Case Report

Acute myocarditis masquerading as anterior wall myocardial infarction: A case report

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ABSTRACT

Introduction: Myocarditis is the inflammation of the myocardium. The clinical presentation of myocarditis ranges from asymptomatic state to acute heart failure which may mimic acute coronary events. Because of the similar presentation of chest pain, elevated cardiac enzymes, and electrocardiographic (ECG) changes, acute myocarditis may rarely masquerade as acute myocardial infarction (AMI). High suspicion is often necessary for treating such patients, keeping in mind the different treatment protocols required for managing each pathology.

Methods: SCARE 2020 Guidelines.

Case report: We report a case of a young female presenting with sudden onset chest pain with ECG changes suggestive of acute anterior wall myocardial infarction (AWMI). Further investigations ruled out AWMI, and she was diagnosed with acute myocarditis. She was treated for myocarditis and showed a prompt recovery.

Discussion: Acute myocarditis may present clinically similarly to ACS; hence a high level of suspicion is required to differentiate the two entities. The absence of significant cardiovascular risk factors for atherosclerotic coronary artery disease, absence of RWMA, and normal coronary angiogram are further suggestive of myocarditis.

1. Introduction

Myocarditis is the inflammation of the myocardium. It can occur secondary to various etiologies like viral infections, autoimmune disorders, myocardial toxins, radiation injury, hypersensitivity reactions, or even idiopathic phenomena. Acute myocarditis may rarely masquerade as AMI because of similar clinical presentation, ECG changes and raised cardiac enzymes. The absence of significant coronary artery atherosclerotic risk factors and a history of inciting events suggest myocarditis instead of ACS. The lack of regional wall motion abnormality (RWMA) on two-dimensional echocardiography (2D-Echo) and normal coronaries on angiography suggests against the diagnosis of ACS. Serological evidence of viral pathology may be found in many cases. The definite diagnosis by endomyocardial biopsy is rarely performed. We report a case of a young female presenting with typical clinical and ECG manifestations of AWMI that later got ruled out on further evaluation. She was diagnosed with acute myocarditis and treated for the same.

2. Case report

A 33-year-old female presented with sudden onset retrosternal chest pain radiating to the left arm. Her blood pressure was 100/74 mm Hg, pulse rate was 98/minute. On auscultation, S1 S2 were present with no murmur. The chest was bilaterally clear. ECG showed ST-segment elevation in lead V2-V5, I, and aVL with reciprocal depression in lead II, III, and aVF (Fig. 1). 2D-Echo revealed left ventricular ejection fraction (LVEF) of 45% but no RWMA. There was minimal pericardial effusion. Her troponin I was 5.77 ng/ml (reference range 0.00–0.30 ng/ml) and NT-proBNP was over 25,000 pg/ml (reference range 0.00–125 pg/ml). Her coronary angiography revealed normal epicardial coronaries. Serial ECGs showed persistent ST-segment elevation, intermittent junctional rhythm, and frequent ventricular premature complexes (VPC). There was no history of prior drug intake or any chronic illness. Serological diagnosis for adenovirus, coxsackievirus B, parvovirus B19, human herpesvirus 6, Epstein-barr virus, HIV, hepatitis B and C were negative. RT-PCR for COVID-19 was negative. For further evaluation, the patient was advised cardiac magnetic resonance imaging (CMR).
which she declined due to financial constraints. We (cardiologist) diagnosed her with idiopathic myocarditis. Along with the supportive therapy, she was treated with intravenous methylprednisolone, to which she showed dramatic improvement. The ECG changes settled by day seven of steroid therapy, and LVEF improved to 55% (Fig. 2). At follow-up of one year, she remained asymptomatic, and her review 2D-Echo revealed normal left ventricular (LV) functions with LVEF of 65%.

3. Discussion

Myocarditis is the inflammation of the myocardium. It is generally mild and self-limiting pathology but may have a grave prognosis leading to inflammatory cardiomyopathy. The prognosis of inflammatory cardiomyopathy is poor in patients with LV dysfunction and heart failure [1]. Myocarditis may present with a fulminant course characterised by sudden onset severe LV dysfunction leading to cardiogenic shock or arrhythmias. Fulminant myocarditis should be considered among the top differential diagnoses in young patients with cardiogenic shock [2,3]. When treated promptly by early diagnosis and specific treatment strategies, myocarditis can have a good prognosis. Infectious agents mainly induce myocarditis, most commonly viral agents but can also be by bacteria, e.g., Borrelia, protozoa like Trypanosoma cruzi or fungi. Toxins, drugs or immune-mediated illnesses can also induce myocarditis. The most common viruses linked to myocarditis include adenovirus, enterovirus, parvovirus b19, human herpes virus 6, Ebstein bar virus, cytomegalovirus, HIV, hepatitis C, influenza virus and recently severe acute respiratory syndrome Coronavirus (SARS-CoV) [4].

The clinical presentation of a myocarditis patient may vary from mild symptoms to frank heart failure or sudden cardiac death. The patient may present with chest pain, fatigue, dyspnea, palpitations or syncope [2]. Myocarditis is the cause of sudden cardiac death (SCD) in 10% of cases of SCD in young patients less than 35 years old [5]. Careful history taking can elicit a history of prodromal events like fever, flu-like symptoms, and gastrointestinal upset in up to 80% of patient’s weeks before presentation with myocarditis [1]. Our patient presented with typical clinical symptoms of ACS. ECG changes and raised cardiac enzymes directed towards a diagnosis of AMI. However, the absence of coronary artery atherosclerotic risk factors and absence of RWMA on echo made us think of the possibility of myocarditis masquerading as acute MI. Prior cases of myocarditis presenting as acute MI have been reported.

Hou et al. reported case of middle-aged male patient presenting with chest pain and raised cardiac enzymes after a flu like illness. Viral serology revealed high titers of rubella immunoglobulin and late gadolinium enhancement (LGE) on CMR. The patient showed significant improvement with anti-viral and supportive therapy [6]. A study of 45 patients suspected of acute MI with normal coronary angiogram was undertaken to assess a myocarditis diagnosis. 35 out of 45 patients showed either diffuse or focal myocarditis on myocardial indium-111 antimyosin antibody or Thallium-201 imaging [7]. Another case series of 21 patients with acute myocarditis mimicking the AMI was reported. All patients had raised cardiac enzymes and ECG changes of acute MI but normal coronary angiograms. All except two patients remained asymptomatic at long-term follow-up [6].

Our patient was negative for viral serology. Careful history taking did not reveal any flu-like illness. No history of any toxin or drug intake was found. We diagnosed her with a case of myocarditis of idiopathic origin. Along with supportive therapy she was treated with intravenous methylprednisolone 1mg/kg body weight for a week followed by oral prednisolone 1mg/kg therapy to which she showed dramatic improvement. Her symptoms reverted to normal at the seven of starting steroids.

Prior studies have shown that the long-term prognosis of myocarditis is better in patients with mild clinical presentation than those with LV dysfunction or heart failure. In a multicentre registry of 443 patients, it was observed that serious adverse events of SCD or heart transplantation at five year was 14.7% in case of complicated myocarditis while it was 0% in uncomplicated myocarditis [1].

CMR play an important role in the risk stratification of patients with myocarditis and preserved LVEF. The pattern of LGE on CMR provides important prognostic information. In the ITAMY study, it was found that LGE in mid wall layer of anteroseptal segment is associated with a worse prognosis and was the best and independent predictor of SCD, appropriate ICD firing, resuscitated cardiac arrest and hospitalisation for heart failure [9]. A prospective study on 672 patients with suspected myocarditis showed that tissue characterisation provides effective risk stratification in patients with suspected myocarditis. The presence of LGE was associated with more than doubling the risk of MACE, i.e. 4.8% versus 2.1% annual MACE rate when LGE was present and absent, respectively. The same study showed that septal and mid-wall LGE had the strongest association with MACE [10]. Acute myocarditis may present clinically similarly to ACS; hence, a high suspicion level is required.

![Figure 1](image-url). Twelve lead ECG showing ST-segment elevation in lead V2-V5,I and aVL with reciprocal depression in lead II, III, and aVF.
to differentiate the two entities. Because of the potential to develop inflammatory cardiomyopathy and its serious consequences of SCD, arrhythmia, and heart failure, acute myocarditis should be properly treated. Patients should be investigated to look for causative factors and treated accordingly. CMR is the gold standard non-invasive modality of choice for diagnosing myocarditis. It is a class one recommendation for the characterisation of myocardial tissue for the diagnosis of myocarditis [11]. The ability to recognise myocarditis in patients presumed to be acute MI is invaluable because when treated timely, the prognosis in such patients can be good. Finally, this report has been made following SCARE guidelines [12].

4. Conclusion

Acute myocarditis may present clinically similarly to ACS; hence a high level of suspicion is required to differentiate the two entities. A history of prodromal symptoms can be elicited in up to 80% of patients, which may be an important clue in differentiating myocarditis from ACS. The absence of significant cardiovascular risk factors for atherosclerotic coronary artery disease, absence of RWMA, and normal coronary angiogram are further suggestive of myocarditis. CMR plays an important role in the diagnosis and prognostication of myocarditis. Prompt treatment can impart a good prognosis with no residual LV dysfunction.

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SV, AY, PS has written the first draft. Final draft preparation and proofreading has been performed by Shazia khan and Vikash Jaiswal.

Registration of research studies

1 Name of the registry: NA
2 Unique Identifying number or registration ID: NA
3 Hyperlink to your specific registration (must be publicly accessible and will be checked): NA

Consent

A written informed consent has been taken from the patient for the case and images. A copy of it will be made available upon reasonable request from corresponding author.

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Declaration of competing interest

None declared by any authors involved in the manuscript.

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