Amnesia: a matter of the heart

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Introduction

Infective endocarditis can be difficult to diagnose, especially in the absence of typical clinical features or Duke criteria. Seeding of emboli to the cerebral cortex can give rise to neurological symptoms. In the case presented here amnesia was, unusually, the most prominent feature.

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Case report

A 56-year-old man presented to the Accident and Emergency Department with a three-day history of frontal headache, hot sweats and intermittent confusion. One week before the onset of this headache, he had experienced night sweats, which lasted for three or four days. His partner also claimed that the patient had become extremely forgetful in the preceding few days. There was no significant medical history and he was not on any regular medication. He had recently travelled to the Dominican Republic.

On examination, he was apyrexial and had no signs of meningism or focal neurological deficit. He was alert but slightly disoriented, with a mini mental test score of 8/10 (he could not recall his address or the date). Long-term memory and immediate recall were intact but short-term memory was markedly impaired. Systemic examination was unremarkable. Inflammatory markers were elevated (WBC 14.5x10^9/L, neutrophil count 11.9x10^9/L, CRP 159 mg/L); chest X-ray and urinalysis were within normal limits. A CT brain scan was uninformative and he proceeded to have a lumbar puncture. Opening pressure was elevated at 26 cm$H_2$0 but CSF microscopy was unremarkable and cultures were aseptic. A diagnosis of probable viral encephalitis was made and intravenous acyclovir was commenced.

Over the next 48 hours he developed swinging pyrexia and his short-term amnesia seemed to become worse. There were no new physical findings at this stage. The leucocytosis resolved but his inflammatory markers continued to escalate. An EEG was reported to show episodic bursts of irregular delta activity over both temporal areas. These findings were thought to be consistent with, although not typical of, viral encephalitis. The blood cultures taken on admission remained sterile.

There had been no significant improvement by day three, when re-examination revealed sinus tachycardia, a hyperdynamic apical impulse and new ejection systolic and early diastolic murmurs. A trans-thoracic echocardiogram was carried out, which revealed a prolapsing posterior aortic valve cusp with an associated echogenic mass (figure 1). There was a broad jet of aortic regurgitation into a hyperdynamic left ventricle. Three further sets of blood cultures were taken and he was commenced on intravenous benzylpenicillin, flucloxacillin and gentamicin.

The last set of blood cultures grew a *Streptococcus viridans,* which was highly sensitive to penicillin. Brain MRI showed several small focal abnormalities in the white matter of both cerebral hemispheres, with a large lesion in the right frontal lobe (figure 2). The appearances were thought to be consistent with multiple embolic lesions related to endocarditis and therefore urgent aortic valve replacement was undertaken. At surgery, six days following admission, the non-coronary cusp was found to be virtually destroyed, with a cauliflower mass extending into the left ventricular cavity and an abscess below the commissure of the non-coronary and right coronary cusps.

The aortic valve was replaced with a stentless prosthesis and he was treated with a prolonged course of intravenous antibiotics following the procedure. He made a steady recovery but still had a mild short-term amnesia on discharge.

Figure 1. Echocardiogram, showing a mass at the aortic root

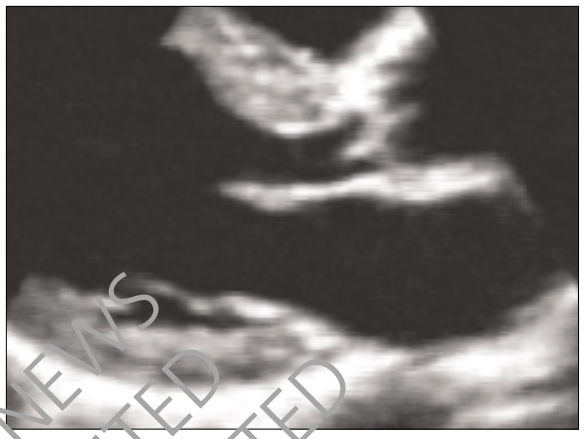

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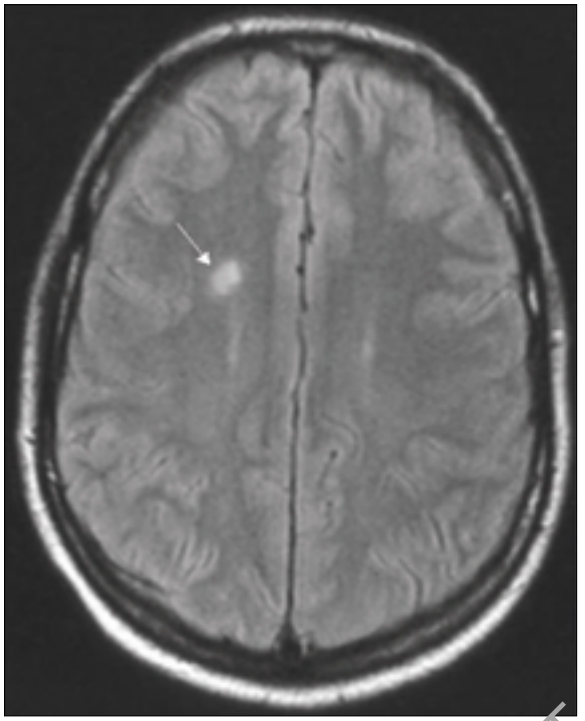
Figure 2. MRI scan, showing a large lesion in the right frontal lobe



Key messages

- Physical cardiac signs may be absent or delayed in a significant proportion of patients with infective endocarditis
- Hospital in-patients should have a thorough physical examination at least daily, particularly when the diagnosis is unclear
- Patients with a pyrexia of unknown origin should have multiple repeat blood cultures, even after commencement of antimicrobial therapy

Discussion

The manifestations of infective endocarditis (IE) are protean, and in the absence of typical clinical features or Duke criteria[1] the diagnosis is challenging and requires a high index of clinical suspicion. This case emphasises several important aspects of the presentation and management of suspected endocarditis.

Approximately one third of patients with IE develop neurological complications, most commonly in the form of a transient ischaemic attack.[2-4] However, seeding of emboli to clinically silent areas of the cerebral cortex can give rise to non-focal neurological symptoms such as headache, encephalitis or meningitis.[5] The specific cortical areas affected will determine the clinical features and patients frequently manifest psychiatric symptoms, such as confabulation, delusions and irritability. Isolated amnesia, as in this case, is unusual; there is one other report in the literature of a patient with endocarditis presenting with amnesia due to presumed temporal lobe infarction.[6] IE should be considered in the differential diagnosis of a toxic confusional state, particularly in the presence of atypical features or when the patient fails to improve as expected. Neurological complications occur more often with *S aureus* (67%) than with *Strep viridans* (22%): in one study of IE the frequency of encephalitis was 22% in *S aureus* compared to 7% in *Strep viridans* infections (meningitis 17% *vs.* 0%).[3]

The delay in diagnosing IE in our patient was primarily due to the absence of clinically evident cardiac pathology at presentation. However, it should be appreciated that cardiac signs are often delayed and that up to one third of patients with IE do not have a detectable murmur at initial evaluation.[5] The case also highlights the importance of daily systemic examination of hospital in-patients, especially when the diagnosis is unclear. If IE is suspected on clinical grounds, early echocardiography is mandatory.

The diagnostic difficulties in this patient were further compounded by the fact that the initial blood cultures were sterile. Previous studies have suggested that up to 20% of cases of IE who present with neurological symptoms are associated with initially negative cultures.[5] The commonest cause of culture-negative endocarditis is prior antibiotic treatment, but less common infectious agents such as fastidious bacteria or non-bacterial organisms should be considered.[7] Establishing the diagnosis in these patients relies on a combination of clinical judgement, application of Duke criteria and trans-oesophageal echocardiography.[8] This case also serves as a powerful reminder that investigation of patients presenting with unexplained pyrexia should include several repeated blood cultures, preferably from multiple sites.

References

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