After initial stabilization, the patient experienced repeated episodes of severe dyspnoea with clinical and radiological evidence of pulmonary congestion. During these episodes the respiratory rate increased to 30 to 40 per min, and additional i.v. furosemide (40–125 mg) was administered. In those instances there were no ECG changes and the patient denied any angina symptoms, but transient increases in CPK and CK-MB were observed (Fig. 1).

We describe a patient with mitral valve disease, who suffered from repeated episodes of severe dyspnoea and hyperventilation in the course of pulmonary congestion, followed by transient elevations of CPK and CK-MB. The increase in the plasma levels of these parameters is unlikely to have been caused by myocardial ischaemia, because the patient denied anginal symptoms, but transient increases in CPK and CK-MB were observed (Fig. 1).

Changes of creatine phosphokinase (CPK; normal range: 0–70 U.l⁻¹; □) and creatine kinase isoenzyme (CK-MB; normal range: 0–10 U.l⁻¹; ○) during hospital stay; △ marks the additional treatment with i.v. furosemide.

Streptococci strike twice

We wish to report an interesting case of infective endocarditis. A 75-year-old Ugandan Asian retired businessman was first noted to have a pansystolic murmur when he presented with a parotid abscess, which was drained with appropriate antibiotic cover. He had no history of rheumatic fever and had suffered no cardiac symptoms. An echocardiogram revealed mitral regurgitation. Eighteen months later he presented with a short history of fever, night sweats and shortness of breath: six sets of blood cultures yielded penicillin sensitive Streptococcus bovis, and he responded rapidly to intravenous benzylpenicillin and gentamicin for 10 days. Echocardiogram confirmed mitral regurgitation and thickened aortic valve cusps. Transoesophageal echocardiography was not performed.

Four months later, he represented with a 5 week history of fever, night sweats, declining exercise tolerance and orthopnoea. Splinter haemorrhages were noted. Auscultation and echocardiogram were unchanged. Three sets of blood cultures yielded Streptococcus sanguis, fully sensitive to penicillin. He received intravenous benzylpenicillin and gentamicin for 2 weeks followed by 5 weeks oral penicillin therapy. A barium enema showed scattered colonic diverticulae.

He experienced an episode of slurred speech and four episodes of facial weakness whilst on antibiotics, and these were felt to be embolic in origin. A CT scan of his brain was normal; and blood cultures were repeatedly sterile.

Six months later he underwent dental clearance for widespread periodontal disease, with co-amoxiclav and gentamicin prophylaxis. He has remained well since.

The prevalence of infective endocarditis is estimated to be between 0.3 and 3.0 per thousand hospital admissions[11]. This equals about a thousand cases per year in England and Wales[2]. The oral streptococci are the commonest cause of bacterial endocarditis, and of these Streptococcus sanguis is one of the most frequent[3]. However, a recent review confirmed the traditional association between dental disease or dental treatment and the causative organism of endocarditis in only 15% of cases[21].

Simonson et al.[4] reported a case of an intravenous drug abuser who suffered nine separate episodes of endocarditis over a 17-year period. Our patient, however, illustrates the traditional association of endocarditis and dental disease. His episode of bacterial endocarditis with Streptococcus bovis, although treated for a shorter period than is recommended[5], has shown no signs of recurrence. His case reminds us that dental examination and appropriate dental treatment is warranted after an episode of endocarditis from whatever source, to help reduce the future risk of infection to an already damaged valve.

R. PARNABY
Public Health Laboratory, Central Middlesex Hospital NHS Trust, London, U.K.

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References