Multi-embolic ST-elevation Myocardial Infarction Secondary to Aortic Valve Endocarditis

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We present the case of a 42 year-old woman admitted to hospital with ST-elevation myocardial infarction involving two separate coronary territories. Angiography revealed multi-embolic occlusions of her left anterior descending (LAD) and first obtuse marginal (OM1) coronary arteries. Transoesophageal echocardiogram (TOE) showed a lesion attached to the left cusp of the aortic valve and she was treated for infective endocarditis. We discuss the management issues raised from this unique patient, including reperfusion strategies in endocarditis-associated myocardial infarction.

Keywords
Cardiology • Myocardial infarction • Percutaneous coronary intervention • Thrombectomy • Bacterial endocarditis

Clinical Case

Ambulance paramedics were called to a 42 year-old woman with severe central chest pain. Heart rhythm briefly showed ventricular tachycardia, before she became unresponsive in ventricular fibrillation. She reverted to sinus tachycardia with one 150J biphasic defibrillation however she was intubated due to persistent, uncontrollable agitation.

12-lead electrocardiogram (ECG) suggested an inferolateral ST-elevation myocardial infarction (STEMI). She was transferred to the catheterisation laboratory where angiography demonstrated two occlusions – at the apical part of the left anterior descending (LAD) coronary artery and the first obtuse marginal (OM1) branch of a co-dominant left circumflex artery (LCx), with otherwise normal coronary arteries (Figure 1). Aspiration thrombectomy was performed on the LAD and OM1 occlusions, but neither occlusion was suitable for stenting. Ticagrelor 180 mg and abciximab (12.5 mg intra-coronary loading) were administered before transfer to the intensive care unit (ICU) for inotropic and ventilatory support.

On admission to ICU the patient was afebrile. Salient examination features included a right thumbnail splinter haemorrhage, clubbed fingers and bi-basal lung crepitations. No heart murmur was audible. At presentation, white blood cell count was 23.4 x 10^9/L (4.5-13) with neutrophilia, 20.4 x 10^9/L (1.5-8), C-reactive protein was 18 mg/L (<10 mg/L), peaking two days later at 200 mg/L. Troponin I was 9.84 µg/L (<0.03 µg/L), increasing to 59.8 µg/L. Creatine kinase peaked at 2430 µmol/L. Serum electrolytes, urea, creatinine, liver enzymes and clotting times were normal. Chest radiograph confirmed mild pulmonary oedema, treated with intravenous (IV) frusemide 40 mg daily. Hospital records revealed a past history of asthma, depression, gastro-oesophageal reflux disease and IV drug use.

Transoesophageal echocardiogram (TOE) showed an oscillating 3 mm spherical lesion attached to the left cusp of the aortic valve, close to the left main coronary artery entrance (Figure 2), with trivial aortic regurgitation and moderate, segmental left ventricular (LV) dysfunction. There was no aortic root abscess, cusp perforation or other vegetations, and
no patent foramen ovale. The TOE findings led to a working diagnosis of infective endocarditis (IE) and empiric IV antibiotic therapy was commenced. Repeat blood cultures, including three sets before antibiotic administration, remained negative, as did serology of organisms commonly causing culture-negative endocarditis. There were no cerebral emboli seen on computed tomography of the brain.

The patient was transferred out of ICU on day 9 and continued to make a good clinical recovery. A peripherally inserted central catheter (PICC) line was placed for long-term IV antibiotic administration. Repeat transthoracic echocardiogram (TTE) showed improved systolic function, with mild-moderate segmental LV dysfunction and a thin, akinetic apex. Bisoprolol 5 mg daily and ramipril 1.25 mg daily were continued for the cardiomyopathy with low-dose aspirin and frusemide. Repeat TOE at two months demonstrated normalised LV systolic function with no evidence of the previously seen vegetation. All medications except aspirin were ceased, with a further TTE and cardiology review planned for six months time.

Discussion

Acute myocardial infarction (MI) is an uncommon complication of IE [1]. Mechanisms of MI in this setting include extrinsic compression of the ostium from aortic valve vegetations, periannular complications including abscesses and mycotic aneurysms, severe aortic regurgitation, and rarely coronary artery embolisation [2,3]. First reported by Virchow and initially only found with vegetation fragments at autopsy [4], there have since been several case reports of coronary embolism secondary to IE [2–13]. This patient is a unique case with ST-elevation myocardial infarction, dual coronary emboli and subsequent clear angiographic and echocardiographic evidence of the diagnosis. A similar case in a patient with nonbacterial thrombotic endocarditis was recently reported [14].

As reported above, angiography demonstrated the unusual appearance of two distal coronary artery occlusions with no atherosclerosis elsewhere, suggesting an embolic phenomenon. The confirmed valvular vegetation, combined with the history of IV drug use and evidence of arterial embolism led to a presumptive diagnosis of IE according to the Duke diagnostic criteria [15]. Repeat negative blood cultures and the presence of clubbing raise the possibility of a chronic vegetation in the setting of subacute culture-negative endocarditis.

Endocarditis-associated MI creates unique management difficulties, with the optimal strategy for reperfusion yet to be identified. Traditional percutaneous intervention (PCI) involving balloon angioplasty and stent placement have had varied outcomes in reported cases; from successful...
restoration of flow to failure of the procedure, fatal left main coronary artery embolism post cardiac catheterisation or the development of mycotic aneurysms [3,7–10,16]. Aspiration thrombectomy, as used for the LAD occlusion in this case, may be safer and more effective, but data from cases remains limited [3,10,16]. Abciximab was administered in this patient, and may be a viable adjunct performing a similar role to its use in complicated PCI of atherosclerotic occlusions. Percutaneous or direct incision embolectomy done concurrently with valvular surgery is another reported alternative [10,11], although these patients are often poor surgical candidates.

While no direct comparative data exists with mechanical reperfusion, thrombolysis has been associated with negative outcomes when used in the presence of IE, with high rates of intracranial haemorrhagic events [3,5,12,13]. In geographical areas where primary PCI is not readily available, following standard revascularisation pathways may therefore increase morbidity and mortality in patients with IE.

The diagnosis of IE in a patient presenting initially with an acute coronary syndrome is extremely difficult. However, it should be considered when there are predisposing factors for or a history of IE, or in a young patient with minimal traditional cardiovascular risk factors. The best management of endocarditis-associated MI remains uncertain, with several cautionary lessons established from the literature. Aspiration thrombectomy may have a significant role, but the current choice of intervention remains heavily influenced by individual circumstances and accessibility to services. Further research is needed to assist decision-making in such complex cases.

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References