# Uncertainties in managing myocardial infarction associated with infective endocarditis

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Embolic myocardial infarction is an uncommon but increasingly recognised complication of infective endocarditis. This complication has a high mortality rate and is deemed a relative contraindication to thrombolytic therapy. The present article describes an episode of acute myocardial infarction associated with infective endocarditis. Systemic thrombolytic therapy was administered, which resulted in resolution of cardiac ischemia but was complicated by a fatal intracerebral bleed. There are a number of published cases describing the use of systemic thrombolysis, primary percutaneous intervention and early valvular surgery in this circumstance, but the optimal course of treatment for myocardial infarction in the context of infective endocarditis remains to be elucidated. Additional guidance for those who are likely to encounter this condition in clinical practice would be welcomed.

**Key Words:** *Complications; Infective endocarditis; Percutaneous intervention; Thrombolysis*

Embolic myocardial infarction is an uncommon but increasingly recognized complication of infective endocarditis and is deemed a relative contraindication to thrombolytic therapy. The mortality and morbidity rates associated with this presentation are high, despite the application of a number of management approaches including conservative medical management, thrombolytic therapy, primary percutaneous intervention and early elective valvular surgery (1-17). Affected individuals comprise a heterogeneous group and may follow a fulminant course that precludes successful percutaneous intervention or surgery (1-4). A review of the literature indicates that coronary artery emboli are equally likely to originate from mitral and aortic valves, usually precipitating predominantly anterior wall myocardial infarction, with inferior myocardial infarction occurring less frequently. In the present article, an unusual case of inferior ST elevation myocardial infarction as a consequence of infective endocarditis is reported. The paucity of information available to guide management in this circumstance is also discussed.

## CASE PRESENTATION

A 54-year-old woman with an unremarkable medical history was referred to the medical assessment unit for evaluation. She had a six-week history of fever, night sweats, weight loss and intermittent transient memory impairment. On physical examination, she appeared cachectic and had low-grade pyrexia. There were no peripheral stigmata of infective endocarditis, although an apical pansystolic murmur was heard. The examination was otherwise unremarkable and, notably, she had no focal neurological deficit.

Hematological investigations revealed normocytic anemia, neutrophilia and reactive thrombocytosis. Renal function was normal and liver function tests were deranged with a hepatitic picture. Urinalysis and a plain chest radiograph were both normal, and electrocardiography demonstrated a normal sinus rhythm.

The diagnosis of infective endocarditis was confirmed following isolation of *Streptococcus* from multiple blood cultures, and appropriate antibiotic therapy was commenced. A transthoracic echocardiogram identified vegetations on both the anterior and posterior mitral valve leaflets with mitral regurgitation. The left atrium was normal and left ventricular function was preserved. In view of the transient memory impairment, a computed tomography (CT) brain scan was performed (without the use of contrast, due to a known iodine allergy). The CT scan revealed a curvilinear calcification in the left parieto-occipital region, with no surrounding edema and mass effect. At the time of CT examination, this was not believed to be an acute pathology.

Two days following admission, the patient developed severe, acute chest pain with inferior ST segment elevation and underwent thrombolytic therapy for acute inferior myocardial infarction using tenecteplase, followed by an intravenous bolus of 3000 units (30 mg) of enoxaparin and subsequent weight-based subcutaneous injection. One hour after thrombolysis, an electrocardiogram demonstrated good resolution of the ST segment changes, but the patient rapidly became obtunded and hypertensive, and developed a right hemiparesis. A CT brain scan without contrast was performed immediately and confirmed the presence of an extensive subarachnoid and intraventricular hemorrhage, which was not amenable to surgical intervention and rapidly became fatal.

Subsequent postmortem examination confirmed the presence of coronary atherosclerosis, and death was deemed to be due to massive hemorrhage into a cystic cerebral infarct.

## DISCUSSION

The present case highlights the difficulties in deciding how best to manage acute ST elevation myocardial infarction in the context of infective endocarditis and is unusual in that none of the previously reported cases have been associated with proven atherosclerotic disease of the coronary arteries. Embolic myocardial infarction is an increasingly recognized but uncommon complication of infective endocarditis and is deemed a relative contraindication to thrombolysis due to the risk of embolic events, including cerebral infarction, which may predispose to hemorrhagic complications. Embolic events have been estimated to occur in 22% to 44% of cases of infective endocarditis, with cerebral manifestations being more common than peripheral manifestations. Features that predict an increased risk of embolism include the presence of a mobile vegetation, vegetation length of more than 10 mm and infection with staphylococci or non-viridans streptococci as the causative organism. The risk of embolism decreases after the commencement of antibiotic therapy (18).

When considering intravenous thrombolytic therapy for acute myocardial infarction, it is imperative to assess the risk of precipitating severe or life-threatening hemorrhage. Absolute contraindications to

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thrombolytic therapy are well recognized and include known recent or active bleeding, any previous hemorrhagic stroke and a history of ischemic stroke within one year. Low molecular weight heparin is usually given with thrombolytic agents to reduce the risk of reinfarction; however, this increases the risk of major bleeding (19). Intracerebral hemorrhage complicating systemic thrombolysis has been reported in three of six cases of myocardial infarction associated with infective endocarditis, two of which were fatal and one of which required neurosurgical intervention (5-10). Another patient treated with thrombolytic therapy sustained an acute upper gastrointestinal bleed requiring transfusion (13).

There appears to be a trend toward the use of primary percutaneous intervention; however, evidence regarding the safety and efficacy of this approach is also limited. Such interventions may be complicated by a high failure rate, which appears to be the case with balloon angioplasty, and an increased risk of further embolic phenomena, coronary artery mycotic aneurysm formation or stent infection (1,7,12,15). Successful outcomes have been described in some patients undergoing primary percutaneous intervention (8,13,14). Urgent valve replacement and bypass grafting have also been successful in some cases (7,12,13,16,17).

In summary, the optimal management of myocardial infarction as a consequence of infective endocarditis remains to be elucidated, and there is currently little evidence to support either systemic thrombolysis or primary percutaneous intervention in this situation. In patients who can be stabilized with conservative medical management, early valvular surgery may be the safest option. Further guidance for those who are likely to encounter this condition in clinical practice would be welcomed, but in the interim, optimal management must be decided on an individual patient basis, taking into consideration the risks of severe hemorrhage, embolic phenomena and the complications associated with percutaneous intervention including reocclusion, mycotic aneurysm and stent infection.

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