Fatal Embolic ST-Elevation Myocardial Infarction Secondary to Healed-Phase Mitral Valve Infective Endocarditis

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Summary

Embolic myocardial infarction (MI) caused by infective endocarditis (IE) is rare, but it is increasingly recognized as an important complication. This complication typically occurs in patients with aortic valve endocarditis during the acute phase of the infection. It is also known to have a high mortality rate; however, the best practice for its management is unclear owing to scarce available data. In addition, most cases of embolic acute MI (AMI) caused by IE are indirectly diagnosed with a combination of angiographic examination such as coronary angiography or cardiac computed tomography. Herein, we report a case of fatal embolic ST-elevation MI (STEMI) caused by mitral valve IE during the healed phase, which was clearly proven by the pathology findings.

Key words: Infection, Valvular heart disease, Ischemic heart disease

Embolic myocardial infarction (MI) caused by infective endocarditis (IE) is rare, but it is increasingly recognized as a notable complication.1,2 Coronary embolisms are typically observed in patients with aortic valve endocarditis and is a known severe complication with a high mortality rate that may occur during the acute phase of infection.2-6 Herein, we report a case of a fatal embolic ST-elevation MI (STEMI) caused by mitral valve IE vegetation during the healed phase, which was proven on postmortem examination.

Case Report

A 79-year-old Japanese woman, who had DDD pacemaker for complete atrioventricular block implanted a year ago, was admitted to our hospital with a history of fever and headache. On admission, her body temperature was 40.3°C, and her C-reactive protein (CRP) level was 6.69 mg/dL. Transthoracic echocardiography (TTE) revealed a left ventricular ejection fraction (LVEF) of 76% and immobile mitral annulus calcification, as well as an independently oscillating mobile vegetation on the anterior mitral valve leaflet, which was 15 mm in size (Figure 1). Three consecutive blood cultures identified Streptococcus agalactiae. Accordingly, she was diagnosed with IE according to the modified Duke criteria. Three days after taking piperacillin-tazobactam under microbiology, her temperature dropped to 36°C, and her CRP level rapidly declined. Brain magnetic resonance imaging showed a small silent cerebral infarction associated with septic embolism. We proposed the need for considering surgical intervention; however, the preoperative risk scores indicated a high mortality rate (STS score, 28.9%; EuroSCORE II, 25%), and the patient and her family did not agree to go through with it.5-10 She was treated with antibiotic therapy for 4 weeks based on IE management guidelines.11 Follow-up TTE showed a decrease in the size of vegetation (8 mm) with less mobility (Figure 2). Repeat blood cultures were also negative. IE did not relapse after 4 weeks of antibiotic therapy.

Almost 3 months later, while waiting on transfer to another hospital for further rehabilitation, she suddenly deteriorated and became hemodynamically unstable. An electrocardiogram showed ST elevation in the V1-V3 and aVR leads, with reciprocal ST depression in the V4-V6, II, III, and aVF leads (Figure 3). Laboratory tests showed the following: an elevation in troponin I at 2.52 ng/mL (normal, < 0.1 ng/mL) and creatine kinase 640 U/L (normal, 41-153 U/L), white blood cell count 6600 per μL (normal, 3300-8600 per μL), and CRP 0.56 mg/dL (normal, < 0.2 mg/dL). TTE revealed an LVEF of 50% secondary to hypokinesis of the anterior segment. She was diagnosed with a STEMI and acute heart failure. Respiratory and cardiac failure rapidly deteriorated; however, her family declined invasive therapy involving primary coronary percutaneous intervention. She died on the 85th hos-
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FATAL EMBOLIC STEMI CAUSED BY HEALED IE

Figure 1. A, B: Four-chamber view of TTE showing highly mobile vegetation attached to the anterior mitral valve leaflet. Mitral annulus calcification involved both anterior and posterior segments. The vegetation size was 15 mm (arrow). C: Enlarged four-chamber view with vegetation attached to the anterior mitral valve leaflet (arrow). LA, left atrium; LV, left ventricle.

Figure 2. A, B: At 2 months after admission, follow-up TTE showed a decreased size of vegetation, measuring 8 mm, with less mobility (arrow). LA, left atrium; LV, left ventricle.

Hospital day, 1 day after the onset of acute MI (AMI).

Postmortem examination revealed subendocardial necrosis in the anterior left ventricular wall (Figure 4A). A vegetation was attached to the anterior mitral valve leaflet near the calcified annular segment, and its size was smaller than that seen on the last TTE (Figure 4B). The vegetation was mostly composed of granulation tissues and calcification without the presence of predominantly neutrophil inflammatory cells (Figure 4C). An embolus was found in the left coronary artery, which was identified as a component of the vegetation attached to the anterior mitral valves (Figure 4D). No evidence was found in the coronary arteries of plaque morphologies potentially responsible for an AMI, including plaque rupture, calcified nodule, or plaque erosion. There was no morphological abnormality of the aortic valve. No patent foramen ovale, atrial septal defect, or ventricular septal defect in the heart was found.

Discussion

We identified three key clinical features in this case: first, even healed-phase mitral valve IE could cause an AMI; second, the mechanism of STEMI caused by emboli originating from a vegetation was clearly revealed by pathology findings; and third, a STEMI secondary to IE is a high mortality complication, even when occurring during the healed phase.
This case showed that even healed-phase mitral valve IE could cause an embolic AMI. Clinically apparent AMI associated with IE is a rare complication, with an incidence rate of around 2% in patients with a definite IE diagnosis. There are varying mechanisms, including coronary compression caused by an abscess or a periaortic pseudoaneurysm; however, the most common cause is a coronary embolism. Embolic MI is typically observed in patients with aortic valve endocarditis, with a reported prevalence of approximately 80% of patients with AMI complicated with IE. This high prevalence is likely owing to the close location of the aortic vegetation to the coronary ostia. In addition, most episodes of AMI occur during the acute phase of the infection. One large prospective observational cohort study found that AMI associated with IE occurred at a mean 17 ± 8 days after the initiation of antibiotic treatments. In the 2015 ESC guidelines for the management of IE, following the initiation of antibiotic therapy, the risk of a new embolism is known to rapidly decrease after 2 weeks. In the presented case, despite occurring during the healed phase of IE, the pathology findings revealed that the AMI was caused by emboli originating from vegetation and not from unstable plaque. Although the present case had some risks of embolism, such as advanced age and a previous history of an embolic event, our patient received adequate and sufficient antibiotic therapy, and the IE considered to be under control as per her vital signs, laboratory data, blood cultures, TTE, and pathology findings. This case highlights that even IE with a stabilized clinical course can subsequently cause AMI at 3 months after effective antibiotic therapy is discontinued.

As mentioned, in this case, the mechanism of STEMI caused by emboli originating from a vegetation was apparent in the pathology findings. Most previously reported cases of coronary embolism were accidentally detected during necropsy of patients with IE because the infarction sizes were small and these patients showed no clinical manifestation of the embolism. Several case reports indirectly demonstrated the underlying mechanisms by a combination of the above findings and angiographic examinations such as coronary angiography or cardiac computed tomography. However, there have been few reports in which the clinically diagnosed AMI with mitral IE was clearly proven to be caused by coronary embolism with pathology findings. Thus, this case is novel because the embolic STEMI caused by mitral valve IE was revealed by postmortem examination.

The present case suggests that a STEMI secondary to IE is a high mortality complication, even if occurring dur-
Figure 4. A: Subendocardial necrosis was found in the anterior left ventricular wall. B: A vegetation attached to the anterior mitral valve leaflet (arrow). C: Histopathological appearance showed that the vegetation was mostly composed of granulation tissues and calcification without the presence of predominantly neutrophil inflammatory cells, objective 400 ×. Hematoxylin and eosin. D: The fragment of vegetation was found in the left coronary artery. Its size was 4.02 mm², objective 20 ×. Hematoxylin and eosin.

Concerning treatment strategy for IE itself, surgical intervention is known to reduce mortality and avoid embolic events. However, the decision to operate to prevent embolism is always difficult because IE is often associated with severe complications and a high mortality. A previous large prospective study showed that about one-quarter of patients with an indication for surgery did not undergo the operation because of sepsis or poor prognosis regardless of treatment. On the other hand, this study also revealed that patients who have a high operative risk with surgical indications who did not undergo surgery had a worse prognosis than those who did. The present case was a patient with a high operative risk; however, considering the above study, surgical intervention might have improved her prognosis. Another recent report also showed similar results using CONUT score, an index of immune function and nutritional status. Thus, we should consider the need for surgery even in patients with a high surgical risk such as the present case.

Meanwhile, as for the management of embolic AMI caused by IE, clear guidelines have not been established to date owing to the scarcity of published data. We reported a case of fatal embolic STEMI secondary to healed-phase mitral valve IE that was proven by postmortem examination. Even if the inflammation caused by the IE becomes stabilized with effective antibiotic treatments, patients with IE should be carefully followed up and the need for surgical intervention should be considered because healed-phase mitral valve IE could cause fatal embolic STEMI.

Disclosure

Conflicts of interest: None.

References


