

Acute myocardial infarction and concomitant ischemic stroke as an unusual presentation of native mitral valve endocarditis

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Abstract: ST-elevation myocardial infarction (STEMI) due to septic coronary embolism is a rare complication of infective endocarditis (IE) and is associated with high mortality rates. When common signs of IE are often overlooked on admission, the diagnosis may be established through complications, which may cause prominent symptoms. Here, we report a case of native mitral valve endocarditis with an unusual presentation with STEMI and concomitant ischemic stroke, which was due to multiple coronary and cerebral septic embolisms.

Keywords: coronary embolism, cerebral embolism, infective endocarditis, myocardial infarction, vegetation

Introduction

Although the majority of cases of acute coronary syndrome are caused by atherosclerotic disease, rarely, coronary embolisms may cause acute ST-elevation myocardial infarction (STEMI) preventing perfusion of the distal coronary bed. Infective endocarditis (IE) may be complicated with vascular complications, such as septic coronary and cerebral embolisms. IE complicated by acute myocardial infarction is associated with high mortality and morbidity rates; however, the optimal management strategy of this rare condition remains controversial because of lack of established guidelines [1]. Percutaneous coronary intervention (PCI), fibrinolytic therapy, surgical revascularization, and conservative management with antibiotherapy are the available therapeutic options. Transthoracic and more effectively transesophageal echocardiography (TEE) may help in detecting the cardiac origin of septic embolisms. Cranial computed tomography and/or diffusion magnetic

resonance imaging (MRI) may be useful to investigate concomitant cerebral embolisms. Here, we report a case of native mitral valve endocarditis with vascular complications. The unusual presentation with STEMI and concomitant stroke was due to multiple coronary and cerebral septic embolisms.

Case Presentation

A 64-year-old man was admitted to emergency service with angina pectoris and weakness on the left upper limb. His blood pressure was 110/70 mmHg and body temperature was 37.5 °C. Electrocardiography showed sinus rhythm with 2 mm ST segment elevation in leads II, III, and aVF and 1-mm reciprocal ST segment depression in leads I and aVL (*Fig. 1*). He was transferred to coronary angiography (CAG) unit with the diagnosis of acute inferior wall myocardial infarction. Subsequent CAG showed normal left anterior descending and circumflex

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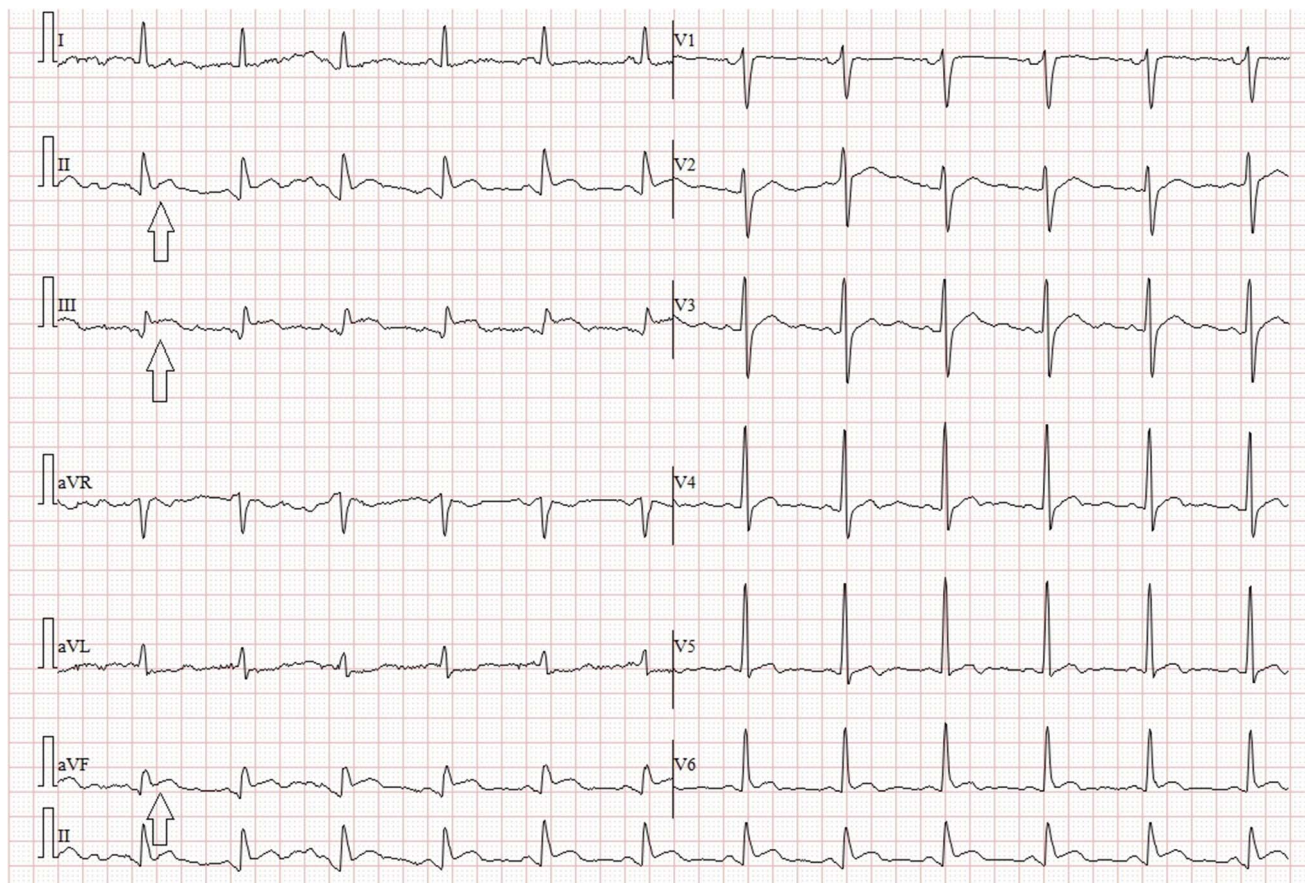


Fig. 1. Electrocardiography on admission showed sinus rhythm with 2 mm ST segment elevation in leads II, III, and aVF and 1 mm reciprocal ST segment depression in leads I and aVL

coronary arteries with a total thrombosed obstruction in the distal posterolateral segment of right coronary artery (Fig. 2A). Since the right coronary artery was normal on CAG which had been performed 2 years earlier (Fig. 2B), the total occlusion was thought to be derived from a coronary embolism. Conservative management was preferred because of small vessel size in the obstructed coronary segment. The cardiac surgery team was consulted in the acute phase; however, they refused the surgery, despite the ongoing embolization due to high surgical risk. The neurological status of the patient was progressed to left hemiparesis and partial visual loss (right homonymous hemianopia). The patient was confused and disoriented. Diffusion MRI was performed to investigate cranial perfusion. Diffusion defects were detected in a large area including left parahippocampal gyrus and left occipital lobe (Fig. 3A) with patchy infarct areas in right frontoparietal lobe (Fig. 3B). Transthoracic and subsequently TEE were performed to evaluate any cardiac origin of the two coronary and cerebral embolisms. A hypermobile mass (5 × 10 mm) isoechogenic to myocardium was revealed on P1 and P2 scallops of the posterior mitral leaflet (Fig. 4A and 4B). Laboratory tests showed leukocytosis and increased C-reactive protein. The patient received half-dose low molecular weight

heparin (enoxaparin 40 mg, twice a day) to prevent further embolisms. Empiric antibiotherapy with vancomycin (30 mg/kg/day intravenous) and gentamicin (3 mg/kg/day intravenous) was started just after taking blood samples for aerobic and anaerobic cultures. Coagulase negative *Staphylococcus aureus* was isolated from blood cultures. However, hemodynamic instability had occurred during the third day of admission and the patient passed away after a cardiopulmonary arrest.

Discussion

IE is a disease in which an infectious lesion or vegetation forms on cardiac structures, especially on heart valves. Risk factors for developing endocarditis include pre-existing rheumatic valvular lesions, congenital heart defects, prosthetic heart valves, and intravenous drug abuse. Several stigmata of IE include splinter hemorrhages, Roth spots, Janeway lesions, and splenomegaly [2]. Embolic phenomenon is common in IE, occurring in 20%–50% of patients most typically affecting the central nervous system [3]. Despite the high frequency of systemic embolization, coronary embolism remains a relatively infrequent complication.

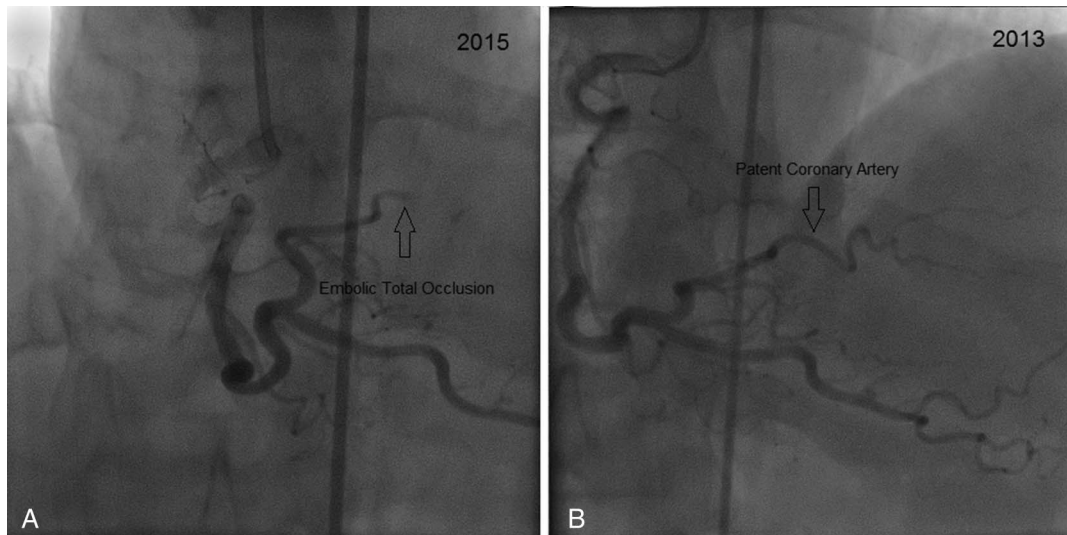


Fig. 2. (A) Coronary angiography (CAG) showed a total obstruction in the distal posterolateral segment of right coronary artery. (B) The right coronary artery was normal on CAG which had been performed 2 years earlier

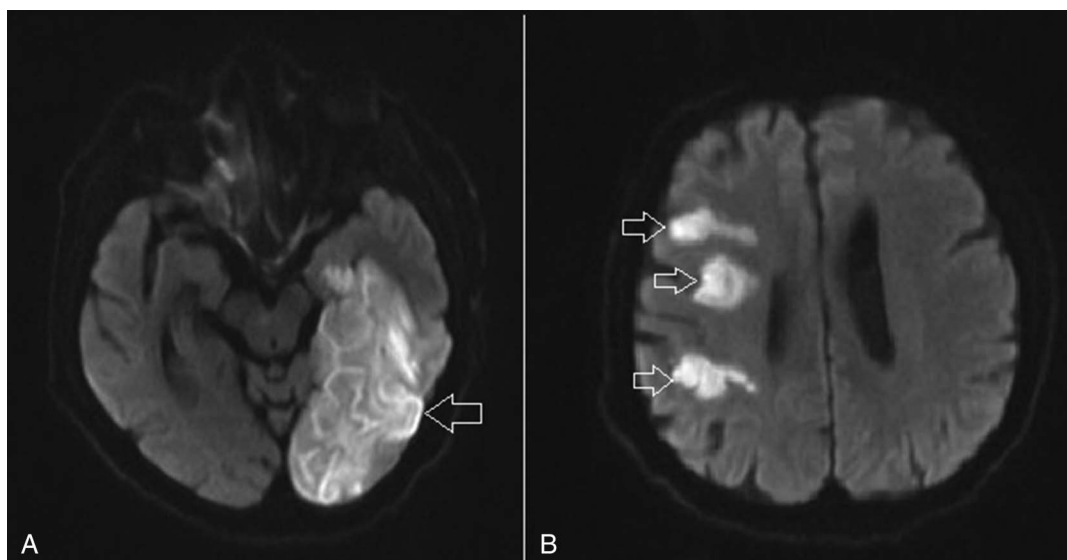


Fig. 3. Diffusion defects were detected in a large area including (A) left parahippocampal gyrus and left occipital lobe with (B) patchy infarct areas in right frontoparietal lobe on diffusion magnetic resonance imaging

TEE and CAG are the diagnostic tools for IE-associated coronary embolisms. TEE can define possible sources of embolism on cardiac structures. Certain findings on the CAG can lead to the diagnosis of coronary embolism causing acute coronary syndrome. The lack of atherosclerotic plaques and the lack of collateral vessels are the important negative signs that support the diagnosis of coronary embolism. Another important finding is the abrupt occlusions of specific coronary artery, whereas the other coronary arteries are normal as in the present case.

Although autopsy studies have shown microemboli in the coronary arteries of as many as 60% of patients with IE [4], the incidence of clinically detectable acute coronary syndrome was reported to be 0.6% [5]. Fragments from the valvular vegetation may dislodge and occlude the coronary arteries, causing an abrupt decrease in perfusion resulting in myocardial infarction. Coronary embolism most commonly occurs in the left-sided coronary arteries; however, right coronary embolization may rarely occur, as in the present case.

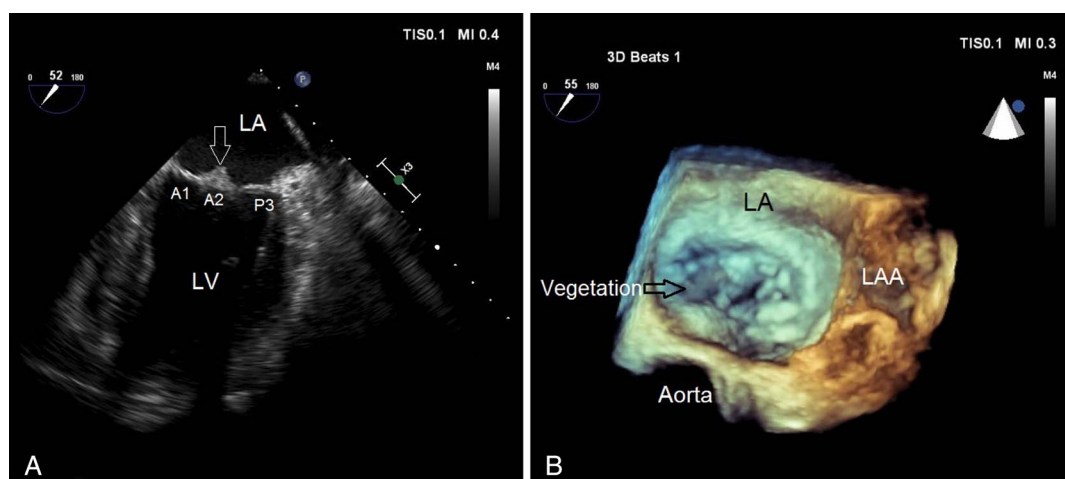


Fig. 4. (A) Two-dimensional and (B) real-time three-dimensional transesophageal echocardiography showed a hypermobile mass isoechogetic to myocardium on P1 and P2 scallops of the posterior mitral leaflet

Considering IE as a source of coronary embolism is important because its management may differ from that of atherosclerotic acute coronary syndrome. Initial management should be the same, including pain relief with nitroglycerin and morphine, supplemental oxygen, aspirin, beta-blockers, and other inhibitors of platelet aggregation and coagulation. Thrombolytic therapy should be avoided in IE-associated acute coronary syndrome because of the risk of cerebral and systemic hemorrhage. The risk of bleeding may outweigh the benefit of thrombolysis in these cases.

While conservative strategy may be appropriate for some patients, alternative therapy includes urgent cardiac catheterization and attempts to reperfuse by coronary intervention for most cases. For non-stent-based interventional management, balloon angioplasty and aspiration thrombectomy should be preferred as first-line strategies. Stenting in this setting may be associated with the risk of stent infection, mycotic aneurysm formation, and coronary perforation [6]. There are several case reports of successful catheter aspiration in appropriate situations [7]. General considerations in IE are treatment with intravenous antibiotics and subsequent surgical replacement of the infected valve as needed. The presence of resistant pathogens, ongoing bacteremia, and progressive structural cardiac compromise accompanied by worsening valve regurgitation and heart failure are the main indications for surgery. The main goal of the therapy is to prevent embolic events. Fast and proper antibiotherapy is the most effective way to reduce systemic embolism in IE [8].

Conclusions

Vascular complications of IE are frequent and life-threatening for patients. When common signs of IE are often overlooked, the diagnosis may be established

through complications, such as acute coronary syndrome and acute stroke, which may be the prominent symptoms on admission. Optimal management for acute coronary syndromes related to IE should be decided on an individual patient basis, considering the risks of hemorrhagic and embolic events.

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Abbreviations

STEMI	: ST elevation myocardial infarction
IE	: infective endocarditis
PCI	: percutaneous coronary intervention
TEE	: transesophageal echocardiography
MRI	: magnetic resonance imaging
CAG	: coronary angiography

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