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BRIEF COMMUNICATION

Percutaneous Coronary Intervention for Septic Emboli in the Left Main Trunk as a Complication of Infective Endocarditis

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Infective endocarditis (IE) complicated by acute myocardial infarction (AMI) is frequently fatal and may require emergent interventions. However, the optimal treatment of this rare condition remains controversial as it lacks established guidelines. We successfully treated a patient with IE complicated by AMI during the acute phase using percutaneous coronary intervention (PCI) followed by surgery. A 73-year-old man was diagnosed with IE of the mitral and aortic valves caused by *Streptococcus oralis*. Four weeks after the initiation of antibiotics sensitive to the causative bacteria, he suddenly developed AMI manifested by chest pain and dyspnoea with cardiovascular collapse. Emergent coronary angiography revealed that the myocardial infarction was secondary to septic emboli in the left main trunk. Emergent PCI comprising aspiration and stent deployment, was successfully performed, and his vital signs were immediately stabilised. He subsequently underwent mitral and aortic valve replacement and debridement without major post-operative complications. Although the optimal treatment strategy for haemodynamically unstable AMI secondary to IE requires further discussion, the present case indicates the importance of early diagnosis and the potential effectiveness of aggressive PCI as a bridge to the following surgery.

Keywords Myocardial infarction • Percutaneous coronary intervention • Infective endocarditis • Coronary emboli • Left main coronary artery

Introduction

We report a patient who was diagnosed with infective endocarditis (IE) of the mitral and aortic valves caused by *Streptococcus oralis*. After initiation of antibiotic therapy, he developed acute myocardial infarction (AMI) that was successfully treated during the acute phase by percutaneous coronary intervention (PCI) followed by later surgery. The myocardial infarction was found to be secondary to septic emboli in the left main trunk (LMT). Acute myocardial infarction as a complication of IE is frequently fatal and may require emergent interventions. However, the optimal treatment of this rare but important condition remains controversial. Sharing experiences of case reports may lay a foundation for establishing guidelines in this field. The present case indicates the importance of early diagnosis and the potential effectiveness of emergent PCI in these patients.

Clinical Case

A 73-year-old man with a two-month history of intermittent fever, weight loss, and fatigue following a dental procedure that had taken place six months previously was referred to

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our cardiovascular institute for investigation of suspected IE. Physical examination showed body temperature of 38.9 °C, heart rate of 85 beats/min, blood pressure of 107/42 mmHg and peripheral oxygen saturation of 94%. He had a Levine grade II/VI diastolic heart murmur at the upper right sternal border. His electrocardiogram was within normal limits. Chest radiography showed mild pulmonary oedema and pleural effusion. Laboratory tests showed a white blood cell count of 14.69 × 10^3/μL with neutrophilia, a serum C-reactive protein level of 6.28 mg/dL, and a plasma brain natriuretic peptide level of 768.8 pg/mL. Blood cultures were positive for *Streptococcus oralis.* Echocardiography showed a mobile mass of approximately 10 × 7 mm attached to the left coronary cusp, a mobile mass of approximately 5 × 5 mm attached to the right coronary cusp, prolapse of the left cusp with severe aortic regurgitation, a 14 × 5 mm mass attached to the anterior leaflet of the mitral valve, and prolapse of the middle anterior leaflet with moderate mitral regurgitation (Figure 1A). The left ventricular ejection fraction was preserved (60%), but the left ventricular dimension was increased (left ventricular end-diastolic/systolic dimension = 62/37 mm). A diagnosis of IE was made according to the Duke diagnostic criteria [1]. The patient was initially treated with antibiotics (benzylpenicillin and gentamicin) and diuretics, which alleviated his heart failure symptoms and gradually improved the inflammatory markers levels. An integrative intracranial investigation that included brain magnetic resonance imaging showed findings of acute cerebral infarction, and surgery was scheduled.

Four weeks after the initiation of antibiotics, however, he developed sudden chest pain and dyspnoea with cardiovascular collapse (systolic blood pressure 78 mmHg). An electrocardiogram showed atrioventricular junctional rhythm with ST-segment elevation in leads I, aVL, V5, and V6 and ST-segment depression in leads V1, V2, and V3 (Figure 1B). Following administration of antiplatelet agents (clopidogrel 300 mg and aspirin 200 mg), emergent coronary angiography was performed that revealed a severely obstructed distal

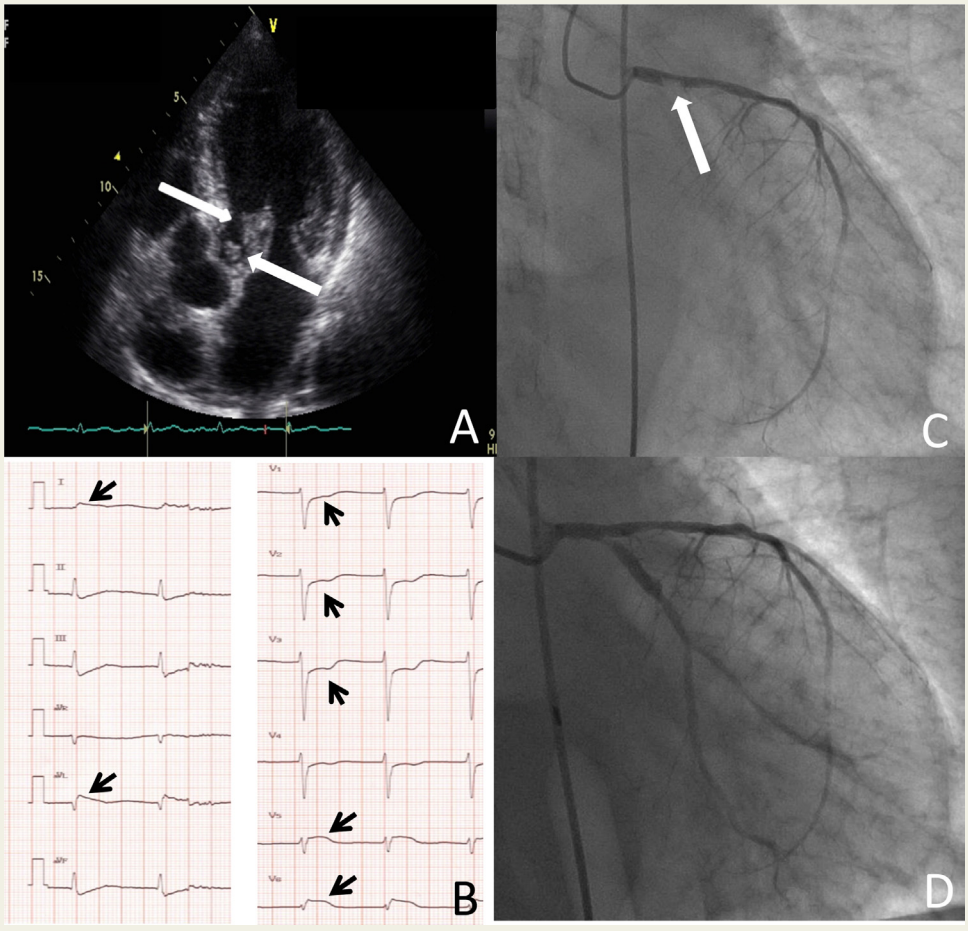

Figure 1 (A) Echocardiography reveals area of vegetation (white arrow). (B) Electrocardiogram shows atrioventricular junctional rhythm with ST-segment elevation in leads I, aVL, V5, and V6 and ST-segment depression in leads V1, V2, and V3 (black arrows). (C) Pre-treatment coronary angiography reveals septic emboli (white arrow) in the left main trunk. (D) Post-treatment (aspiration thrombectomy and stent deployment) angiography shows optimal coronary artery flow.