

CASE REPORT

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Coronary artery spasm due to acute myocarditis in an adolescent: a case report

Hoon Ko¹, Taehong Kim^{1*} , Hyoung Doo Lee¹, Jung-Hee Byun¹ and Ki Seok Choo²

Abstract

Background: Myocarditis refers to the inflammation of the myocardium caused by infection or autoimmune disease that may or may not present with clinical manifestations, such as gastrointestinal symptoms, dyspnea, chest pain, or sudden death. Although myocarditis and coronary artery vasospasm may mimic ST-segment elevation myocardial infarction (STEMI) with normal coronary arteries on angiography, acute myocarditis rarely causes coronary artery spasm. Here, we report a case of coronary artery spasm with reversible electrocardiographic changes mimicking STEMI in an adolescent with acute myocarditis.

Case presentation: A 15-year-old boy present with sudden-onset repeated chest pain following a 3-day history of flu-like illness. Cardiac biomarkers were significantly elevated. Electrocardiography showed ST-segment elevation in the absence of detectable vasospasm on coronary angiography. These findings were consistent with the diagnosis of coronary artery spasm secondary to acute myocarditis. Treatment with immunoglobulin for 2 days improved his condition. The patient was discharged on the 12th day with complete resolution of symptoms and normalization of electrocardiogram findings.

Conclusions: We reported a case of coronary artery spasm due to acute myocarditis. This study highlights the importance of considering coronary artery spasm due to acute myocarditis as a differential diagnosis in patients presenting with signs of STEMI as these diseases have different medical management strategies.

Keywords: Acute myocarditis, Coronary artery spasm, ST-segment elevation, Case report

Background

Myocarditis refers to the inflammation of the myocardium diagnosed using established histological, immunological, and immunohistochemical criteria [1]. Cell death and injury due to inflammation may cause structural and functional cardiac abnormalities presenting with various clinical manifestations. Because acute myocarditis may mimic ST-segment elevation myocardial infarction (STEMI), its early identification may have important therapeutic and prognostic implications [2, 3]. Coronary

artery spasm is a sudden, intense, transient and reversible vasoconstriction of an epicardial coronary artery that can also result in ST-segment elevation. However, acute myocarditis rarely causes coronary artery spasm.

Here, we report an unusual case of coronary artery spasm secondary to acute myocarditis in a 15-year-old boy presenting with chest pain, ST-segment elevation on electrocardiogram (ECG), and highly elevated cardiac biomarkers in the absence of detectable vasospasm on coronary angiography.

Case presentaion

A 15-year-old boy presented with sudden-onset chest pain at 02:30 AM characterized as squeezing on the left parasternal area that persisted for over 2 hours following a 3-day history of flu-like illness. Although this resolved

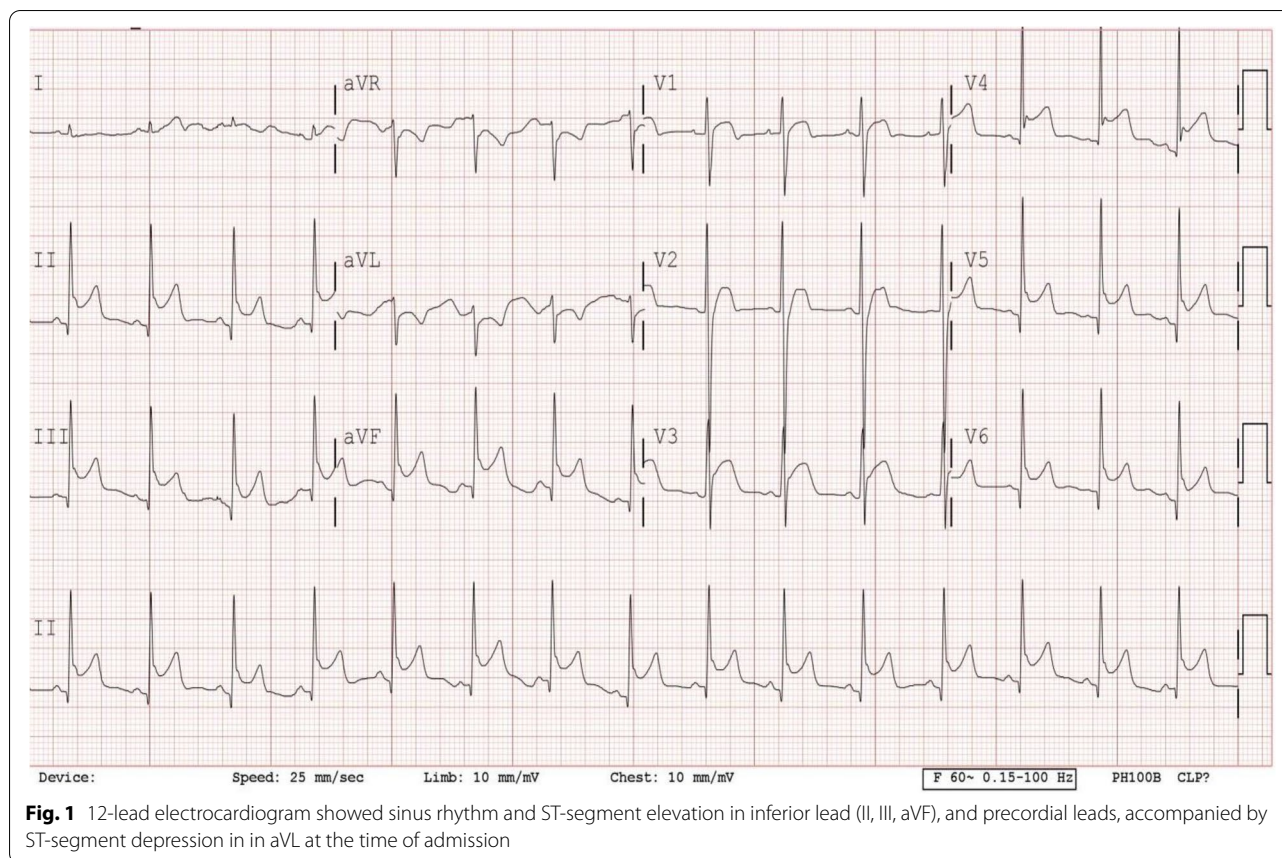
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spontaneously at first, he experienced chest pain and dyspnea 18 hours later, which prompted a visit to the emergency room. During admission, his creatine kinase MB fraction was elevated at 49.2 ng/ml (normal range: 0 to 3.6 ng/ml; AMI cut off: 5.0 ng/ml) and Troponin-I was elevated at 15.5 ng/ml (normal range: 0 to 0.056 ng/ml; AMI cut off: 0.6 ~ 1.5 ng/ml). ECG showed ST-segment elevation at I, aVL, and precordial leads.

The patient was then transferred to the emergency room of our hospital. He was middle school student with no known comorbidities, such as hypertension, diabetes, or cardiovascular disease. He had no family history of coronary artery disease. He denied alcohol consumption and smoking.

His blood pressure was 110/70 mmHg, the heart rate was 86 bpm, the respiratory rate was 15/min, and the body temperature was 38.5°C. His oxygen saturation at room air was 99%.

Physical examination, including cardiovascular examination, was unremarkable. The chest X-ray revealed normal heart size and clear lung fields. ECG showed sinus rhythm with ST-segment elevation in the inferior and precordial leads (Fig. 1). Transthoracic echocardiography

revealed normal left ventricular ejection fraction (66.04%) and very mild anteroseptal hypokinesis.

After 23 h from the onset of chest pain, myoglobin was elevated at 363.2 ng/mL (normal range: 12 to 80 ng/ml), high sensitive-Troponin I increased to 21,538.68 pg/mL (normal range: 0 to 19.8 pg/ml), and creatine kinase MB fraction also increased to 170.0 ng/mL (normal range: 0.5 to 3.1 ng/ml). To differentiate STEMI or other combined coronary vessel anomalies, coronary angiography was performed, and it revealed normal coronary arteries (Fig. 2). Treatment with immunoglobulin for 2 days improved his condition and cardiac markers (myoglobin: 8.6 ng/mL, high sensitive-Troponin I: 7466 ng/mL, creatine kinase MB fraction: 3.0 ng/ml) and ECG. On the 3rd night of admission, he experienced another episode of chest pain during sleeping with increased cardiac markers (myoglobin: 10.5 ng/ml, high sensitive-Troponin I: 16,164.61 ng/ml, creatine kinase MB fraction: 7.8 ng/ml) and ST-segment change on ECG (Fig. 3). The chest pain resolved without medication.

All of these findings were consistent with the presumptive diagnosis of acute myocarditis and coronary artery spasm leading to transient ST-segment elevation

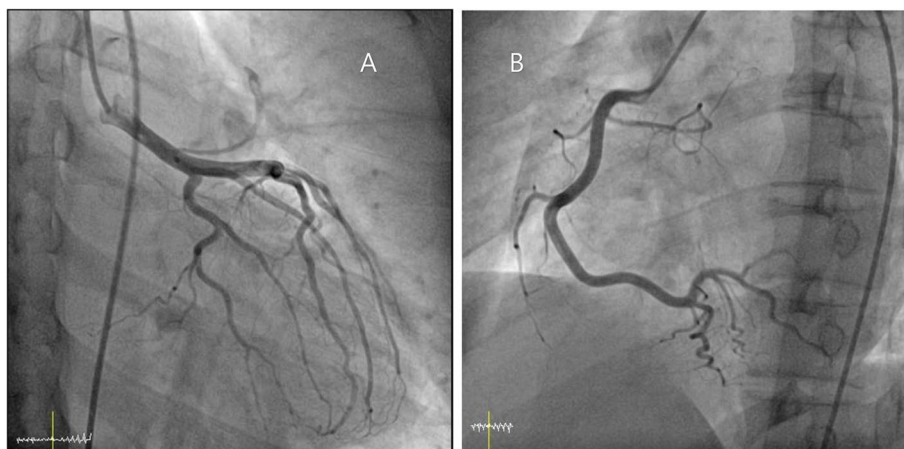


Fig. 2 Coronary angiography demonstrated normal left (A) and right (B) coronary arteries

on ECG. To confirm the diagnosis of myocarditis, we performed cardiac magnetic resonance imaging (MRI) and it showed myocardial edema and subepicardial enhancement in the anterolateral and infero-lateral wall of mid base left ventricle (Fig. 4). On the 12th day of admission, the patient was discharged with complete resolution of symptoms and normalization of the ECG findings (Fig. 5).

Discussion and conclusions

Myocarditis refers to the inflammation of the myocardium due to infectious (typically viruses) and non-infectious causes (i.e., toxins, hypersensitivity reactions, and

autoimmune disease) [4]. The most common causes of viral myocarditis include parvovirus B19, human herpesvirus 6, enterovirus, cytomegalovirus, adenovirus [5].

The pathogenesis of viral myocarditis involves three chronologically and pathologically distinct phases. In the first phase of myocarditis, the virus enters and proliferates in the myocardium, causing direct myocellular necrosis and apoptosis. This is followed by the initiation of the innate immune response. Both the direct myocardial damage cause by the virus and the subsequent immune response result in the cardiomyocytes destruction, mimicking signs and symptoms of acute myocardial infarction, such as focal ST-segment

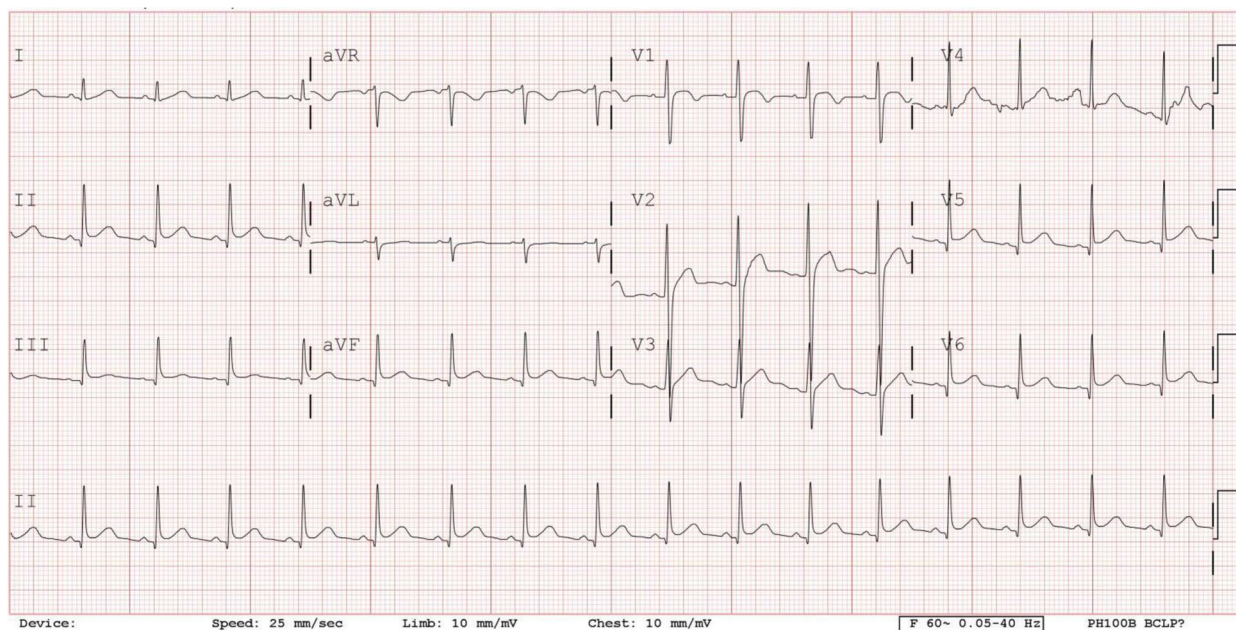


Fig. 3 12-lead electrocardiogram on 4th day of admission showing sinus rhythm with mild ST segment change in II, V5 and V6

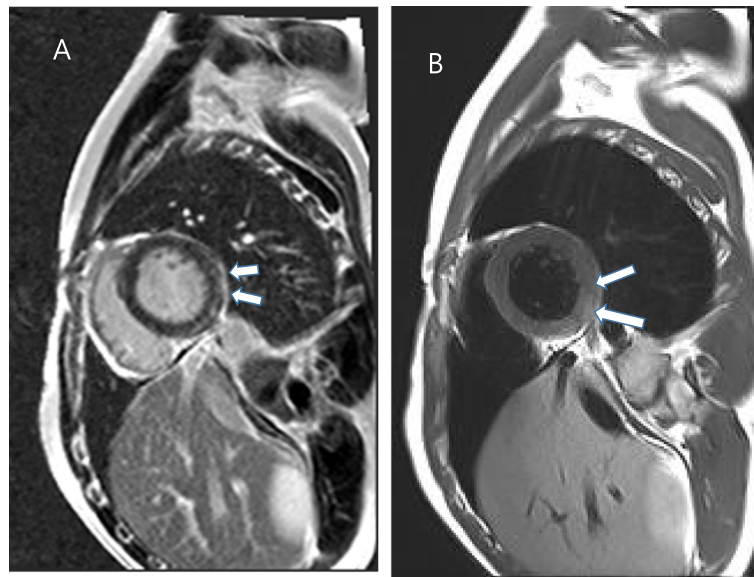


Fig. 4 Cardiac magnetic resonance image. Short-axis late gadolinium enhanced (LGE) MR images (a) show subepicardial enhancement (arrow) in the anterolateral and inferolateral wall of mid base left ventricle (LV) level. Short-axis T2-weighted MR images (b) show high signal intensities (arrows) in those regions with enhancement regions, suggesting myocardial edema

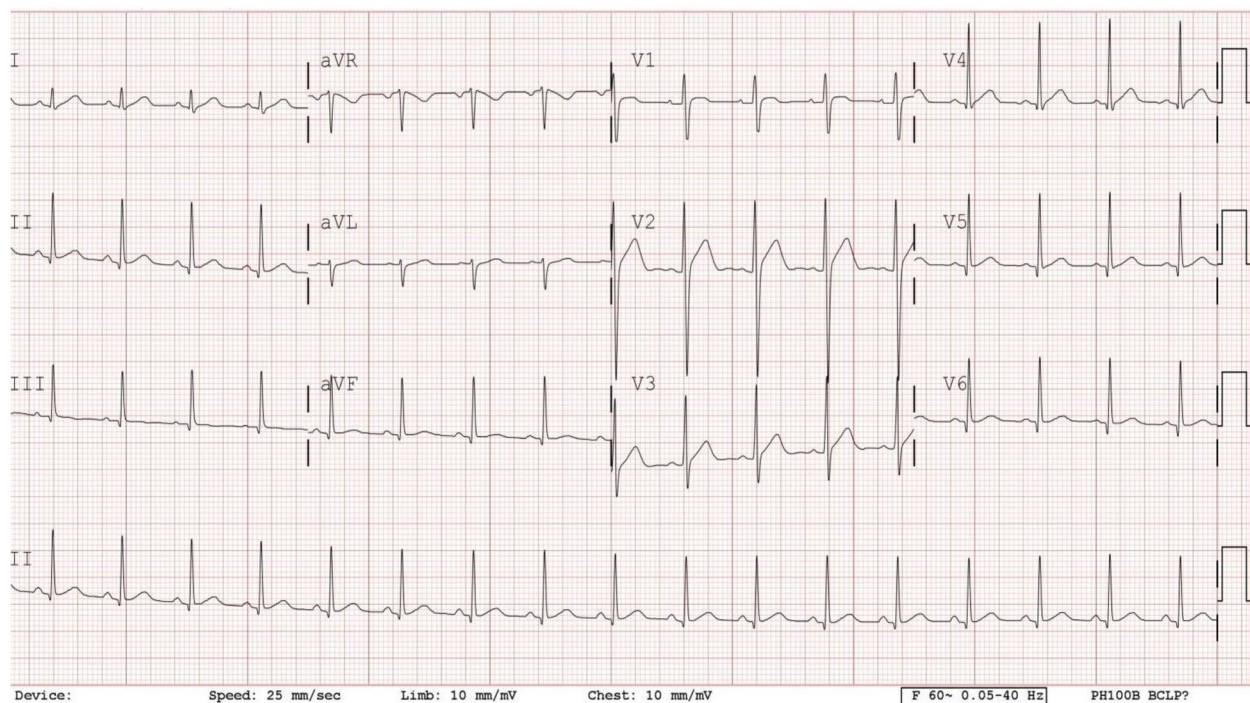


Fig. 5 Repeat electrocardiogram on the day 12 of hospitalization showing near complete resolution of ST-segment elevation

elevation on ECG and elevated serum cardiac enzymes [6]. Following the first phase, patients may recover or progress into the next phase. In the second phase, the adaptive immune response is activated. In the third

phase, extensive myocardial injury and dilated cardiomyopathy may develop [5].

Myocarditis may or may not present with several non-specific clinical manifestations, such as gastrointestinal

symptoms, dyspnea, chest pain, cardiogenic shock or sudden death. Gastrointestinal symptoms (e.g., abdominal pain, nausea, poor appetite and vomiting) are the most common presentations of acute myocarditis [7]. ECG, cardiac biomarkers (i.e., creatinine kinase MB fraction and troponin I), cardiac MRI and endomyocardial biopsy are useful tools in the diagnosis of acute myocarditis [5, 6].

Prinzmetal et al. postulated the concept of coronary artery spasm by describing pain associated with transient non-progressive ST-segment elevation appeared at rest or during regular activity but was not brought on by exercise or emotional disturbance. Coronary artery spasm is a reversible vasoconstriction due to a spontaneous vascular smooth muscle hypercontractility and vascular wall hypertonicity. This cause luminal narrowing of normal or atherosclerotic coronary arteries, compromising the myocardial blood flow and usually leads to transient myocardial ischemia, myocardial infarction, and sudden death [8, 9]. A typical clinical presentation of coronary vasospasm is variant angina, in which chest pain usually occur at rest between night and early morning, not induced by exercise or effort during daytime and spasms are usually brief, but can persist for more than 15 minutes. This is an important feature of the history and often used in diagnostic criteria [10]. Although the exact pathophysiological mechanism of coronary artery spasm is not clearly understood, abnormal response of the autonomic nervous system, endothelial dysfunction, inflammation, oxidative stress, vascular smooth muscles hypercontractility, and other factors (e.g. magnesium deficiency, inheritance, or specific anatomy of the coronary artery) have been proposed [11].

Myocarditis and coronary artery spasm are well-known causes of ST-segment elevation, mimicking acute myocardial infarction. The cardiac MRI findings, past history, age, risk factors, subjective symptoms, value of cardiac markers, dynamic changes in ECG are very useful for differential diagnosis. To the best of our knowledge, coronary artery spasm precipitated by acute myocarditis has rarely been reported in the pediatric population. The pathophysiological mechanism of coronary artery spasm due to acute myocarditis remains unclear. There are several mechanisms suggested that vasoactive substances (e.g. thromboxane A₂), myocarditis-induced endothelial dysfunction, smooth muscle hyperactivity may induce coronary artery spasm in acute myocarditis patients [12–14].

In our case, myocarditis was confirmed using cardiac MRI. Moreover, the coronary artery spasm was consistent with the features of chest pain, ST-segmental elevation and spontaneous resolution, and normal coronary angiographic findings. We presumed that acute

myocarditis precipitated coronary artery spasm as a result of myocarditis-induced coronary endothelial dysfunction or coronary smooth cell hyperactivity similar to the findings of other studies [12–14].

In conclusion, we reported a case of coronary artery spasm due to acute myocarditis. This study highlights the importance of considering coronary artery spasm due to acute myocarditis as a differential diagnosis in patients presenting with signs of STEMI as these diseases have different medical management strategies.

Abbreviations

STEMI: ST-segment elevation myocardial infarction; ECG: Electrocardiography; MRI: Magnetic resonance imaging.

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Not applicable.

Authors' contributions

Conceptualization, T.H.K. and H.D.L.; Investigation, T.H.K., H.K. and J.H.B.; Data curation, T.H.K., H.D.L.; Visualization, T.H.K. and K.S.C.; Writing-original draft, T.H.K., H.K. and K.S.C.; Writing-review & editing T.H.K. All authors have read and agreed to the published version of the manuscript.

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Availability of data and materials

The data presented in this study are available on reasonable request from the corresponding author.

Declarations

Ethics approval and consent participate

This study was conducted according to the guidelines of the Declaration of Helsinki and approved by the Institutional Review Board (IRB) of Pusan national university Yangsan hospital (No. 05–2021-131).

Consent for publication

Written consent was obtained from patient's patient for publication of this case report and any accompanying images.

Competing interests

The authors declare no conflicts of interest.

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