Acute Viral Myocarditis Mimicking ST Elevation Myocardial Infarction: Manifestation on Cardiac Magnetic Resonance

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A 58-year-old man presented himself to the emergency room with chief complaints of chest pain and progressively exacerbated dyspnea for 4 days. Electrocardiography showed Q wave and ST-segment elevation in inferior leads. A thallium scan showed mixed ischemia and infarction in the inferior wall of the left ventricle. However, coronary artery angiography showed normal coronary arteries. Cardiac magnetic resonance showed a typical pattern of delayed enhancement in acute myocarditis. A viral serological study identified coxsackievirus B6 infection. We report a case of acute viral myocarditis mimicking ST elevation myocardial infarction and suggest that cardiac magnetic resonance with Gadolinium enhancement might be an effective method to detect acute viral myocarditis.

Key Words: Cardiac magnetic resonance • Coxsackievirus B • Delayed enhancement • Myocardial infarction • Myocarditis

INTRODUCTION

Acute myocarditis is one of the most challenging diagnoses in cardiology. It can present with various clinical manifestations, including mimicking acute ST-elevation myocardial infarction.1 The diagnostic gold standard of myocarditis is endomyocardial biopsy (EMB) with the histological Dallas criteria. However, EMB is limited by high variability and sampling error. Recently, some new non-invasive methods have been proposed to accurately differentiate acute myocarditis from acute myocardial infarction.2,3 We report here a case of acute viral myocarditis mimicking ST elevation myocardial infarction with typical manifestations on cardiac magnetic resonance (CMR) with gadolinium enhancement.

CASE REPORT

A 58-year-old man had symptoms of acute upper respiratory infection, including cough, poor appetite, and watery diarrhea, that had been ongoing for 1 week before hospitalization. He experienced one episode of severe chest pain about 4 days before admission. He complained of intermittent chest pain and progressively exacerbated dyspnea on exertion since then. An electrocardiogram (ECG) obtained in the emergency room showed Q wave and ST-segment elevation in inferior leads (Figure 1A). Chest X-ray showed cardiomegaly and pulmonary congestion. Laboratory data indicated elevated cardiac enzyme concentrations (troponin I: 6.63 ng/ml; normal range: < 0.11 ng/ml). The patient was admitted to the cardiovascular care unit.

Trans-thoracic echocardiography showed diffuse hypokinesis, especially of the inferior wall, with impaired left ventricular ejection fraction of 47%. He received a thallium-201 scan, which indicated a mixed type of perfusion defect with ischemia and infarction in the inferior wall of left ventricle. Given a tentative diagnosis of recent ST-elevation myocardial infarction com-
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Figure 1. (A) Twelve-lead electrocardiogram on admission shows Q wave and ST-segment elevation in inferior leads. (B) Coronary angiography. Right-anterior-oblique (RAO) cranial view of left coronary artery shows patent left anterior descending artery and left circumflex artery. (C) Left-anterior-oblique (LAO) view of right coronary artery shows patent right coronary artery. (D) Follow-up twelve-lead electrocardiogram 2 months after discharge still shows Q wave in inferior leads.
complicated by congestive heart failure, coronary angiography was performed (Figures 1B, C). However, coronary angiography showed that the coronary arteries were normal.

Acute viral myocarditis was highly suspected. CMR with intravenous injection of gadolinium was performed on the second day after undergoing coronary angiography and showed subepicardial and centromyocardial delayed enhancement in the inferior and posteroseptal wall of the left ventricle (Figure 2). Viral serological analysis detected a coxsackievirus B6 infection. The patient’s symptoms were relieved soon after treatment for heart failure, and he was discharged in stable condition 6 days later.

Follow-up echocardiography at 2 weeks and 2 months showed normal global left ventricular function with ejection fractions of 61% and 67%, respectively. However, the twelve-lead ECG (Figure 1D) followed-up at 2 months still showed Q wave in inferior leads which could not be used to differentiate myocarditis from myocardial infarction.

DISCUSSION

Myocarditis is a disease with variable prognosis, ranging from spontaneous complete recovery to dilated cardiomyopathy and even sudden death. The pathogenesis of viral myocarditis follows a chronological sequence of three pathologically distinct phases: direct viral invasion, immune dysregulation, and extensive myocardial injury. The direct viral-mediated destruction that occurs in the first phase results in focal myocardial injuries, that can mimic symptoms of acute myocardial infarction, such as focal ST-segment elevation on ECG and elevated cardiac enzyme concentrations. Viral infections trigger cellular and humoral immune responses. Some mimicked epitopes are shared between the viral and cardiac antigens. During the second phase, auto-antibodies against cardiomyocytes are induced by molecular mimicry between the viral and cardiac antigens, which is followed by autoimmune responses. Finally, extensive myocardial injury and even dilated cardio-

Figure 2. (A) Short-axis FIESTA MR image shows focal hyperintense signals indicating edematous changes within the myocardium and subepicardium of inferior and posteroseptal wall of the mid level of the left ventricle (arrow). (B) Short-axis first-pass perfusion MR image shows focal perfusion defect in the area (arrow) mentioned in Figure (A); this defect corresponds to the delayed enhancement seen in the delayed enhancement image (C) (arrow).
myopathy can develop in the third phase. These pathogenic processes might explain the clinical manifestations of this patient. All of the ECG and imaging studies, including the thallium-201 scan and the echocardiography, showed focal regional wall damage that might have resulted from direct viral invasion during the first phase of pathogenesis. The diffuse hypokinesis of the left ventricle, which was evident on echocardiography, might have resulted from autoimmune responses during the second phase of pathogenesis.

CMR has proven helpful in not only diagnosing myocarditis but also in showing the extent of myocardial injuries with delayed contrast enhancement. In addition, EMB from a location determined by CMR can increase the chances of detecting acute myocarditis pathologically.

The mechanism of delayed enhancement of CMR in acute myocarditis has not been fully elucidated. CMR contrast agents, such as gadolinium, cannot enter cardiomyocytes because of their intact cell membranes. In the setting of acute myocarditis, the ruptured sarcolemmal membranes of damaged cardiomyocytes allow the extracellular contrast to diffuse into the cells. During healing, the intercellular spaces increase because necrotic cardiomyocytes are replaced by fibrous tissue, which increases the distribution space for extracellular contrast agents. Both enlarged intracellular and intercellular contrast distribution volume are possibly responsible for the delayed enhancement during CMR.

The typical pattern of delayed enhancement during CMR for acute myocarditis is a patchy distribution originating from the epicardial quartile of the wall with one or several foci. The prevalence of delayed enhancement, by location, is as follows: lateral free wall (85%), posterior and inferior walls (46%), septum (31%), and anterior wall (23%). Another distinct pattern of delayed enhancement originates from the subendocardium corresponding to a coronary artery distribution is typical in myocardial infarction, but has never been seen in myocarditis. In the present case, we could not distinguish myocardial infarction from myocarditis by ECG or with a thallium-201 scan, which showed only inferior wall damage. However, a typical pattern of subepicardial and centromyocardial delayed enhancement in the inferior and posteroseptal wall was evident by CMR, which indicated acute myocarditis and was compatible with the ECG changes and thallium-201 scan findings.

The clinical symptoms experienced by our patient (e.g., acute chest pain and elevated ST-segment changes) can indicate of several different types of cardiac disease, including coronary artery diseases, acute myocarditis, and pericarditis. The present findings indicate that CMR with contrast enhancement might be an effective method for diagnosing acute myocarditis.

REFERENCES