INTRODUCTION
Infective endocarditis (IE) is associated with a number of complications. Peripheral systemic embolism is a common and serious complication of infective endocarditis linked to migration of vegetations. The brain and spleen are the most common place in endocarditis involving left side of the heart, while pulmonary embolism is common in native valve endocarditis involving right side of the heart and endocarditis related to pacemaker lead. Myocardial infarction is a rare complication and can arise due to coronary embolism and external coronary compression. Coronary ischaemia can also be due to a large vegetation obstructing the coronary ostium or severe aortic regurgitation. Surgery on left side of the heart for endocarditis can also lead to coronary emboli and myocardial infarction. These complication can pose serious management issues as described in the present case report.

CASE REPORT
A 26 years old female presented to the emergency department with sudden onset of chest heaviness and dyspnoea since 3 hours. The pain was central with radiation to both arms. She had no history of such complaints in the past. Systemic review of her history revealed that she had low grade fever since one year, which was associated with fatigue and weakness. She had no risk factors for ischaemic heart disease. She had suffered an ischaemic stroke one year back; was later diagnosed as tuberculous meningitis and started on anti-tuberculous treatment.

At presentation, she had a temperature of 99.5°F and was well oriented in time, place and person. Her blood pressure was 110/70 mmHg and pulse 110/minute and regular. Chest auscultation revealed occasional wheezing, a diastolic murmer at left sternal border and a systolic murmer at the apical area. Chest radiograph was performed on the day of admission, which showed increased cardiothoracic ratio and clear lung fields. Electrocardiogram was abnormal with ST-segment elevation in leads V1 to V6, Q-waves in lead II,III and aVF and poor R wave progression (Figure 1). Serum creatinine kinase (CK-MB) was raised more than eight times above the normal value and showed decline in level at 5th day after myocardial infarction. BUN, creatinine and electrolytes were within normal limits. Complete blood count performed few hours after admission showed white cell count of 16,000/cubic millimeter and a haemoglobin of 10.5 g%. Transthoracic echocardiography performed on next day demonstrated

Figure 1: Electrocardiogram showing antero-lateral myocardial infarction.
severe aortic regurgitation, mild to moderate mitral regurgitation and mobile echogenic masses attached to non-coronary and right coronary cusps and also on atrial side of anterior mitral leaflet. Left ventricular diastolic and systolic dimensions were 64 mm and 39 mm respectively and ejection fraction was 40% with anterior-inferior wall hypokinesia. Blood culture identified *Streptococcus* species sensitive to chloramphenicol, penicillin and vancomycin and resistant to erythromycin and ciprofloxacin. Penicillin minimum inhibitory concentration was 0.06 microgram/ml. During acute presentation in ER, treatment for acute coronary syndrome was given which also include thrombolyis but results were not satisfactory. The ECG after thrombolyis showed only minor resolution of ST-elevation and developed Q-waves in the follow-up electrocardiogram. Empirical treatment was started with benzylpenicillin and gentamycin and the patient responded well clinically and also by decrease in total leukocyte count. She was treated for 4 weeks.

**DISCUSSION**

Embolism to coronary arteries is a documented complication of bacterial endocarditis but it rarely causes acute myocardial infarction. The need for speedily restoring coronary artery blood flow and the little time available for clinical decisions may prevent diagnosis of endocarditis before any pharmacologic or mechanical thrombolyis can be carried out. In this case it was realized late during acute presentation and we performed pharmacologic thrombolyis although we could have attempted mechanical thrombolyis in this case. A case report described the first documented cases of coronary angioplasty in 2 patients with acute myocardial infarction caused by bacterial endocarditis, and reviews the literature on coronary artery complications of bacterial endocarditis. The first patient developed a coronary artery aneurysm and the second experienced a small intracerebral haemorrhage following reperfusion. It is not a good idea to generalize from two cases, but we consider that in patients in whom the endocarditis is the cause of acute myocardial infarction, the use of conventional strategies for coronary reperfusion should be done cautiously because of risk of possible detrimental consequences.7

There is inadequate information available to guide us about the best treatment options in patients who present with such difficult scenarios. A case report of coronary embolism secondary to aortic valve endocarditis showed that it was treated with streptokinase and aspirin. The patient survived but suffered a large myocardial infarction and a major gastrointestinal bleed. When myocardial infarction is due to coronary embolism from endocarditic valves standard thrombolyis regimes should be avoided.8 Although thrombolyis was performed, no acute complications were encountered as related to it.

Another case report showed successful outcome from thrombolyis in the setting of acute myocardial infarction with infective endocarditis. The case highlighted the current lack of definitive data on the optimal acute management of such an unusual clinical scenario. Although there is serious concern that thrombolytic treatment for myocardial infarction in the setting of infective endocarditis may be associated with higher risk of cerebral haemorrhage, there is little documented evidence supporting the safety of primary percutaneous coronary intervention with these patients.8

This case report is exceptional because acute myocardial infarction is a rare presentation of infective endocarditis and also as it raises concerns regarding lack of data on proper management approach in such cases. We suggest that there is a need to develop some strategy to treat patients who present with endocarditis and acute coronary syndrome.

**REFERENCES**