Novel treatment (new drug/intervention; established drug/procedure in new situation)

Successful treatment of ST elevation myocardial infarction caused by septic embolus with the use of a thrombectomy catheter in infective endocarditis

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Summary

A 71-year-old patient suffered a transmural (ST elevation) myocardial infarction (MI) as a result of a septic embolus from an infected tissue aortic valve replacement. Following failed fibrinolysis, his MI was successfully treated with thrombectomy using an export catheter. He suffered bleeding complications following the administration of tenecteplase and required aortic valve and root replacement due to ongoing systemic embolisation.

BACKGROUND

Systemic embolisation is a common complication in the course of infective endocarditis occurring in up to 50% of cases. The major arterial beds are all at risk from systemic embolisation, with the most common site being the cerebral circulation where up to 65% of emboli lodge.¹ ST elevation myocardial infarction (MI) remains a rare complication of infective endocarditis² for which the optimal treatment has yet to be defined.

CASE PRESENTATION

A 71-year-old man was admitted to a local hospital with a 2-day history of rigors and headache. He had undergone tissue aortic valve replacement for severe aortic regurgitation 18 months previously and balloon angioplasty for coronary artery disease 15 years ago. He also described 6 weeks of malaise, lethargy and loss of appetite. On examination, he was febrile (38.1°C) and tachycardic. Examination revealed a flow murmur across his aortic valve. Respiratory, abdominal and neurological examination on admission were normal. The chest x-ray was unremarkable and urine microscopy and culture were negative. C reactive protein (CRP) was 204 IU (normal range < 5) and the white cell count was 22.8×10^9 cells/l (20.7×10^9 neutrophils/l). Blood was drawn for culture and empirical intravenous antibiotic (piperacillin and tazobactam) treatment commenced, although the source of sepsis was unclear. Blood cultures subsequently yielded *Enterococcus faecalis*, which was sensitive to penicillin and gentamicin. His fever and tachycardia settled initially with this management; however, 36 h into the admission he developed central crushing chest pain. This was accompanied by ST segment elevation in leads V1–V4 of the 12-lead ECG, with ST depression in the leads III and aVF (figure 1A) and a prolonged PR interval of 312 ms (normal range 120–200 ms). A diagnosis of acute anterior MI was made and he

was treated with fibrinolysis (tenecteplase). However, the chest pain and ST segments had failed to settle and he was transferred to St Thomas' Hospital in London for rescue percutaneous coronary intervention.

INVESTIGATIONS

Coronary angiography revealed midcourse obstruction of the left anterior descending artery (LAD) (figure 1C) with angiographically minor plaque disease elsewhere. White thrombus was aspirated from the LAD with an export catheter (figure 2A) and TIMI 3 flow restored (figure 1D), without evidence of a significant underlying atherosclerotic lesion. In view of his history and the absence of obstructive coronary disease, we considered the possibility of prosthetic aortic valve endocarditis complicated by embolisation of a vegetation down the LAD. As such we did not carry out further treatment of the vessel by balloon inflation or stent deployment. Following thrombus aspiration the patient's pain improved and the ST segments returned to baseline (figure 1B). Transthoracic echocardiography, the following day revealed evidence of a large apical infarct and overall mild systolic impairment (ejection fraction 40–45%). The tissue aortic valve replacement was well seated with good leaflet mobility and no obvious abscess formation. There was evidence of mild aortic regurgitation through the prosthesis but no vegetations were demonstrated and no left ventricular thrombus.

The patient suffered a gastrointestinal bleed the following morning and after endoscopic treatment the gastroenterology team advised delaying further intubation of the oesophagus for transoesophageal echocardiogram (TOE).

On day 2, the patient described loss of balance, a sensation of dizziness and double vision. Neurological examination revealed a lateral gaze palsy and MRI of the brain revealed multiple foci of acute infarction within the

Figure 1 (A) Initial ST elevation ECG showing ST segment elevation in leads V1–V4 of the 12-lead ECG, with ST depression in the leads III and aVF and a prolonged PR interval of 312 ms (normal range 120–200 ms). (B) Post angiography ECG. (C) Occluded LAD on angiography. (D) Restoration of TIMI 3 flow with smooth unobstructed artery

cerebellum, left thalamus and both cerebral hemispheres. The central nervous system infarcts were in several arterial territories suggesting a likely cardiac source for the emboli. The MRI scan demonstrated that these infarcts had undergone haemorrhagic transformation, most likely the result of the administration of thrombolytic therapy. MRI of the spine and ultrasound imaging of the renal tract did not reveal any evidence of emboli or abscesses. Apart from haemorrhagic transformation of the intracerebral infarcts and gastrointestinal bleed, the patient experienced frank haematuria in the days following thrombolysis.

The material from the LAD (figure 2A) was sent for microscopy and culture. Although neither pus cells nor organisms were seen on initial examination, when incubated it grew *Enterococcus*. Histological analysis of the embolus revealed fibrin, acute inflammatory cells and bacterial colonies (figure 2B). Gram stain of the embolus demonstrated Gram-positive cocci within fibrin (figure 2C). TOE demonstrated a well-seated biological valve replacement with no vegetations. An echo-free space was identified between the left atrium and aorta which was thought most likely to represent a postsurgical pouch or an abscess cavity.

TREATMENT

Our patient's antibiotic treatment was changed to amoxicillin and gentamicin. He continued to improve clinically and his CRP and white cell count improved. His dizziness and co-ordination responded well to neurophysiotherapy. Seven weeks following his initial presentation, the patient again developed vertigo and clinical signs of cerebellar dysfunction. Repeat MRI brain demonstrated a punctate

focus of restricted diffusion in the right postcentral gyrus that was suggestive of an acute infarct. The patient was reviewed by the cardiothoracic surgical team who agreed that in the context of ongoing septic embolisation despite appropriate antibiotic therapy surgical intervention was indicated. He was taken to theatre for an aortic valve replacement. At operation, a partly healed abscess cavity was visualised and there was residual vegetation on the tissue aortic valve. The aortic valve was completely excised and the aortic root was replaced with a Medtronic freestyle prosthetic root.

OUTCOME AND FOLLOW-UP

Although the patient recovered well following surgery, he did require a permanent pacemaker for second degree heart block. He completed a further week of antibiotics following surgery and underwent further neurorehabilitation before being discharged with cognition and function back to his admission baseline.

DISCUSSION

There has been considerable interest in the ability of echocardiography to predict whether the patients are at high risk of embolisation. Results have been varied with some studies suggesting higher rates of embolisation with particular vegetation characteristics (size, mobility and location of vegetations)^{3–5} and others demonstrating no discriminatory ability of echocardiography.⁶ The risk of embolisation is highest during the first days following initiation of antibiotic therapy (as in our patient)⁴ and then declines significantly following 2–3 weeks of successful therapy.¹

Figure 2 (A) Aspirated embolus. (B) Histological analysis of embolus showing fibrin, neutrophils and red blood cells. H&E, $\times 200$. (C) Gram stain of embolus demonstrating purple coloured Gram-positive bacterial colonies within fibrin. Gram stain, $\times 600$.

Myocardial ischaemia is recognised to occur in the setting of infective endocarditis and the clinical features are indistinguishable from myocardial ischaemia in any other setting.⁷ In one study, 2.9% of patients being treated for infective endocarditis developed an acute coronary syndrome in the course of their illness. Myocardial ischaemia was the result of extrinsic compression of the coronary arteries (due to large periannular aortic complications) or septic embolisation.⁷ Atherosclerotic plaque rupture is another cause of myocardial ischaemia that must be considered with an increased risk in situ thrombus formation due to a hypercoagulable state on account of ongoing inflammation.² ST elevation MI remains a rare complication of infective endocarditis.² Reported complications of septic embolisation causing MI include left ventricular free wall rupture,⁸ mycotic aneurysm⁹ and death.¹⁰

The appropriate treatment of embolic transmural (ST elevation) MI secondary to infective endocarditis has not been established. As the incidence is rare, controlled studies are unlikely. The use of thrombolytic agents carries a risk of intracerebral haemorrhage when used with standard indications.¹¹ This risk is likely to be significantly increased in patients with infective endocarditis who are at high risk of having had cerebral emboli, as occurred in our patient. Fatal cerebral haemorrhage has been reported in this context,¹² and the overall experience has been unfavourable.² Percutaneous intervention offers an alternative strategy and successful treatment of ST elevation MI has been reported with balloon angioplasty alone,¹³ intracoronary

stent placement¹⁴ and thrombectomy followed by intracoronary stenting.¹⁵ The benefit of catheter thrombus aspiration prior to percutaneous coronary intervention in the setting of ST elevation MI has been established¹⁶ and is likely to have an important role in MI secondary to septic embolisation. Mycotic aneurysm has been reported following angioplasty in this setting,^{17–19} and some authors advocate follow-up angiography to screen for this complication if angioplasty is performed.⁷ It has been suggested that the deployment of intracoronary stents in this setting may increase the probability of developing a mycotic aneurysm.¹³ The decision regarding deployment of a stent device must be based on an analysis of the risk of placing foreign material in the context of a bacteraemia with the potential risk of subsequent mycotic aneurysm formation as well as the requirement for antiplatelet therapy following their use. Having avoided the placement of an intracoronary stent, our patient was able to continue single antiplatelet therapy only.

Valve surgery remains a key consideration in the management of patients with infective endocarditis in the acute and convalescent phase. Recurrent systemic embolisation, despite appropriate antibiotic therapy is an indication for surgical intervention²⁰ as occurred in our patient. Surgical intervention was not indicated in our patient until it was established that he was experiencing ongoing systemic embolisation despite appropriate antibiotics. At this stage aortic valve and root replacement was the appropriate therapy.

Learning points

- ▶ It is important to maintain a high index of clinical suspicion for infective endocarditis in patients presenting with unexplained sepsis, particularly in the context of previous valve surgery.
- ▶ Remember to consider alternative causes of myocardial ischaemia to atherosclerotic plaque rupture in patients in whom infective endocarditis is a possible diagnosis.
- ▶ Defining the aetiology of myocardial ischaemia is crucial in selecting the optimal reperfusion strategy for patients at high risk from bleeding complications in the cerebral circulation and beyond.
- ▶ In cases of definite infective endocarditis, thrombolysis is contraindicated and early coronary angiography with thrombectomy may be a successful reperfusion strategy.

Competing interests None.

Patient consent Obtained.

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