Myocardial Infarction with Normal Coronary Arteries in a Patient with Heroin Use and Infective Endocarditis

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Myocardial infarction with normal coronary arteries typically occurs in patients under 50 years of age. There is usually no history of angina or previous myocardial infarction, and risk factors for ischemic heart disease are frequently absent. We report a 27-year-old heroin user with normal coronary arteries and inferior wall infarction secondary to infective endocarditis. The left ventricular dysfunction normalized after antibiotic and surgical treatments for infective endocarditis. He was followed at our outpatient clinic for one year without recurrence.

Key Words: Heroin • Infective endocarditis • Left ventricular ballooning syndrome • Myocardial infarction with normal coronary arteries

INTRODUCTION

When myocardial infarction with normal coronary arteries occurs, it typically affects patients under 50 years of age. Usually there is no history of angina or previous myocardial infarction, and risk factors for ischemic heart disease are generally absent. Takotsubo cardiomyopathy (TTC) is a form of myocardial stunning following a stressful event that evokes intense coronary vasospasm, which may or may not be atherosclerotic. 1-3 In both diseases, symptoms and electrocardiographic findings are similar to those of myocardial infarction with angiographic coronary disease, though the infarct sizes tend to be smaller. Here, we report a 27-year-old heroin user with normal coronary arteries and inferior wall infarction secondary to infective endocarditis. The possible mechanism involved in this patient’s medical circumstance are herein discussed.

CASE REPORT

A 27-year-old man presented to our emergency department (ED) because of deteriorated dyspnea. The patient was a smoker (i.e., one package of cigarettes daily for 10 years) and an intravenous heroin user for about 10 years; however, his past medical history was otherwise unremarkable. The patient had experienced intermittent fever and chills in the previous two months and had taken acetaminophen for symptom relief. At the ED, the patient’s body temperature was 36.2 degrees Celsius, with a respiratory rate was 21 breaths per minute. The heart rate was approximately 125 beats per minute, and the initial blood pressure was 118/70 mmHg. Heart auscultation revealed a grade IV/V holosystolic murmur at the left lower sternal border and apex.

The initial patient electrocardiographic (ECG) revealed sinus tachycardia with ST-elevation in the inferior
leads and reciprocal ST-depression in leads V1-V3 (Figure 1A). The right side ECG did not show any significant ST-elevation in the precordial leads. Subsequent laboratory tests revealed leukocytosis with a white blood cell count of 30,520/ul (bands: 7%, segments: 84%, lymphocyte: 1%, monocyte: 5.5%, metamyelocyte: 2%), red blood cell count: 464 × 10^6/ul, hemoglobin: 14.4 g/dL, platelet: 47000/ul; biochemical analysis: blood urea nitrogen: 23.7 mg/dL, creatinine: 1 mg/dL, sodium: 127 mmol/L, potassium: 3.5 mmol/L, elevated C-reactive protein (15.9 mg/dL), and elevated cardiac enzymes (serum creatine kinase: 405 IU/L; CK-MB: 18 IU/L; troponin I: 5.26 ng/mL). Additionally, the serum morphine level was 2803 ng/ml (reference range: 0-300 ng/mL).

It was suspected that the patient had an acute inferior wall ST-elevation myocardial infarction, and he underwent urgent coronary angiography which revealed patent coronary arteries (Figure 2A and B). A left ventriculogram (LVG) revealed impaired left ventricular (LV) contractility with inferior wall akinesis (LV ejection fraction: 55%) (Figure 2C and D).

After admission, a comprehensive physical examination revealed Janeway lesions over both hands and feet. Echocardiogram disclosed inferior wall akinesis and an oscillating mass (1.7 cm × 1 cm) attached to the tricuspid valve with mild tricuspid regurgitation (TR) (Figure 2E and F). Blood cultures grew oxacillin-sensitive Staphylococcus aureus, and infective endocarditis was diagnosed using the Duke criteria. Consequently, the patient was started on oxacillin. Three days after admission to our facility, the follow-up ECG revealed partial resolution of the ST-elevation (Figure 1B), and the echocardiogram showed normalization of the inferior wall akinesis (LV ejection fraction: 70%).

![Figure 1](image)

**Figure 1.** (A) Electrocardiography (ECG) at the emergency department revealed ST-elevation in the inferior leads with reciprocal changes in leads V1-V3. (B) ECG 3 days after admission revealed partial resolution of the ST-segment elevation in leads II, III, aVF. (C) ECG 14 days after admission revealed further decrease in the elevated ST segment with T wave inversion in leads II, III, aVF. (D) ECG 2 months later revealed pathologic Q wave with T wave inversion in leads II, III, and aVF.
After two weeks of antibiotic treatment, intermittent fever, dyspnea, and leukocytosis were still observed. The patient’s ECG at two weeks revealed further resolution of the ST-elevation with T wave inversion in leads II, III, and aVF (Figure 1C). A follow-up echocardiogram revealed no resolution of the tricuspid valve vegetation, severe TR, and new mitral valve vegetation with severe mitral regurgitation (MR). Chest radiographs showed multiple ill-defined nodular lesions, and chest computed tomography confirmed the diagnosis of septic emboli. Mitral valve replacement and tricuspid annuloplasty were performed because of the uncontrolled infection and refractory congestive heart failure (related to severe MR and TR). The patient’s pathology report substantiated the diagnosis of infective endocarditis. Following surgery, the patient gradually recovered with antibiotic treatment and was discharged four weeks later (oxacillin 2000 mg q4h for a total of six weeks). The ECG taken 2 months later showed pathologic Q wave with T-wave inversion in leads II, III, and aVF (Figure 1D). He was followed at our outpatient clinic for one year without disease recurrence.

DISCUSSION

Acute myocardial infarction may occur in young people with angiographically normal coronary arteries but the pathophysiology of this condition is usually unclear. The possible mechanisms underlying myocardial infarction with normal coronary arteries are coronary vasospasm, thrombosis, embolization or myocardial injury (left ventricular ballooning) following a stressful event.

Prinzmetal angina (coronary vasospasm)-induced acute myocardial infarction was one of the differential diagnoses in our patient. Angelini described the pathophysiologic theories in Prinzmetal angina and left ventricular apical ballooning syndrome: in Prinzmetal angina, the coronary spasm is usually focal and causes transient segmental ischemia; in left ventricular apical ballooning, the coronary spasm is generalized and causes diffuse ischemic injury and persistent myocardial stunning. The distinct pathophysiologic theories as noted above contribute to different clinical presentation and laboratory findings: the duration of chest pain is usually
shorter and the recurrence rate is higher in Prinzmetal angina than in left ventricular ballooning. The characteristic of electrocardiogram of Prinzmetal angina is transient ST-elevation which returns to baseline rapidly upon resolution of symptoms. The ECG change of TTC varies, including ST elevation (which usually returns to normal within 3 days), second peak negative T wave and Q wave formation.\(^3,5\) Our patient had no history of repetitive episodes of chest pain, yet the serial ECGs showed persistent electrocardiographic changes. Therefore, Prinzmetal angina was less favored as a final diagnosis.

There were two case reports of heroin-induced acute myocardial infarction secondary to coronary spasm.\(^6,7\) Both cases involved normal coronary arteries proven by coronary angiography. The authors suggested that the coronary spasm was caused by heroin-induced parasympathetic activation. Because the patient used heroin, heroin withdrawal was also a potential cause. Two case reports of TTC secondary to methadone withdrawal were identified in the literature.\(^8,9\) Opioid withdrawal likely induces an increase in catecholamine plasma concentrations, which contributes to the development of TTC.\(^9\) Sztajzel et al. reported a 25-year-old woman who developed an acute anterior ST-elevation myocardial infarction due to acute thrombosis of the left anterior descending coronary artery after heroin use.\(^10\) This patient also had smoked 10 cigarettes a day for 5 years. The authors hypothesized that the heroin might have had a direct toxic effect on the coronary arteries and induced an acute coronary occlusion, either by provoking local coronary artery spasm or inflammation. Thus, urgent coronary angiography is still required for the differential diagnosis in young patients with a low coronary risk.

TTC is characterized by transient and reversible systolic dysfunction that usually involves the apical and/or mid-segments of the left ventricle. First described in the early 1990s in Japan,\(^1\) TTC can mimic acute myocardial infarction; however, little or no evidence of obstructive coronary artery disease is noted. A single center study in Taiwan revealed a prevalence of 0.9% for acute coronary syndrome,\(^2\) which is slightly lower than rates reported in a systematic review (1.7 to 2.2%).\(^3\) Typically, the LVG reveals hypokinesis or akinesis of the mid- and apical segments, and the term “apical ballooning syndrome” was applied to the condition. Several atypical forms have been less frequently reported, including an apical-sparing variant (i.e., preserved function at the apex with hypokinesis of the mid-segments) and an inverted Takotsubo type (i.e., hypokinesis of the basal segment with preserved apical function).\(^11,12\) First reported in Japan in the early 1990’s, TTC was named for the shape of the LVG during the systolic phase, which resembles an octopus trapping pot.\(^1\) Because several atypical forms of this cardiomyopathy did not involve the apical segment, the term “left ventricular dysfunction (ballooning) syndrome” has been used instead of the “apical ballooning syndrome.”\(^11,13\) The most common electrocardiographic abnormality is ST-elevation. In a systematic review including 14 studies, ST-elevation was detected in 81.6% (208/255) of patients with TTC. Among these patients, 83.9% had ST-elevations in the precordial leads.\(^3\) The other 16.1% of patients did not have the location of the ST segment elevation reported. Therefore, the true proportion of ST-elevation in the precordial leads may be even higher than initially reported.

There are disagreements about the clinical features and outcomes between typical and atypical (midventricular) TTC. Kurowski et al. reported that there were no differences in demographic information, clinical findings, laboratory findings, angiographic findings, or outcomes between typical (n = 22) and atypical (n = 13) TTC patients.\(^11\) In contrast, Chen et al. reported that the patients with atypical TTC had the same clinical presentations, ejection fractions, and in-hospital courses as those with typical TTC, but tended to be younger and have a lower incidence of ST-elevation.\(^12\) In 2009, Rognoni et al. reported a 50-year-old woman who presented with 30 minutes of chest pain, ST-elevation in the inferior leads on the ECG, and elevated cardiac enzymes.\(^13\) Urgent coronary angiography revealed coronary arteries free of lesions, but the LVG showed dyskinesia of the inferior wall. The patient was discharged home after 5 days, and the follow-up echocardiogram two months later showed no wall motion abnormalities.\(^13\) The authors named this condition an atypical left ventricular ballooning syndrome. Similar to our patient, they could not identify a definitive etiology in their patient.

In conclusion, coronary artery spasm related to heroin use or myocardial injury following a stressful event (infective endocarditis or heroin withdrawal) is the possible mechanism for this patient with myocardial infarc-
tion and normal coronary arteries.

REFERENCES