Viral myopericarditis masquerading as acute coronary syndrome: Diagnostic role of cardiac MRI

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ABSTRACT
Myopericarditis is commonly misdiagnosed as an acute coronary syndrome (ACS) due to the similarities in clinical presentation. Hence, accurate diagnosis is important to ensure appropriate care and improve outcomes. Here, we present the case of a 58-year-old male initially diagnosed with ACS, which was eventually identified to be a case of myopericarditis and managed accordingly. The patient presented with fever for 1 week during the coronavirus disease pandemic and developed signs of ACS subsequently. Clinical history, biomarker profile, and echocardiogram suggested ACS. However, cardiac magnetic resonance imaging and coronary angiography findings revealed myopericarditis and it was managed accordingly. Clinicians should be aware of myopericarditis masquerading as ACS in the post-viral period.

Key words: Acute coronary syndrome, Myopericarditis, Coronavirus disease 2019

CASE REPORT
A 58-year-old male with a history of hypertension and diabetes (for the past 5 years), presented to a peripheral hospital with complaints of fever, cough, and throat pain for the past week. In-hospital vitals showed a pulse rate of 120/min, blood pressure of 110/80, and temperature of 38.8°C. General examination showed conjunctival congestion with an oxygen saturation of 96%.

Blood tests confirmed that the fever was not related to coronavirus disease (COVID-19), and he was hospitalized for further treatment. He subsequently developed chest pain, sweating, and left-hand pain. Electrocardiography revealed ST elevation in inferior leads and T-wave depression in V4-V6, indicating inferior wall ST-elevation MI.

The patient was then shifted to a tertiary care hospital, where a reverse transcription-polymerase chain reaction test indicated no contraction of COVID-19. Further, the chest X-ray exhibited woolly and fluffy patches in the right lung. Computed tomography of the chest suggested pulmonary edema and achalasia cardia. On assessment, he was marked as COVID-19 reporting and data system category 3. The troponin-I levels were elevated, marking to 4820 units, which was a clear warning of ACS. The echocardiogram displayed wall motion abnormalities which confirmed inferior wall (lateral) MI.

A primary diagnosis of ACS was made, and low molecular weight heparin, nitrates, dual antiplatelet drugs, and statins were administered. However, cardiac magnetic resonance imaging and coronary angiography findings revealed myopericarditis and it was managed accordingly. Myopericarditis often masquerades as an acute coronary syndrome (ACS), due to the common clinical manifestations such as angina pain or even cardiogenic shock and similarities in electrocardiogram (ECG) or echocardiogram changes, along with variations in serum biomarker levels [1]. Although ACS may be attributed to the presence of acute thrombosis or severe obstructive, atherosclerotic disease, which are evident in coronary angiography (CAG) in most cases, this criterion may not be met in a large minority of cases [2]. Similar observations have been made in patients with myocardial infarction (MI) with non-obstructive coronary arteries, who exhibit clinical evidence of MI, but CAG does not reveal any obstructions [3]. Further, disorders such as small vessel disease, coronary embolism, hematologic disease leading to in situ thrombosis, and congenital anomalies can also cause acute MI in the presence of healthy coronary arteries or without significant stenosis [2]. Hence, it is vital to accurately diagnose patients presenting with signs of ACS and manage them appropriately to prevent further complications.

Here, we present a noteworthy case of a middle-aged man who initially presented with febrile illness and was diagnosed with ACS, which was eventually detected to be a case of myopericarditis and managed accordingly.
administered to the patient. Once the patient was stabilized, CAG was performed as an additional diagnostic investigation, which illustrated normal coronary blood vessels and eliminated the possibility of ACS. Laboratory reports such as elevated levels of D dimer and activated partial thromboplastin clotting time (APTT) were suggestive of myocardial injury. Throat swab analysis revealed the presence of the influenza virus and validated the cause of viral fever, precipitating into ACS due to plaque rupture (Table 1).

Cardiac magnetic resonance imaging (MRI) depicted the presence of inflammatory infiltrates, edema, and scar in the posterior part of the epicardial portion of the inferior wall, along with thin pericardial effusion (Fig. 1). Based on the detailed investigation, a final diagnosis of viral myopericarditis was made and the therapeutic approach was accordingly modified to include steroids, proton pump blockers, coenzyme Q, carnitine, and low doses of diuretics. Symptomatic relief was subsequently noted, and the patient was discharged. Follow-up after 3 months revealed improvement in the left ventricular function (ejection fraction: 44%). At 6 months follow-up, the ejection fraction had improved to 58%.

DISCUSSION

One of the most common etiological factors of myopericarditis is the presence of viral infection [1]. Influenza has been suggested to be a triggering factor for numerous cardiovascular events including acute MI, myocarditis, ventricular arrhythmia, and heart failure. As part of the host immune response, the influenza virus can trigger numerous proinflammatory cytokines and chemokines and subsequent recruitment of immune cells [4]. Preexisting systemic inflammatory responses, along with direct effects of the influenza viral infection on the endothelial cells or atherosclerotic plaques, may result in the progression of atherosclerosis or plaque rupture, subsequently leading to acute coronary events [4].

Early symptoms of myopericarditis include fatigue, precordial chest pain, palpitations, dyspnea, and fever. In case of substantial myocardial involvement, pain may be continuous, and it may be difficult to differentiate it from myocardial ischemia, especially in patients with cardiovascular risk factors [5]. Further, the cardiac involvement may be localized, intramyocardial, and/or epicardial and not transmural. Echocardiography may often be unable to demonstrate these alterations [2]. The changes may also be non-specific and hence not clearly useful to differentiate acute MI from myocarditis [6]. Cardiac MRI is considered a vital diagnostic tool in such patients as it can help evaluate the degree of pericardial and myocardial involvement. While subendocardial or transmural myocardial enhancement in one arterial territory may be noted in patients with ACS, subepicardial or mid-myocardial inflammatory changes, along with myocardial edema involving different vascular territories, are common findings in myopericarditis [5].

In the present study, cardiac MRI illustrated certain distinctive characteristics such as inflammatory infiltrates, edema, scar in the inferior wall, and thin pericardial effusion, which facilitated differentiation between acute myopericarditis and ACS. A few other similar studies have also highlighted the benefits of cardiac MRI in differentiating cases such as stress-induced cardiomyopathy [7], acute viral myocarditis [8], and dilated myocarditis [9] which masqueraded as ACS. Hence, it is necessary for practicing cardiologists to transform the traditional ways of diagnosis and make cardiac MRI a part of the regular protocol for all the patients who present with an initial febrile illness followed by MI, despite the risk factors associated with this modality.

Table 1: Diagnostic tests for infection and cardiac injury

<table>
<thead>
<tr>
<th>Diagnostic test</th>
<th>Result</th>
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<tbody>
<tr>
<td>Throat swab analysis</td>
<td>Influenza A H3 and respiratory syncytial virus were detected</td>
</tr>
<tr>
<td>D-Dimer</td>
<td>360 ng/mL</td>
</tr>
<tr>
<td>Troponin</td>
<td>4820 pg/mL</td>
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<tr>
<td>Activated partial thromboplastin clotting time (APTT)</td>
<td>45.9 s</td>
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Figure 1: Cardiac magnetic resonance imaging showing inflammatory infiltrates, edema, and scar. (a) T2-weighted four-chamber view; (b) late gadolinium enhancement – four-chamber view; (c) Late gadolinium enhancement – short axis view
CONCLUSION

It is important for physicians to note that myopericarditis can masquerade as ACS in the post-viral period. Imaging modalities such as MRI play a crucial role in the precise diagnosis of myopericarditis, while other diagnostic approaches such as measurement of serum biomarkers, ECG, and echocardiogram may not provide conclusive evidence. Hence, it is vital for clinicians to have a keen eye for the details and utilize non-invasive imaging modalities such as MRI for the early diagnosis and effective management of these conditions.

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REFERENCES


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