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Infective Endocarditis Revealed by an Acute Myocardial Infarction: A Case Report

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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Case Report

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ABSTRACT

Acute myocardial infarction caused by septic embolism is usually fatal. A 43-year-old male patient presented to the emergency department with severe chest pain within 4 hours. An electrocardiogram showed prominent anterolateral ST elevation in precordial leads (V2-V6) and lateral leads (DI and aVL). His body temperature was 38.3°C. He underwent blood cultures prior to parenteral antibiotic therapy. Staphylococcus aureus has been confirmed. Coronary angiography was performed and revealed a septic embolism in the distal left circumflex artery. Transthoracic echocardiography showed. migratory vegetation on the mitral valve. After 6 weeks of antibiotic

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therapy, he was completely cured and discharged. He was readmitted 6 months later and died of complications of recurrent infective endocarditis.

Keywords: Infective endocarditis; acute myocardial infarction; blood pressure; right coronary artery (CAD).

ABBREVIATIONS

IE : Infective Endacarditis
CAE : Coronary Artery Embolism
AMI : Acute Myocardial Infarction
ECG : Electrocardiogram
LCx : Left Circumflex Artery
TTE : Transthoracic Echocardiography
LAD : Left Main Artery
CAD : CAD

1. INTRODUCTION

"Infectious endocarditis (IE) is a bacterial or fungal infection of the heart valve or endocardial surface" [1]. "IE can present with a variety of clinical syndromes that make early clinical diagnosis elusive" [2]."IE often forms vegetation on heart valves and can lead to systemic embolism. Transplanted vegetation rarely leads to coronary embolism (CAE) and subsequent acute myocardial infarction (AMI)" [3].We report a rare case of AMI as the first symptom of IE. AMI was caused by CAE, a rare complication of IE.

2. CASE REPORT

A 43-year-old man with no known risk factors for coronary artery disease , presented to the emergency department with sudden left precordial chest pain with shortness of breath that began 4 hours before his presentation. Physical examination revealed that his temperature was 38.3°C, his blood pressure was 113/67 mmHg, his heart rate was 112 beats/min, his oxygen saturation in room air was 97%, and his respiratory rate was 23 beats/min. Cardiovascular examination revealed a Levine grade II/VI systolic murmur at the apex. Upon arrival, an electrocardiogram (ECG) and measurements of myocardial enzymes, including highly sensitive cardiac troponin T and creatine kinase-MB, were performed. ECG (Fig. 1) showed sinus tachycardia with acute ST elevation in the D1-aVL and V2-V6 leads. The high-sensitivity cardiac Troponin T level was 70.3 ng/mL. Based on clinical presentation and electrocardiographic findings, the patient was referred for percutaneous coronary intervention. An emergency coronary angiography was performed, showing normal blood flow in the left main artery (LAD) and right coronary artery (CAD). The distal left circumflex artery (LCx) was abruptly occluded, causing thrombolysis in grade 2 myocardial infarction flow. Thrombus aspiration was performed and the embolus migrated to the proximal LCx. Antiplatelet agents and anticoagulants were used after coronary angiography.

Subsequent transthoracic echocardiography (TTE) (Fig. 2) showed mildly impaired left ventricular systolic function with anteroseptal hypokinesia and severe anteriorly oriented mitral valves seen on the atrial surface of the mitral valve anterior leaflet and on the roof of the left atrium. Anterior septal hypomotility with regurgitation and vegetation was evident, and blood cultures showed Staphylococcus aureus. Laboratory tests showed that the white blood cell count and C-reactive protein levels had increased to 17.07 x 109 cells/L and over 250 mg/L, respectively. His procalcitonin level was 3.38 ng/ml and he was anemic with hemoglobin 8.6 g/dl. IE was definitively diagnosed and empirical intravenous antibiotic therapy for infective endocarditis was initiated and was based on ceftriaxone 2 g daily.

His condition he stabilized after a month. His infection was brought under control and his symptoms, including fever, chest pain and shortness of breath, eased. Repeated transthoracic echocardiography revealed 8 × 7 mm recession of vegetation at the posterior leaflet of the mitral valve, severe mitral regurgitation.

At the first admission, we were on conventional antibiotic therapy for 6 weeks. He made a full recovery and no organisms were produced on monitor blood cultures.For this reason; no surgical intervention was performed. Half a year later, he was hospitalized again for a recurrence of his IE, showing many mobile vegetation on his mitral valve. The vegetation has grown larger than the previous TEE. A diagnosis of subacute infective endocarditis was made, and two antibiotic therapy (parenteral ceftriaxone 2 g and amikacin 15 mg/kg daily) was restarted. Acute ischemic hepatitis was diagnosed by cutaneous

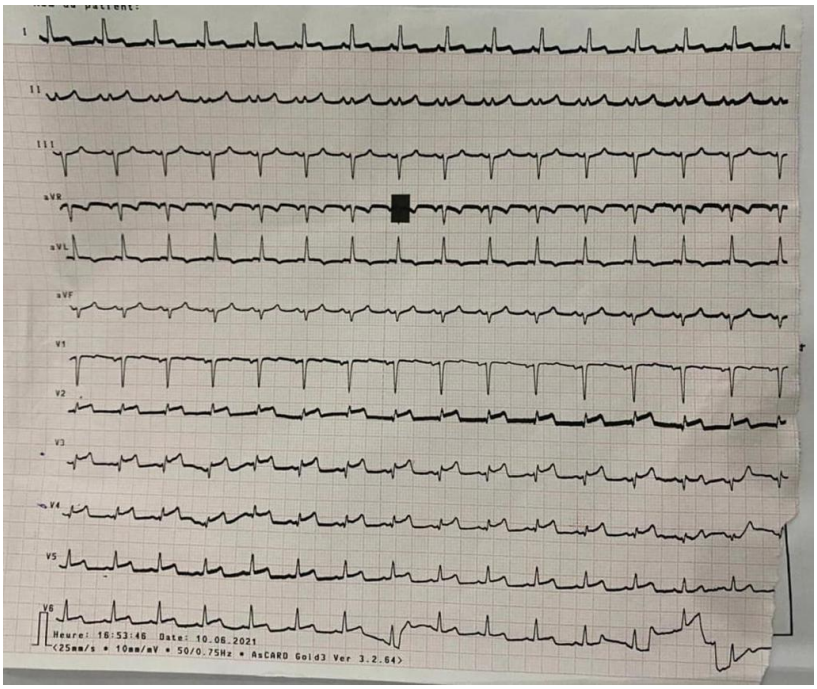

Fig. 1. ECG with acute ST elevations at D1-aVL andV2-V6 derivations

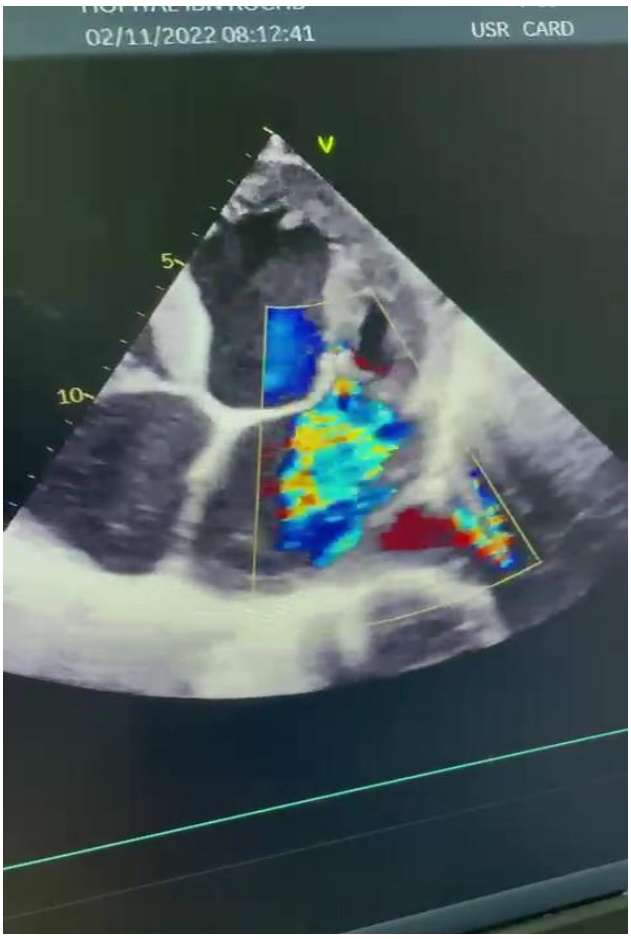

Fig. 2. TTE showed mitral regurgitation and vegetation on the atrial surface

jaundice and slightly elevated levels of aspartate aminotransferase, alanine aminotransferase, direct bilirubin, and total bilirubin. Rifampicin was discontinued due to acute ischemic hepatitis. Cardiac arrest occurred 6 days later, the patient was unresponsive to cardiopulmonary resuscitation, died of septic complications, and was unresponsive to treatment.

3. DISCISSION

“Coronary embolism has been reported in 4% to 13% of patients with acute ST-segment elevation, 8% of which is due to infective endocarditis” [4]. “Most coronary embolisms occur in her LAD. Perhaps its launch and descent course is more advantageous than the vertical launch of RCA and LCX. He is also at risk in IE for SCE from the anterior mitral leaflet to primarily involve her LAD artery, LCX and RCA, respectively”[5]. “The highest incidence of embolic complications in mitral valve disease is observed during the course of IE” [6]. We report fatal coronary sepsis embolism when associated with acute myocardial infarction. No atherosclerotic plaques were detected in his coronary arteries. The appearance of emboli in the LAD was typical of septic embolism. “Septic coronary embolism was a relatively common finding in his previous autopsy studies of IE.AMI is rarely diagnosed throughout life and is usually fatal” [7,8]. “A serious complication of IE is embolic events caused by migration of cardiac vegetation. Distant vegetation can embolize blood vessels throughout the body, leading to ischemic events. Important predictors of embolism in IE are intravenous drug use, S. aureus infection, mitral valve vegetation, and vegetation size >10 mm” [9]. “IE should be treated aggressively to prevent future embolic events. In addition to antibiotics, early valve surgery is recommended for patients with IE and valve dysfunction or recurrent embolism” [10].

4. CONCLUSION

This report describes the process of diagnosis and treatment of cases of AMI with CAE, a rare complication of IE, to guide clinical management. Performing surgical intervention on diseased heart valves after aspiration of coronary embolism during coronary angiography is a plausible strategy for treating CAE in IE.

CONSENT

As per international standard or university standard, patient(s) written consent has been collected and preserved by the author(s).

ETHICAL APPROVAL

As per international standard or university standard written ethical approval has been collected and preserved by the author(s).

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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