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# A Coronary Septic Embolism in Double Prosthetic Valve Endocarditis Presenting as Acute Anteroseptal ST-Segment-Elevation Myocardial Infarction

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## ABSTRACT

Acute myocardial infarction caused by septic embolism is usually fatal. A 40-year-old male patient who presented within 30 minutes of severe chest pain was admitted to the emergency department. An electrocardiogram showed a maximum 6 mm of acute ST elevations at V1-V4 derivations. His body temperature was as high as 38.5°C. Blood cultures were taken three times before parenteral ampicillin/ sulbactam treatment was administered. Later, coagulase negative staphylococci (Methicillin Sensitive *Staphylococcus epidermidis*) were identified from his blood cultures. Coronary angiographic examination was performed. Lobulated contours of a septic embolus was shown in the mid region of left anterior descending artery as an outcome. Trans-esophageal echocardiography showed; mobile multiple vegetations on the prosthetic mitral and aorta valves. After six weeks of antibiotherapy, he was completely healed and discharged from hospital.Six months later, he was rehospitalized and died because of complications of recurrent infective endocarditis.

**Key Words:** Infective endocarditis, prosthetic valve, coronary septic embolism, myocardial infarction, trans-esophageal echocardiography

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## Introduction

Septic coronary artery embolism (SCE) is a well-recognised complication in infective endocarditis (IE) and rarely causes ST-segment-elevation in myocardial infarction (STEMI). However, it is not well known in prosthetic valve endocarditis (PVE) (1). Having established the diagnosis of IE, which constitutes a therapeutic dilemma such as PVE and SCE, conventional anti-ischemic and antibiotic administration was started for the clinically stable patient. Because of the presence of infection, invasive and surgical interventions were avoided. Instead, clinical care and monitorization were preferred (2).

We report an extremely rare case of acute STEMI which was fatal and arose from the embolism of bacterial vegetation with staphylococcus endocarditis of prosthetic mitral and aortic valves in a man.

## Case Report

A 40-year-old man with severe chest pain of astringent type, spreading to the left arm for 30 minutes, who had no known coronary artery disease risk factors, was admitted to the emergency department in our hospital. An electrocardiogram (ECG) showed 6 mm acute ST segment elevations on V1-V4 and aVR and 2 mm ST segment depression on the other derivations (Figure 1A). On physical examination, the patient was febrile (38.5°C) and the heart rate was 90 beats/min/irregular. On all cardiac foci a 3/6 systolic murmur and prosthetic valves sounds were auscultated.

He revealed rheumatic aortic and mitral valves disease. Aortic Valve Replacement (AVR; Medtronic 22) and Mitral Valve Replacement (MVR; Medtronic 29) operation had been performed in another cardiac center ten years previously.

The patient was later transferred to the coronary intensive care unit (CICU) with the diagnosis of acute STEMI. Clopidogrel 300 mg, metoprolol 50 mg, ramipril 2.5 mg, atorvastatin 40 mg, lansaprazol 30 mg once time daily were started per oral and for intravenous (IV) therapy, heparin 1000 microgram/minute and nitrate in perfusion were administered. His chest pain ceased after nitrate perfusion treatment. When the patient's ST segment elevation decreased more than 50%, it was decided that spontaneous reperfusion had occurred. Therefore, no thrombolytic therapy was administrated. In the meantime, a ventricular fibrillation attack was detected and DC shock was applied immediately. Sinus rhythm returned accompanied by a B-Type WPW pattern. Upon the follow-up, troponin T measured 0.02 on admission and later increased to 14.7 ng/mL at the sixth hour. The patient's laboratory data were: erythrocyte sedimentation rate 90/h, International Normalized Ratio (INR) 2.1, hematocrit 32%, hemoglobin 9.5 gr/dL, lactate dehidrogenase 434 U/L and blood urea 51 mg/dl. C-reactive protein was detected as 127 mg/L and the patient's urine examination results contained many erythrocytes. Other biochemical tests were normal. Blood cultures were taken three times and then parenteral ampicillin/sulbac-

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tam antibiotherapy was started. Coagulase negative staphylococci (Methicillin Sensitive *Staphylococcus epidermidis*) were grown on blood cultures. *S. epidermidi*s was susceptible to erythromycin, gentamicin, clindamycin, ciprofloxacin, vancomycin and teicoplanin. *S. epidermidis* was resistant to tetracycline and sulfametoksazol/trimethoprim. On the sixth day of antibiotherapy, gentamicin 80 mg twice daily was added. After the first 48 hours of gentamicin therapy; the patient's body temperature returned to normal.

Transthoracic echocardiography (TTE) examination indicated anteroseptal hypokinesia with left atrium 5.7 cm, left ventricular diastolic diameter 5.1 cm, right ventricular diameter 2.1 cm, interventricular septum 1.1 cm, ejection fraction (EF) 55%, prosthetic aortic valve mean gradient of 30/50 mm Hg and prosthetic mitral valve mean gradient of 8 mm Hg. When INR results returned to normal levels, it was decided to perform coronary angiography (CAG). The patient's CAG examination showed (LAD), lobulated contour in the mid left anterior descending coronary artery and existence of septic embolism due to infective endocarditis. Left circumflex coronary artery (LCX) and right coronary artery (RCA) were normal and atheroma plaques were not present in the coronary arteries (Figure 1B). For further evaluation trans-esophageal echocardiography (TEE) examination was performed, mobile vegetations were detected in sizes of 0.24x0.26 cm on the prosthetic aortic valve and 0.30x0.45 cm on prosthetic mitral valve (Figure 2A, B). No invasive intervention was performed on the lobulated septic embolus. After six weeks of conventional antibiotherapy, he was discharged from hospital in full health. No microorganism was produced on surveillance blood cultures.

After six months, he was rehospitalized because of weakness, cough, abdominal pain and pallor of the skin, while taking benzathine penicillin. The patient had lost weight and he was dyspneic. His sclera and skin were icteric. Furosemide, digitalis and B-blockers were added to the treatment. Later, TTE and TEE were reestablished. EF was measured as 35%. Many mobile vegetations were seen, one of which was the largest, measuring 0.60x0.30 cm, on mitral and aortic valves. Compared to the previous TEE, vegetations were enlarged. Subacute infective endocarditis was diagnosed and triple antibiotic therapy regimen (orally; rifampicin 300 mg three times daily and parenterally meropenem 1g and sefazolin 1g three times daily) restarted. Acute ischemic hepatitis was diagnosed by the icteric skin and the marginally high values of aspartate aminotransferase, alanine aminotransferase, direct bilirubin and total bilirubin. Rifampicin was discontinued because of acute ischemic hepatitis. Six days later, cardiac arrest developed and the patient did not respond to cardiopulmonary resuscitation (CPR) and died.

## Discussion

Most coronary embolization occurs in the LAD. Possibly its take off and downward course is more favourable than the perpendicular take offs of the RCA and LCX. Again, in IE the risk of involving SCE from the mitral anterior leaflet exists mostly in the LAD artery, LCX and RCA, respectively. The LAD involvement has a better prognosis than others, also appropriate antibiotic regimens reduce the risk (2).

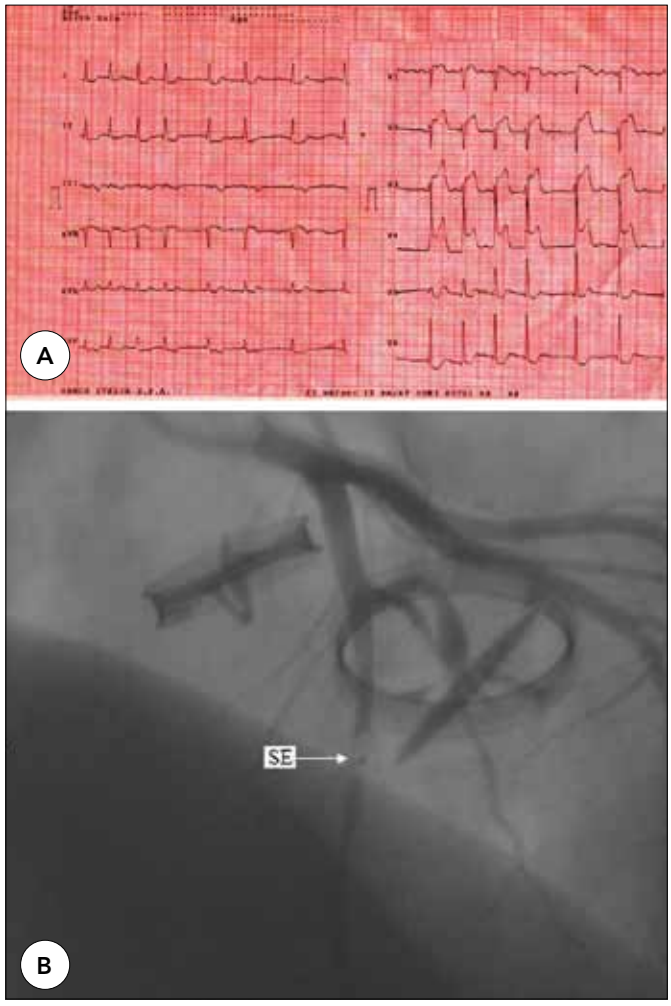


**Figure 1. A.Twelve-lead electrocardiogram during acute chest pain associated with STEMI, B. Coronary angiogram showing a possible septic mobile thrombus (contrast defect arrowed) in the mid left coronary artery (LAD); (SE denotes septic thromboembolism)**

In the course of IE, the highest incidence of embolic complications is seen with mitral valve disease. It is even higher for vegetations present on the anterior mitral leaflet. Infective endocarditis due to *Staphylococcus aureus*, *candida*, HACEK (*Haemophilus, Actinobacillus, Cardiobacterium, Eikinella, Kingella*) and Abiotrophia microorganisms are also at higher risk for embolization. As rare complications of IE, young patients who developed a high body temperature accompanied by acute MI, should be suspected of a septic embolus disease (1).

We report a coronary septic embolism that was demonstrated in double PVE which was fatal when complicated with acute STEMI. In his CAG, atherosclerotic plaques were not detected. The appearance in the LAD of the embolus was typical for septic embolus. Also, all the criteria of PVE in our patient were present.

The treatment strategy is to identify the actual problem. Our patient was administrated anticoagulation therapy, but, anticoagulation does not prevent septic embolization and in some cases may contribute to intracranial haemorrhage, particularly in the presence of recent cerebral infarct or mycotic aneurysm

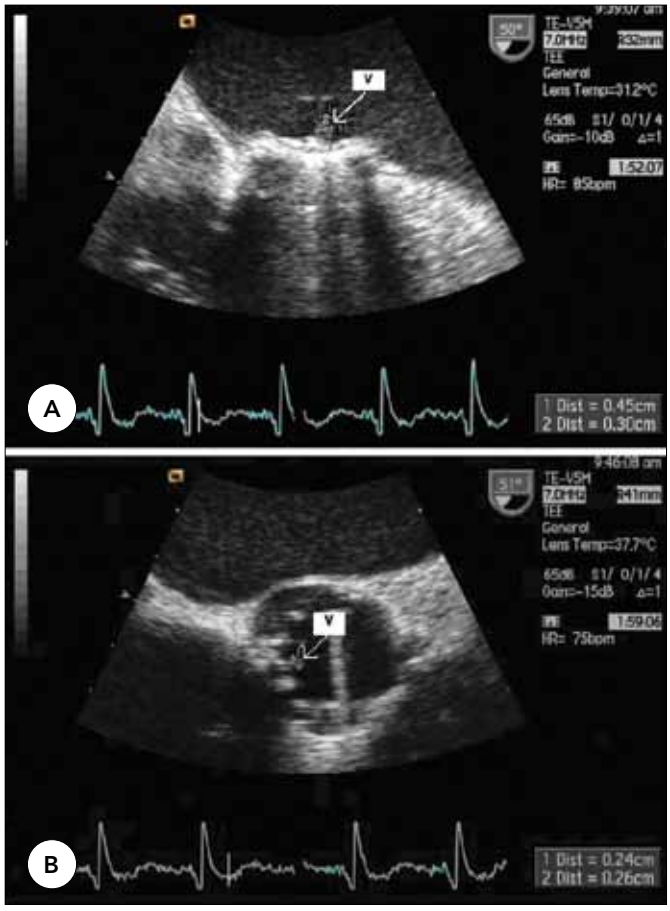


**Figure 2. A. Trans-esophageal echocardiogram demonstrating a mobile vegetation on the ventricular surface of the prosthetic mitral valve (V denotes vegetation), B. Trans-esophageal echocardiogram demonstrating a mobile vegetation on the ventricular surface of the prosthetic aortic valve (V denotes vegetation)**

(3). Experience with thrombolytic therapy has generally been unfavourable and associated with high risk of complication rates and the efficiency is low. It can be harmful, indeed, moreover in some cases, the risk of concurrent intracerebral mycotic aneurysms and intracranial haemorrhage is higher in patients with IE whose treatment involves thrombolytic agents (4, 5).

Herzog et al. (6) in 1991 reported that the use of coronary balloon angioplasty in the treatment of patients with septic embolization, caused acute myocardial infarction. Bacteremia of IE constitutes a relative contraindication to Percutaneous Transluminal Coronary Angioplasty (PTCA). Potential cardiac complications of septic coronary embolization during PTCA are mycotic septic aneurysm, perforation, carditis and resistant vegetations. CAG is reliable in active IE, but PTCA effectiveness and reliability is generally not clear. The stent implantation after PTCA is recommended, to prevent coronary aneurysm in the area of septic aspiration and intracoronary stent placement may be an option (7). Percutaneous invasive interventions are the safest reperfusion strategy in IE, but in patients with bacteremia under infection the PTCA and stent applications can constitute a risk of coronary artery septic mycotic aneurysm and perforation. Also, vegetations are more resistant than fresh thrombi to PTCA intervention so that the chance of success compared to PTCA is decreased (1, 7-9). Another possible alternative solution in some challenging cases may be surgical (10).

While he was hemodynamically stable on observation, no invasive intervention was performed to the coronary arteries because risk of a recurrent ischemic complication had been considered. Also, it was decided that spontaneous reperfusion had occurred. Thus, thrombolytic therapy was not started.

We continued the conventional antibiotic treatment for six weeks in the first hospitalization and he was discharged fully recovered, and no microorganism was produced in his surveillance blood cultures. Therefore; surgical intervention was not performed. After six months, he was rehospitalized because of recurrence of IE, but he died because of septic complications and did not respond to medical treatment.

Septic coronary embolism was a relatively common finding in earlier autopsy studies of IE. Acute MI is seldom diagnosed during life and is usually fatal (1, 2).

## Conclusion

As rare complications of IE, young patients who develop high body temperature accompanied by acute MI, should be suspected of having a septic embolus disease. STEMI treatment in a septic coronary embolism is still controversial and treatment regimens and strategies should be modified individually.

### Conflict of Interest

No conflict of interest was declared by the authors.

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