

# Case report: third-degree atrioventricular block secondary to septic coronary artery embolism following infective endocarditis

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## Background

Coronary artery emboli can occur from a number of rare causes such as arterial thrombo-embolus or septic embolus. This diagnosis generally requires multi-modal imaging including echocardiography, computed tomography, or invasive coronary angiography. Septic coronary emboli is an extremely rare consequence of infective endocarditis (IE), having been reported in <1% of all cases.

## Case summary

A 54-year-old previously healthy Tibetan monk presented feeling generally unwell and lethargic. Electrocardiogram demonstrated sinus rhythm, third-degree atrioventricular block with a left bundle branch escape. Initial transthoracic and transoesophageal echocardiography demonstrated vegetations on the aortic and tricuspid valve as well as intramyocardial abscess. Coronary angiography revealed septic embolus involving the septal perforator coronary artery. He underwent surgical replacement of the infected valves and debridement and repair of a ventricular septal defect.

## Discussion

Infective endocarditis can predispose to a range of cardiac pathology. This case demonstrates that patients can present with cardiac conduction disease from a septic embolus involving a coronary artery as a complication of IE.

## Keywords

Case report • Conduction disease • Infective endocarditis • Septic embolism • Acute coronary syndrome

## Learning points

- Septic embolism is a rare complication of infective endocarditis (IE) and can present with rhythm disturbances.
- Transoesophageal echocardiography can be a valuable tool in the diagnosis of IE.

## Introduction

Coronary artery emboli account for <3% of all presentations of acute coronary syndrome.<sup>1</sup> While usually attributed to arterial thrombo-embolic disease, emboli can also occur secondary to infective endocarditis (IE).<sup>1,2</sup> Direct embolization from a vegetation to a coronary

artery remains an extremely rare complication of IE which has been reported in <1% of cases.<sup>1</sup> This report details the case of a patient with IE who presented with cardiac conduction disease and subsequently developed an intra-myocardial abscess secondary to a septic coronary artery embolism.

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## Timeline

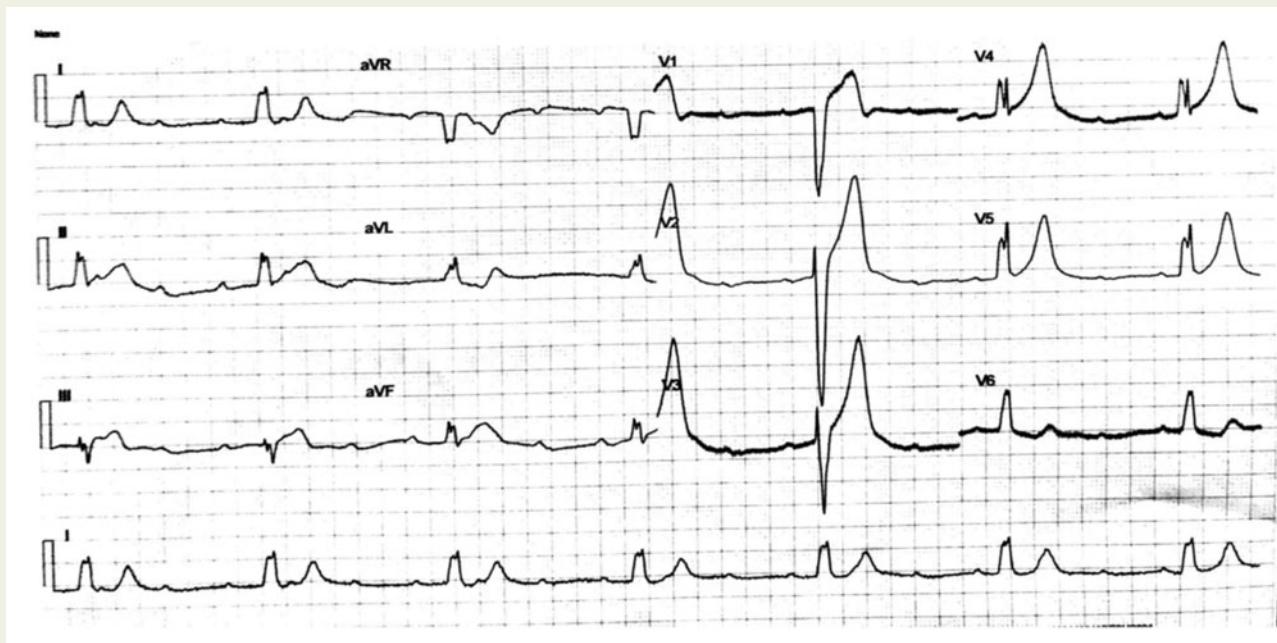
Admission to hospital	Presented with 2 weeks of feeling generally unwell with lethargy. He was febrile on admission with raised inflammatory markers. Electrocardiogram demonstrated sinus rhythm, third-degree atrioventricular block with a left bundle branch block escape. Commenced on empiric intravenous antibiotics
3 days post-admission	Blood cultures return with growth of Group B <i>Streptococcus</i> . Initial transoesophageal echocardiogram reveals aortic and tricuspid valve vegetations indicative of infective endocarditis
4 days post-admission	Coronary angiogram done via right radial artery shows septal coronary embolus involving the first septal perforator artery
13 days post-admission	Repeat transoesophageal echocardiogram shows progression in intra-cardiac infection despite intravenous antibiotic therapy with worsening aortic and tricuspid valvular regurgitation and intra-myocardial abscess formation
16 days post-admission	Underwent aortic valve replacement, tricuspid valve replacement, debridement of abscess surrounding interventricular septum, and patch repair of ventricular septal defect

## Case presentation

A 54-year-old Tibetan monk presented to the Emergency Department with a 2-week history of feeling generally unwell and lethargic. He had associated night sweats with fevers and mild breathlessness for 10 days preceding his hospital presentation and had been commenced on amoxicillin 875 mg/clavulanic acid 125 mg daily, azithromycin 250 mg daily, and prednisone 50 mg daily for 2 days by his local doctor for an atypical lower respiratory tract infection. He did not have any background medical history, did not use intravenous drugs, and did not take any regular medications.

Physical examination revealed a blood pressure of 140/60 mmHg with a heart rate between 45 and 50 b.p.m. Lung auscultation revealed occasional crepitations at both bases with no wheeze. The maximal recorded temperature on admission was 37.9°C. Jugular venous pressure was noted to be elevated to 4 cm with prominent 'v' waves. There were no peripheral stigmata of IE, no audible murmurs, and no peripheral oedema. Dentition was intact with no dental caries. Electrocardiogram demonstrated sinus rhythm, third-degree atrioventricular (AV) block with a left bundle branch escape (QRS 142 ms) (Figure 1).

His white cell count was elevated at  $16.5 \times 10^9/L$  (normal 3.5–11.00  $\times 10^9/L$ ), high sensitivity troponin T was elevated at 2174 ng/L (normal < 14 ng/L), and the C-reactive protein was elevated at 256 mg/L (normal < 3 mg/L). Initial transthoracic echocardiogram (TTE) revealed basal inferior, inferoseptal, and anteroseptal akinesis consistent with segmental left ventricular dysfunction as well as a small echodensity on the ventricular side of the aortic valve with mild central aortic regurgitation. There was no ventricular septal defect seen on the TTE done on admission. Transoesophageal echocardiogram confirmed endocarditis with a 10 mm  $\times$  3 mm mobile echodensity on the ventricular side of the aortic valve (Figure 2) with no evidence of aortic



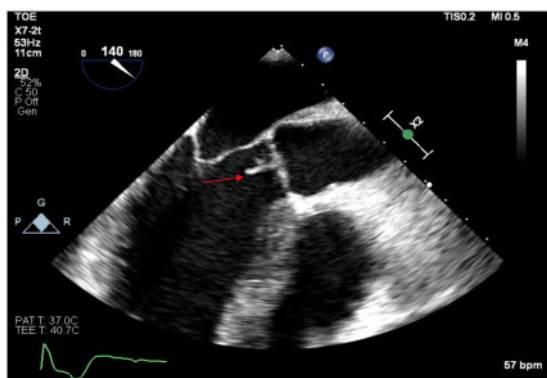
**Figure 1** Electrocardiogram: sinus rhythm with third-degree atrioventricular block with left bundle branch block escape.

annular abscess formation. There was also an independent mobile echodensity (10 mm × 7 mm) attached to the posterior leaflet of the tricuspid valve with leaflet perforation and associated severe eccentric tricuspid regurgitation. Transoesophageal echocardiogram also identified a small patent foramen ovale using colour Doppler.

The patient was commenced on empiric intravenous benzylpenicillin 1800 mg four times a day and vancomycin 1000 mg daily for IE. Blood cultures subsequently grew penicillin-sensitive *Streptococcus agalactiae* (Group B *Streptococcus*) and so vancomycin was ceased. He was reviewed by the cardiothoracic surgical team and was planned for surgical management of his IE. Intravenous antibiotic therapy was continued for 16 days from admission until surgery.

The patient underwent invasive coronary angiography to investigate the suspected myocardial infarct as suggested by the raised troponin and TTE wall motion abnormalities. Computed tomography coronary angiography had been considered but was not performed as a coronary angiogram was additionally required as part of the preparation for bypass surgery. A left ventriculogram and left ventricular pressures were not obtained due to the risk of embolism from crossing the aortic valve. The dominant right coronary artery was entirely normal in its course. The left circumflex artery was normal in its entire course. The left anterior descending artery had sequential 20% stenoses at the proximal, mid, and distal segments, respectively. There was a 100% occlusion at the origin of the first septal perforator artery which had the angiographic appearance of an acute embolus (Figures 3 and 4). He remained in third-degree AV block with left bundle branch block during the procedure and did not require temporary pacing.

Repeat TTE demonstrated progression of intra-cardiac infection with myocardial abscess formation (Figure 5) and the patient underwent emergent replacement of the aortic valve with a 21 mm Perimount Magna Ease bioprosthesis. The ventricular septal myocardial abscess was repaired with a bovine patch. The tricuspid valve was replaced with a 33 mm Magna Mitral Ease bioprosthesis. The patient remained dependent on epicardial pacing wires post-operatively. He passed away 7 days later from complications following breakdown of the septal defect patch repair and formation of a large ventricular septal defect that could not be repaired.

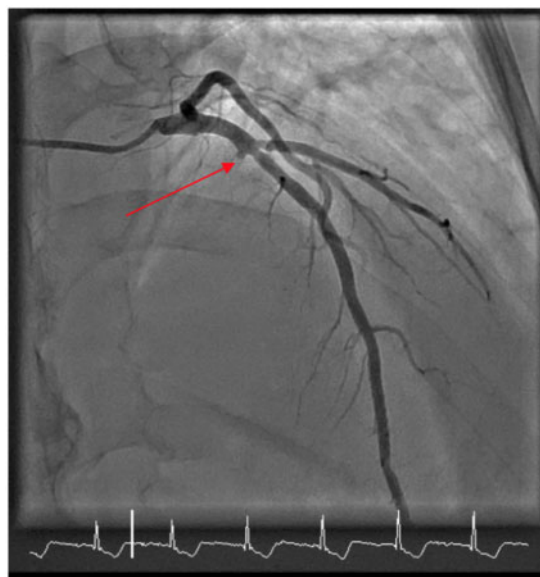


**Figure 2** Mid-oesophageal three-chamber transoesophageal echocardiogram image showing 10 mm × 3 mm vegetation on ventricular aspect of aortic valve.

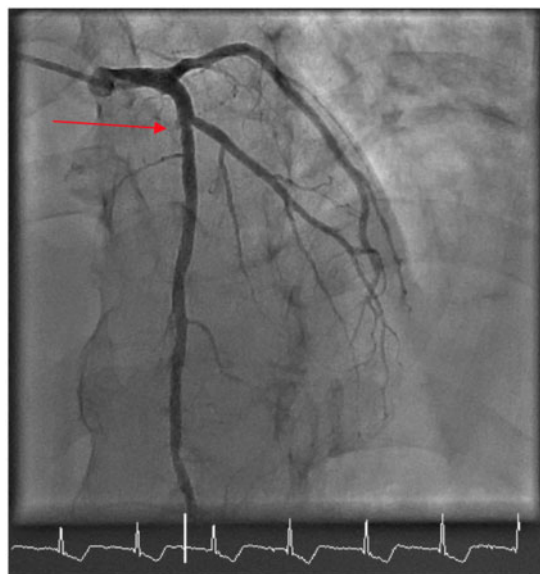
## Discussion

Cardiac complications have been reported to occur in up to 50% of all patients with IE.<sup>1</sup> Congestive cardiac failure from valvular dysfunction, perivalvular abscess formation, and subsequent conduction disorders of the AV node are the most commonly encountered complications.<sup>3</sup> Septic coronary emboli have been reported in <1.0% of all cases of IE and often carry a guarded prognosis due to high rates of congestive cardiac failure (73%) and death (64%).<sup>2</sup>

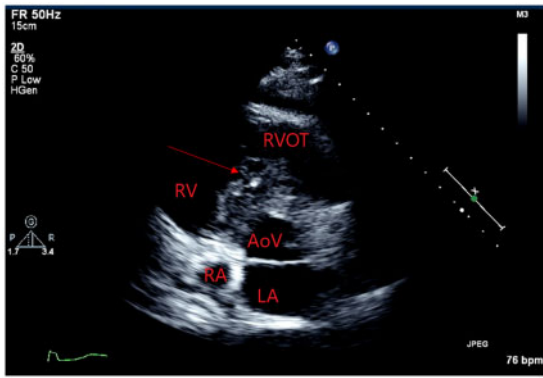
Presentation with septic coronary emboli typically occurs within 2 weeks of endocarditis in a similar time span to how our patient pre-



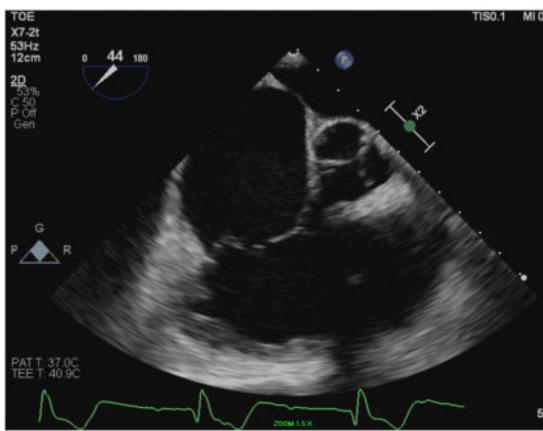
**Figure 3** Cranial projection of LAD showing septic embolus involving first septal perforator artery.



**Figure 4** LAO Cranial projection of LAD showing septic embolus involving first septal perforator artery.



**Figure 5** Right ventricle inflow–outflow transthoracic echocardiogram image demonstrating myocardial abscess with the involvement of base of ventricular septum and extending into right ventricle. AoV, aortic valve; LA, left atrium; RA, right atrium; RV, right ventricle; RVOT, right ventricular outflow tract.



**Figure 6** Mid-oesophageal short-axis transoesophageal echocardiogram image of aortic valve and aortic annulus.

sented.<sup>3</sup> Factors that predispose to coronary emboli following endocarditis include the size of the vegetation with lesions >1 cm conferring the greatest risk.<sup>2</sup> In our case, both valves had quite large vegetations which increase the risk of embolization. This case remains unique in that the complete occlusion of the first septal perforator artery and subsequent ventricular septal abscess formation with likely involvement of the Bundle of His was the cause for the patient's AV block and left bundle escape rhythm. We hypothesize that the mechanism for this is analogous to the AV block observed as a complication in 10–15% of patients following alcohol septal ablation for hypertrophic obstructive cardiomyopathy.

The American College of Cardiology guidelines suggest that valve replacement is indicated in patients with recurrent embolic phenomenon or heart failure which is the trajectory that our case took. Thrombolytic therapy is one potential strategy of treating embolic phenomenon that occur to the coronary arteries.<sup>2,4</sup> However, it has been shown to carry significant bleeding risk driven largely by the increased risk of intracerebral haemorrhage from mycotic aneurysms and cerebral infarcts which are not uncommon complications of IE.<sup>5</sup>

Percutaneous balloon angioplasty with or without stent insertion is another strategy of managing these lesions. However, it also has varied success due to the lack of atherosclerotic plaque available to expand the vessel lumen which is a risk factor for stent thrombosis and re-occlusion.<sup>6</sup> Percutaneous coronary intervention (PCI) itself carries the risk of stent infection and the risk of rupturing mycotic aneurysms in the coronary vasculature which has a high mortality.<sup>2,4</sup> Mechanical aspiration thrombectomy has been shown to be effective in improving myocardial perfusion and improve coronary flow by reducing microvascular obstruction in randomized clinical trials for acute myocardial infarction (AMI).<sup>7</sup> There is some evidence supporting the use of aspiration thrombectomy with or without PCI as a safer approach in cases of AMI associated with septic coronary emboli.<sup>7,8</sup>

## Conclusion

We have reported the case of *Streptococcus* endocarditis presenting with third-degree AV block secondary to a septic embolus involving the septal perforator coronary artery. To date, there are no specific guidelines on the safest and most effective approach to manage these patients due to the rarity of the condition.

## Lead author biography



Abhishek Prashar is currently in his final year of advanced training in Cardiology at St George Hospital in Sydney, Australia and hopes to continue his commitment to research and clinical excellence.

## Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

**Slide sets:** A fully edited slide set detailing this case and suitable for local presentation is available online as [Supplementary data](#).

**Consent:** The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

**Conflict of interest:** none declared.

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