

CASE REPORT

A subarachnoid haemorrhage heralding bacterial endocarditis

A. Klijn¹, L. Valk², J.A.H. van Oers¹Department of ¹Intensive Care Medicine and ²Cardiology, Elisabeth Tweesteden Hospital, Tilburg, the Netherlands

Correspondence

A. Klijn - adine.klijn@gmail.com

Keywords - endocarditis, subarachnoid haemorrhage, mycotic aneurysm**Abstract**

We present the case of subarachnoid haemorrhage caused by a ruptured infectious intracranial aneurysm secondary to endocarditis in a young man. He presented with signs of a neurological event, which turned out to be a subarachnoid haemorrhage. Symptoms of fever, chills and a systolic cardiac murmur on auscultation were the reasons to obtain blood cultures and initiate further diagnostic workup for endocarditis. This confirmed the suspicion of endocarditis of the mitral valve caused by *Streptococcus mitis*.

Introduction

Neurological symptoms complicating endocarditis are frequently reported: in about 25% of all cases.^[1] Cerebral ischaemia, encephalopathy and meningitis have the highest reported incidence. Cerebral haemorrhage heralding endocarditis as an early symptom is rare.^[1,2] Only 0.5% of all patients with infectious endocarditis will develop cerebral haemorrhage caused by ruptured infectious or 'mycotic' aneurysms.^[1]

Case presentation

A 24-year-old man presented to the emergency department with left-sided hemiparesis, aphasia and altered consciousness. For several weeks he had symptoms of general malaise, weight loss, and fever and chills. The patient had no relevant medical history, nor a history of intravenous drug abuse. Recently, he had visited the dentist. A computed tomography (CT) of the cerebrum showed a large intraparenchymal haemorrhage in the right hemisphere with a 7 mm midline shift, a smaller haemorrhage in the left hemisphere and blood in the subarachnoid space (figure 1). The patient was transferred to our centre for neurosurgical diagnosis and intervention. On admission to our hospital his blood pressure was 110/60 mmHg and heart rate 80 beats/min. His body temperature was 37.9 °C. Mouth and throat revealed no abnormalities. His lungs were clear on auscultation, but a grade IV/VI systolic murmur



Figure 1. Subarachnoid and intracerebral haemorrhage of the right hemisphere

with maximum intensity at the apex was heard. Further examination of the skin and nails was without abnormalities. Neurological examination showed a Glasgow Coma Scale of E3M6V3 with hemiparesis of the left side. Laboratory results showed a haemoglobin of 5.2 mmol/l (8.5-11.0 mmol/l), mean corpuscular volume of 79 fl and a C-reactive protein of 65 mg/l (0-10 mg/l); other laboratory studies were within the normal range. Blood cultures turned out to be positive for *Streptococcus mitis*. The patient was initially treated by high-dose ceftriaxone which was converted to benzylpenicillin when the antibiogram was known. Transoesophageal echocardiography confirmed the suspicion of endocarditis of the native mitral valve. There was

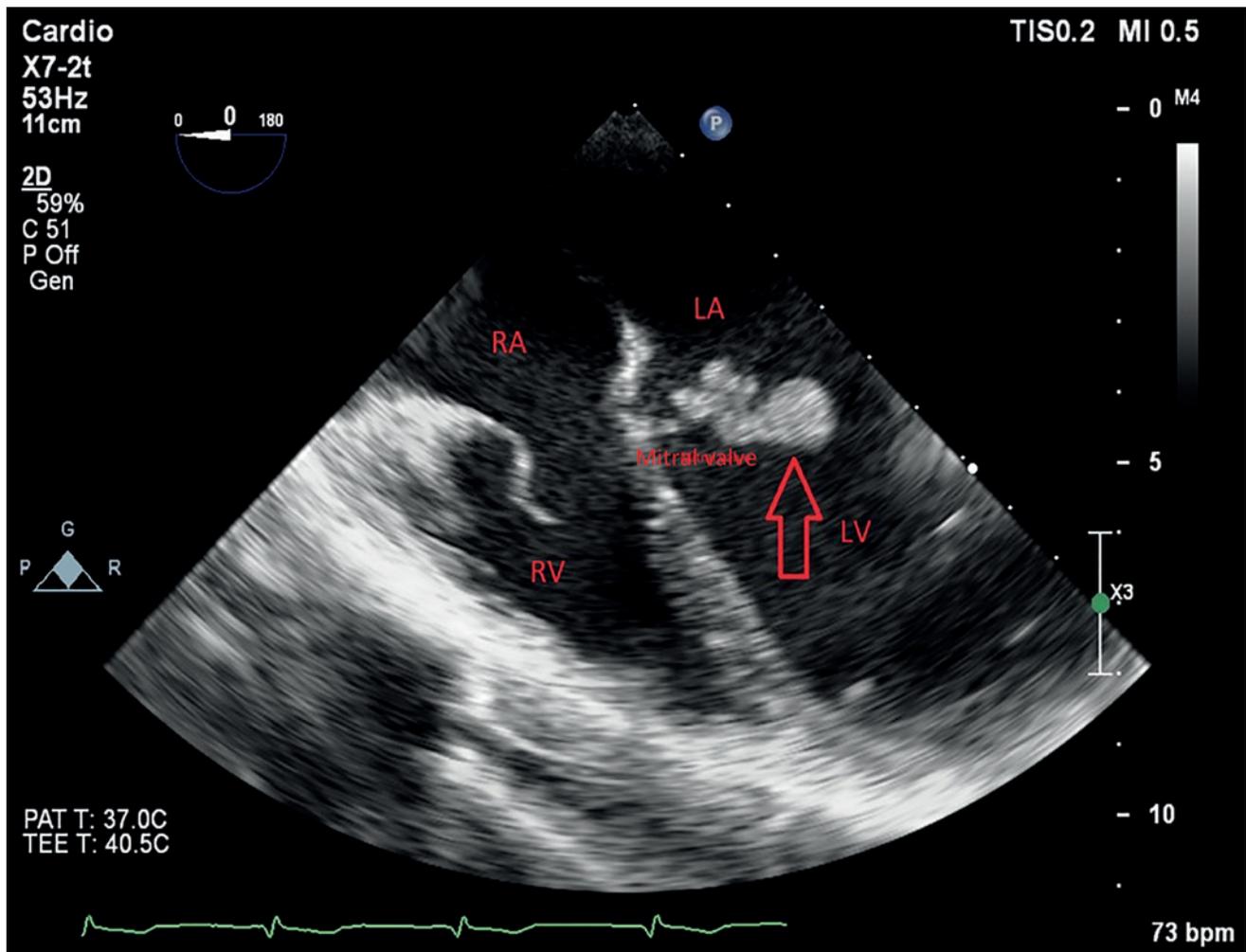


Figure 2. Transoesophageal echocardiography

severe mitral valve regurgitation and a large mobile vegetation 16mm in size at the atrial side, covering the valve both on the anterior and on the posterior leaflet (figure 2). Cerebral angiography (figure 3) revealed a 11 mm aneurysm in the distal right middle cerebral artery (figure 3a) and a smaller one in the distal left posterior cerebral artery (figure 3b). The next day the aneurysm at the right hemisphere was obliterated by selective coiling and the smaller aneurysm at the left hemisphere was glued. CT of the thorax and abdomen showed an aneurysm in the distal superior mesenteric artery and signs of bilateral renal infarction with preserved renal function. His neurological state did not show any improvement. After three days patient was transferred to a university hospital for cardiac surgery. A mitral bioprosthesis was implanted. Three days after surgery the patient was discharged from the ICU to the CCU and after that to surgical ward and one month after his first admission in our hospital and one month after his first admission to our hospital he was discharged to a rehabilitation clinic. His neurological state further improved to nearly his previous condition and continues to improve.

Discussion

Neurological events as a complication of endocarditis are seen in about a quarter to one third of patients.^[1,2] The majority are ischaemic events (56%) due to septic embolisms. Mycotic aneurysms occur in 1-10% of patients with endocarditis.^[1,2] Only 0.5% of all patients with infectious endocarditis will develop subarachnoid or intracerebral bleeding due to ruptured mycotic aneurysms.^[1] Endocarditis is an infection of the endothelium of the heart. Its incidence is about 3-10 per 100,000 people;^[3] 25-30% of the cases of endocarditis are healthcare-associated infections, due to for example intravascular catheters and haemodialysis. The remaining cases are community-acquired infections often caused by bacteria entering the circulation via the oral cavity or the skin.^[3,4] By far the most common micro-organisms associated with infective endocarditis of native valves are Gram-positive cocci, such as *Staphylococcus*, *Streptococcus* and *Enterococcus* species.^[3,5] The term mycotic aneurysm was first described by William Osler in 1885 as a consequence of fungal vegetations.^[6] However, nowadays it is used as a more general description for all aneurysms caused by infectious embolisms

originating from the heart. Mycotic aneurysms or intracranial infectious aneurysms are quite uncommon phenomena. Of all intracranial aneurysms, 0.7-5.4% are mycotic aneurysms.^[7] These are typically seen in patients with endocarditis and less often in patients with intracranial infections such as meningitis or post-surgery infections.^[8] Neurological events due to infectious endocarditis are associated with poorer outcomes; mortality risk is about 45%, which is nearly twofold higher than in patients without neurological events.^[1] Mycotic aneurysms can occur in any cerebral vessel, but more often in the distal branches of the middle cerebral artery.^[9,10] Mycotic aneurysms are caused by either primarily microembolisms from the vaso vasorum or secondary as embolisms as a result of fragmentation of cardiac septic embolisms. Embolisms cause an inflammatory reaction which destroys the adventitia. It spreads through the tunica media where it causes a rupture between the internal elastic layer and endothelium of the vessel. This leads to weakness of the vessel wall and aneurysm formation.^[11] Besides the systemic symptoms of endocarditis (such as general malaise and fever) patients with a ruptured aneurysm may have headache due to high intracranial pressure, confusion, seizures or paresis.^[11,12]

The administration of antibiotics to avoid neurological complications is important. The 2019 Dutch Working Party on Antibiotic Policy (SWAB) guidelines for the antimicrobial treatment of infective endocarditis advise amoxicillin 12 g/day in six doses and ceftriaxone 4 g/day in two doses as empirical therapy in a patient with a native valve and subacute presentation.^[13] When *Streptococcus mitis* is cultured as the causative agent, antibiotics are switched to penicillin 12 million units/day in six doses. One observational study described an absolute reduction in neurological complications of 4% in all patients with endocarditis.^[1] However, antibiotics only is associated with significantly higher mortality than antibiotics combined with surgery (clipping) or endovascular repair (coiling). A systematic review described a significantly higher mortality in patients treated only by antibiotics (27.8%, 95% CI 24.3-31.7) compared with antibiotics and neurosurgical clipping surgery (12.0% 95% CI 8.1-16.9, $p < 0.001$) or embolisation (1.0, 95% CI 0.1-3.4, $p < 0.001$).^[14]

What was unusual in this case was the young age of the patient. While the peak incidence of subarachnoid haemorrhage is around 50 years, our patient was quite young.^[15]

Subarachnoid haemorrhage heralding endocarditis is a known, but rare complication with a high risk of poor outcome or mortality. In young patients presenting with subarachnoid haemorrhage, based on more than one aneurysm in the distal branches of the main cerebral arteries, and fever and general malaise, we should be aware of more than 'just an aneurysm' and consider endocarditis as the underlying cause.

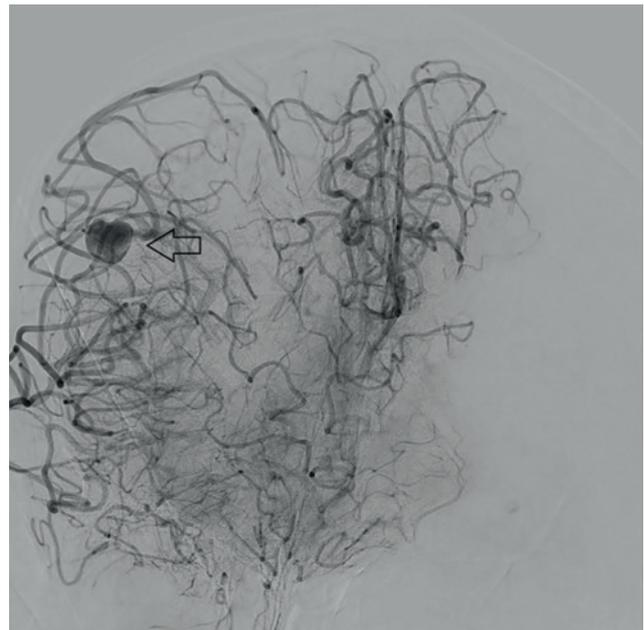


Figure 3. Cerebral angiography. 3A Aneurysm of the right middle cerebral artery; 3B Aneurysm of the left posterior cerebral artery

Disclosures

All authors declare no conflict of interest. No funding or financial support was received.

Informed consent was obtained from the patient for the publication of this case report (and the accompanying images).

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