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Case Report

Myocarditis Mimicking ST-Elevation Myocardial Infarction: A Case Report

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Abstract

Background: Myocarditis is the inflammation of the heart muscle, which affects mostly the young population. Patients may experience symptoms ranging from chest pain, fatigue, dyspnea, to more serious conditions such as heart failure and arrhythmias. Diagnosis of myocarditis may be quite confusing as it may mimic myocardial infarction, particularly when ST-elevation is present on electrocardiogram (ECG). Here, we present a challenging presentation of myocarditis mimicking ST-segment elevation myocardial infarction (STEMI).

Case Report: A 54-year-old man admitted to the emergency department due to severe chest pain of two hours duration. Lab tests revealed high levels of troponin and D-dimer. ECG revealed tachycardia and ST-elevation in the anterolateral leads, making the diagnosis towards STEMI very likely. However, coronary angiogram didn't show any obstruction in the right or left coronary arteries. Cardiac magnetic resonance (CMR) imaging was then performed and showed evidence of myocarditis. The patient was treated with colchicine and beta-blocker in which he showed a gradual recovery.

Conclusion: Our case highlights the difficulties that a physician may encounter when it comes to diagnosing a case of myocarditis. It should always be kept in mind when dealing with patients presenting with chest pain and ST-elevation, as it may mimic a typical STEMI presentation. A thorough clinical evaluation and the choice of appropriate diagnostic tools are key to the diagnosis of such challenging cases.

Keywords: STEMI, Acute myocardial injury, Myocarditis, CMR, Case report

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Background

Myocarditis is the inflammation of the heart muscle, affecting around 22 out of 100,000 patients per year [1,2]. The causes are variable and include infectious, drug-induced, immune mediated, and toxic causes [1]. Patients with myocarditis may present with variable cardiac and extracardiac symptoms, and sometimes mimicking myocardial infarction [3]. Here, we report a case of myocarditis that presented with ST-elevation and elevated troponin levels in the absence of any ischemic lesions of the coronary arteries on angiogram.

Case Presentation

A 54-year male patient-smoker (14 pack-year) diagnosed with coronary artery disease (treated with angioplasty), appeared with severe chest pain that had been going on for two hours. It was a dull pain left-sided, of sudden onset, non-radiating, fluctuating in severity, and not related to any position or effort. The patient had no associated nausea, vomiting, cough, dyspnea, or any other symptoms.

On physical examination, the patient had normal vital signs. The S1 and S2 on the cardiac assessment were normal and had no audible murmurs. The rest of the examination was unremarkable.

The patient was started on intravenous paracetamol 1g and subcutaneous morphine 2mg. The initial suspected diagnosis was STEMI. ECG was performed and demonstrated a sinus rhythm with a heart rate of 99 beats per minute (bpm) with no signs of ischemia. Lab tests revealed a slightly elevated c-reactive protein (30mg/L), elevated D-dimer levels (4858.55 ng/ml), and normal cardiac enzymes.

At this point, we suspected the presence of pulmonary embolism. Therefore, a computed tomography (CT) scan of the chest was planned. During the imaging procedure, the patient developed ventricular fibrillation (Figure 1A), where he was successfully resuscitated by cardiopulmonary resuscitation (CPR) and electrical cardioversion delivered twice.

ECG done afterwards revealed sinus tachycardia (104 bpm), and ST-elevation in leads V3-V6 compatible with acute anterolateral infarction (Figure 1B).

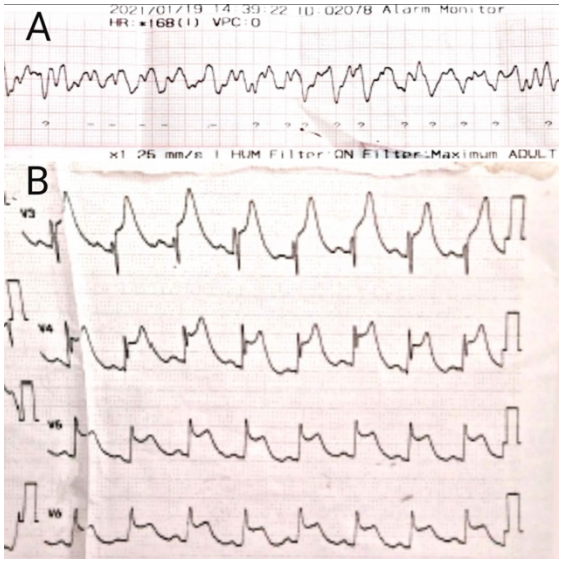

Figure 1: Electrocardiogram. A: Ventricular fibrillation. B: ST-elevation in leads V3-V6 compatible with acute anterolateral infarction

Cardiac enzymes were elevated with troponin t levels of 543.7 ng/ml. An urgent coronary angiogram was performed, but did not reveal any significant stenosis in the coronary arteries (Figures 2, 3A, 3B)

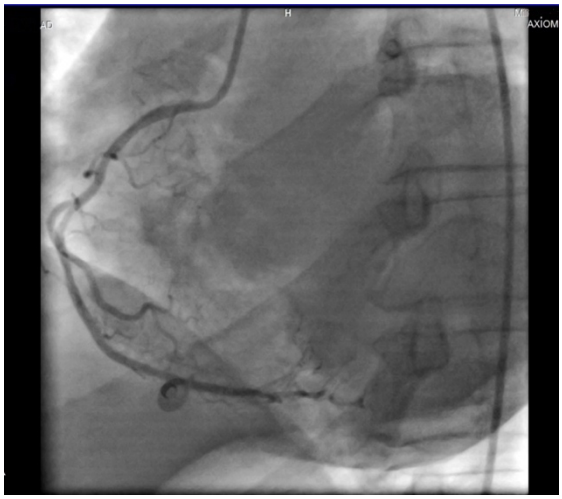

Figure 2: Coronary angiogram showing absence of stenosis in the right coronary artery

CMR imaging was done and showed subepicardial lateral apical delayed gadolinium enhancement in favor of myocarditis, and hyperintense signal of the lateral wall over dark blood T2 weighted signal in favor of myocardial edema. In addition to mid-anterolateral, latero-apical,

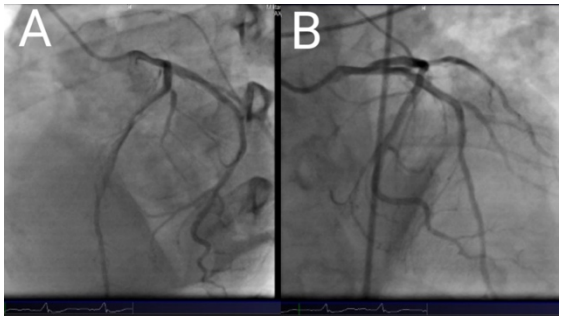

Figure 3: Coronary angiogram. A: absence of stenosis in the left anterior descending artery. B: absence of stenosis in the left circumflex artery.

and mid-inferior akinesis, with left ventricular ejection fraction (LVEF) of 47% (Figures 4A, 4B)

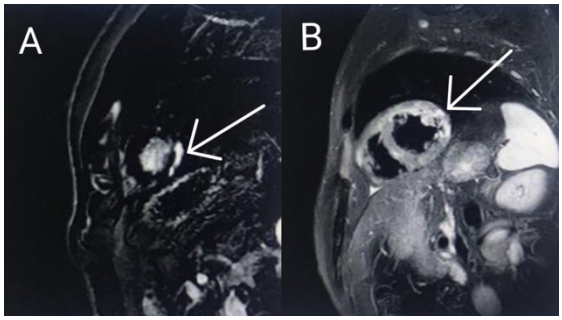

Figure 4: Cardiac magnetic resonance imaging. A: subepicardial lateral apical delayed gadolinium enhancement in favor of myocarditis. B: dark-blood T2-weighted imaging showing hyperintense signal of the lateral wall in favor of myocardial edema

The patient was treated with colchicine and beta-blocker. Follow-up one month later revealed a complete recovery of symptoms.

Discussion

Myocarditis is mostly caused by an infectious organism, predominantly a viral infection. Other potential etiologies include immune-mediated causes (such as systemic lupus erythematosus), toxic causes (such as drugs), and harm from electricity or ionizing radiation.

Patients with myocarditis may present with pain in chest, fatigue, shortness of breath, and cardiogenic shock [4]. Other non-specific respiratory and gastrointestinal symptoms are also commonly present [4]. In a study involving 3055 patients with suspected myocarditis, shortness of breath was the most frequent symptom showed in 72% of patients followed by pain in chest which is 32% and arrhythmias 18% [5].

Diagnosing myocarditis can be quite challenging, especially when its presentation resembles that of STEMI. Arrhythmogenic right ventricular cardiomyopathy, cardiac amyloidosis, and coronary heart disease are among the differential diagnoses [2]. At first, we suspected the presence of pulmonary embolism due to elevated D-dimer levels. Later on, the patient developed high levels of troponin, and ECG revealed ST-elevation, which made the diagnosis of STEMI very likely. However, the absence of obstruction of the coronary arteries revealed by the angiogram excluded such diagnosis.

Cardiac enzymes are usually elevated in myocarditis, with troponin being more commonly elevated than creatinine kinase MB [6]. Although troponin has a high specificity (89%), it has a low sensitivity (34%) [3]. Leucocytes, c-reactive protein, and erythrocyte sedimentation rate are a few more markers that could be increased [6].

ECG findings may reveal abnormalities such as ST-elevation, T-wave abnormalities, and pathologic Q waves [3]. Arrhythmias and heart block may also be present [3]. It has been demonstrated that arrhythmias occur in 18% of myocarditis patients [7]. However, ventricular tachy-cardia, which affects less than 5% of patients, is still a rare early symptom [7].

Myocardial infarction-like findings in myocarditis are supported by several experimental studies; coronary vasospasm following intracoronary acetylcholine provocation testing in the absence of coronary disease was present in 70% of patients diagnosed with clinical myocarditis suspicion and myocarditis confirmed by biopsy [8].

Coronary angiogram helps in narrowing our differential diagnosis as it is normal in myocarditis. 77% of patients with clinical suspicion for myocardial infarction but with normal coronary angiogram were found to have myocarditis on imaging studies [9].

CMR imaging is considered one of the valuable non-invasive tools for diagnosing

myocarditis. Myocarditis is proposed to be diagnosed using the Lake Louise criteria. through CMR imaging in the setting of clinically suspected myocarditis; two or three of the following must be present for the diagnosis: Edema is indicated by high myocardial signal intensity on T2-weighted images, and the ratio of early gadolinium enhancement between skeletal muscle and the heart is also raised on T1-weighted images showing increased blood flow and vasodilation, and increased late gadolinium at least one specific region of the myocardium has improved on T1-weighted images indicating scar tissue retaining contrast [10]. The CMR imaging done in our patient met the Lake Louise criteria.

The most accurate way to diagnose myocarditis is still with endomyocardial biopsy (EMB), being the only method for identifying the underlying etiology, and therefore helping in choosing a suitable treatment plan. However, there is a debate on whether a biopsy should be done routinely in patients diagnosed with myocarditis; unlike the American Heart Association (AHA), European Society of Cardiology (ESC) recommends the routine EMB for the identification of the causative virus [4].

There is no specific treatment for myocarditis. Patients with myocarditis associated with a de-creased LVEF can be treated with beta-blockers or angiotensin-converting enzyme inhibitors/angiotensin receptor blockers [6]. Antiviral treatment is also an option. However, patients frequently present in the chronic phases, and therefore the drug would not produce the optimal effect [6]. Immunosuppression is indicated in patients with non-viral myocarditis, yet a biopsy is required to exclude the presence of a virus [6]. Associated arrhythmias are managed with anti-arrhythmic drugs, and those refractory to the treatment can be referred to catheter ablation [4,11]. implantable cardioverter defibrillator (ICD) should be considered as a part of the long-term treatment after the patient's recovery [4]. In our case, since ventricular tachycardia was transient and the heart failure was well managed with medical treatment, the decision was made not to do an ICD implantation.

Conclusion

The diagnosis of myocarditis may be confusing, as it may mimic the typical presentation of STEMI. As presented in our case, there was high suspicion for STEMI. However, a normal coronary angiogram and CMR changes typical of myocarditis were key to a correct diagnosis. Myocarditis should always be considered by the physician while evaluating patients with chest pain and ST-elevation. A careful clinical assessment and the choice of appropriate diagnostic modalities are essential for the diagnosis and treatment of such cases.

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