit challenging yet vital, if a similar case presents in an older patient with multiple cardiac risk factors. In such instances, coronary angiography is usually essential (1,9).

The presence of elevated troponin T in this case is noteworthy. While typically associated with myocardial injury, it can also be elevated in acute pericarditis, particularly in cases of myopericarditis. This finding aligns with the study conducted by Imazio et al., (10) which investigated the role of cardiac troponin I (cTnI) in acute pericarditis. Their study found that cTnI elevation is common in this condition, often associated with younger age, male sex, ST-segment elevation and pericardial effusion. This finding emphasizes the importance of considering a broader differential diagnosis when evaluating patients with chest pain and elevated cardiac biomarkers (10). An RCT study by M Imazio et al., investigated the effectiveness of colchicine, when given along with conventional anti-inflammatory medications, after a first episode of acute pericarditis. The results suggested reduced rate of incessant or recurrent pericarditis as compared to placebo (11).

The development of liver dysfunction necessitated the discontinuation of the medication in our case. This highlights the importance of close monitoring for adverse effects. While most acute pericarditis cases resolve spontaneously, complications such as pericardial effusion, cardiac tamponade and constrictive pericarditis can occur (1). Fortunately, our patient did not develop any complications and made a full recovery.

Considering, the due order for occurrence in the presented case, early diagnosis and timely management serve as a cornerstone for a complication-free completed recovery in patients with acute pericarditis.

Conclusion

This case highlights the importance of considering acute pericarditis or myocarditis in a young patient presenting with chest pain following a recent viral infection. Taking a focused history, recognizing clinical signs early on and ordering and interpreting appropriate investigations in timely manner helped initiate treatment early and prevent complications. Newer modalities like cardiac MRI are very sensitive in diagnosing myocardial involvement in myopericarditis and help in narrowing down differentials in an acute care setting.

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