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Case Report

Atypical perimyocarditis mimicking ST-segment elevation myocardial infarction: a case report

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Abstract: Perimyocarditis is an inflammation involving pericardium and myocardium with characterized widespread electrocardiography (ECG) ST-segment changes. However, with manifestations like chest discomfort, focal ST-segment elevation on ECG and elevated cardiac enzymes, it could be difficult to differentiate between perimyocarditis and ST-segment elevation myocardial infarction (STEMI). We here report a case of a 73-year-old male presenting atypical perimyocarditis with chest pain, focal ST elevation and increased cardiac markers, which led to emergency coronary angiography that was subsequently found to be normal. The following cardiac magnetic resonance (CMR) showed mild biventricular dysfunction and late gadolinium enhancement (LGE) imaging revealed contrast accumulation in subepicardial, intramyocardial and lateral wall areas. This case highlights that perimyocarditis may mimic myocardial infarction. Coronary angiographies and CMR are necessary to identify atypical perimyocarditis.

Keywords: Perimyocarditis, myocarditis, pericarditis, myocardial infarction, STEMI mimicking

Introduction

The mostly common electrocardiography (ECG) changes of myocarditis and pericarditis are diffuse, non-specific saddle-shaped ST-segment elevations. Perimyocarditis patients presenting with chest pain, elevated cardiac enzymes and focal ST elevation could mimic ST-segment elevation myocardial infarction (STEMI), which are difficult to be recognized [1]. Clinical differentiation between perimyocarditis and myocardial infarction is especially important because lethal effects can occur if reperfusion therapy is administered for a patient with acute perimyocarditis. Cardiac magnetic resonance (CMR) and late gadolinium enhancement (LGE) imaging have emerged as the useful modality in the noninvasive diagnosis of myocarditis due to their ability to detect myocardial edema, hyperemia, necrosis and fibrosis in a safe and reproducible fashion [2, 3]. We herein report a 73-year-old man presenting with chest discomfort, electrocardiographic changes and elevated cardiac enzymes simulated acute myocardial infarction.

Case report

A 73-year-old male was admitted to the emergency department with chest discomfort after a five-day prodrome of dizziness, fatigue, abdominal flatulence and three hours' vomiting. He did not complain of any radiating pain. Previous history showed no hypertension, hyperlipidemia and diabetes. He smoked almost 20 cigarettes per day for more than thirty years and quitted it up half a year ago.

Physical examination revealed normal vital signs and heart sounds with no evidence of pericardial friction rub. Auscultation of lungs showed harsh breath sounds and crackles in the lower lobes. The levels of his white blood cells (WBC) and cardiac biomarkers increased: WBC, 13.51 (normal range [NR] 3.5-9.5) G/L; troponin I, 21.725 (NR < 0.9) ng/mL; creatine kinase-MB, 71 (NR 0-24) U/L; and brain natriuretic peptide 4341 (NR < 100) pg/ml. His inflammatory markers were also elevated, with procalcitonin at 0.281 (NR < 0.05) ng/mL and C-reactive protein at 25.3 (NR < 5) mg/L. A

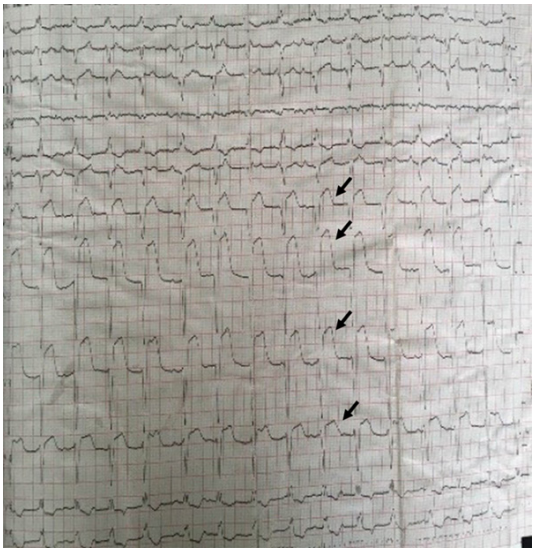

Figure 1. ECG showed sinus tachycardia, type 2 secondary degree AVB, 4-12 mm convex ST elevation (black arrows) in leads V1-4.

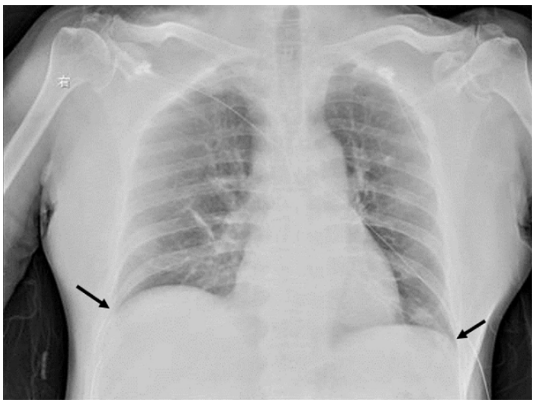

Figure 2. Chest radiography showed bilateral pulmonary inflammation and pleural effusion (black arrows).

12-lead electrocardiography (ECG) performed on arrival at the emergency department demonstrated sinus tachycardia, type 2 secondary degree atrioventricular block (AVB), 4-12 mm convex ST elevation in leads V1-4 (**Figure 1**). Q waves were present in lead V1-3. Chest radiography showed bilateral pulmonary inflammation and pleural effusion suggesting heart failure (**Figure 2**). Two-dimensional transthoracic echocardiography showed normal size of four chambers, ejection fraction 52%, mild mitral and tricuspid regurgitation, global left ventricle hypokinesia and apical akinesia.

Risk factors for CAD included smoking, male gender and old age. Presumptive diagnosis was an anterior-septal STEMI based on the typical electrocardiographic changes and ongoing chest discomfort, he was given dual antiplatelet therapy and underwent urgent coronary angiography. Coronary angiography was performed which revealed no significant stenosis. There were no perfusion defects (TIMI grade 3 flow) in left and right coronary arteries (**Figure 3**). He was subsequently transferred to the coronary care unit for observation.

Following MR showed mild impaired biventricular dysfunction without pericardial effusion, but LGE imaging revealed multifocal areas of contrast accumulation in subepicardial and intramyocardial distribution as well as the lateral wall (**Figure 4**). Diagnosis was based on the signs and symptoms, ST-segment elevation, increased cardiac biomarkers, MR and LGE changes. All the findings were consistent with perimyocarditis. However, his virology screening and autoimmunity screening were unremarkable. Corticosteroid and intravenous immunoglobulin were given and remaining treatment was supportive. The patient remained asymptomatic during the rest of the hospitalization and was discharged in good condition.

Discussion

ESC Working Group just published a position paper on myocardial infarction with non-obstructive coronary arteries. Among patients presenting with STEMI, the majority have coronary artery occlusion on angiography. In contrast, in patients presenting with NSTEMI, approximately 25% have evidence of coronary artery occlusion. Approximately 10% of patients presenting with acute myocardial infarction (AMI) have no significant obstructive coronary artery disease (MINOCA). A diagnosis of MINOCA can be made by many modalities in patients presenting with features consistent with AMI and a coronary angiogram demonstrating non obstructive coronary artery disease. Myocarditis can resemble an acute coronary syndrome, even an ST-segment elevation myocardial infarction. Furthermore, when a coronary angiogram reveals no significant disease, differential diagnosis can be challenging. Endomyocardial biopsy is still the gold standard for the diagnosis of myocarditis, however, its invasive character and limited sensitivity restrict its generalized application to all patients [4].

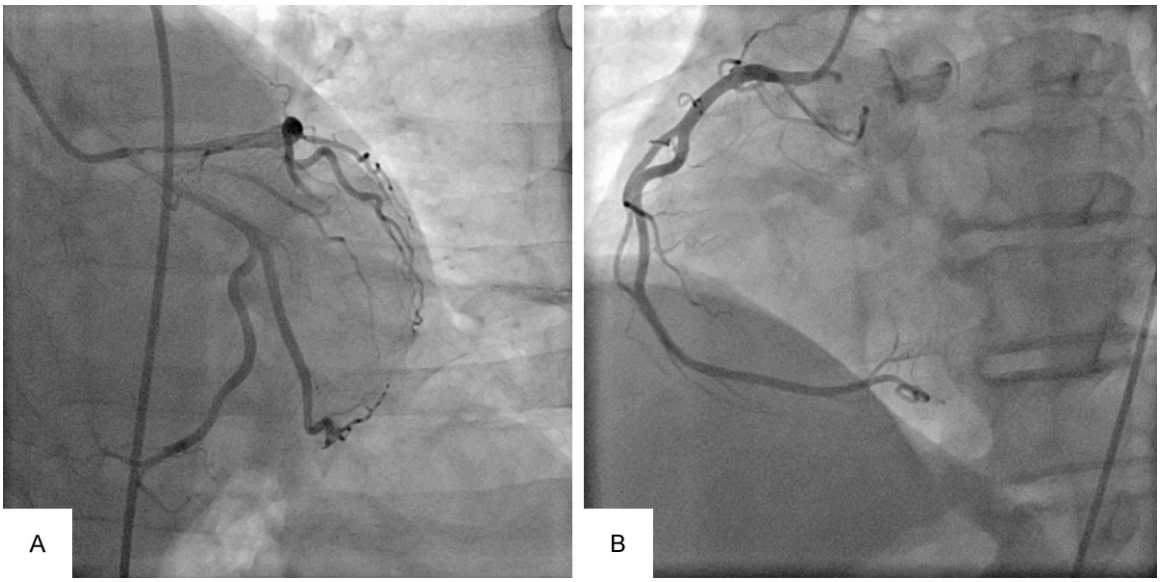

Figure 3. Coronary angiography showed no significant stenosis of the left (A) and right (B) coronary arteries.

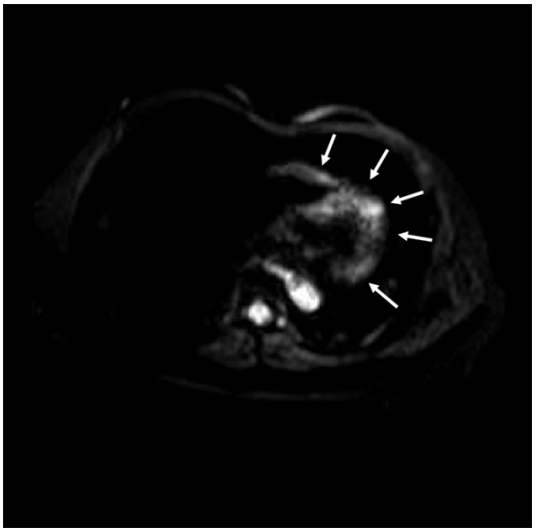

Figure 4. LGE imaging revealed multifocal areas of contrast accumulation in subepicardial, intramyocardial and lateral wall distribution (white arrows).

CMR has emerged as a leading modality in the noninvasive diagnosis of myocarditis due to its ability to detect myocardial edema, hyperemia, necrosis and fibrosis in a safe and reproducible fashion. In patients with an acute coronary syndrome (ACS)-like presentation but normal coronary arteries, CMR is useful not only to differentiate acute myocarditis from an ischemic event but also to identify alternative etiologies. Beyond the evaluation of global and segmental biventricular function and pericardial effusion, the ability of CMR to characterize histological changes of myocarditis relies on the detection of interstitial edema, hyperemia and capillary leakage, cardiomyocyte necrosis and myocardial fibrosis with specific sequences. In ACS, edema is typically localized to the territory of the culprit vessel. In myocarditis, edema may be either segmental or diffuse, which justifies the quantification of myocardial signal intensity in comparison with a reference tissue like skeletal muscle. LGE imaging may reveal focal areas of contrast accumulation secondary to cellular necrosis and/or replacement fibrosis. The characteristic pattern of LGE in myocarditis is patchy or multifocal in a subepicardial or intramyocardial distribution, often involving the lateral wall. This feature is not pathognomonic but is clearly distinct from ischemic heart disease, which typically presents with subendocardial or transmural LGE within a coronary artery territory [2, 3].

In our patient, his presenting ECG showed up to 12 mm ST elevations in anterior chest leads V1-4, which are suggestive of acute myocardial injury, but his transthoracic echocardiography suggested the diagnosis of pericarditis by showing the presence of global wall motion abnormalities, which did not conform to any coronary artery territories. Most importantly, his CMR and LGE imaging displayed the typical changes of myocarditis which were mentioned above. Our patient most likely presented with

acute pericarditis and subsequently developed acute myocarditis, causing a change in electrocardiographic morphology and a rise in cardiac enzymes. This case highlights that perimyocarditis may present with electrocardiographic changes and elevated cardiac enzymes that mimic myocardial infarction. This case also indicated that coronary angiographies may be necessary to exclude an acute STEMI [1, 5-7].

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Disclosure of conflict of interest

None.

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