Case Report

Streptococcus mutans infective endocarditis complicated by vertebral discitis following dental treatment without antibiotic prophylaxis

Sujata Biswas,¹ Ian C. J. W. Bowler,² Christopher Bunch,¹ Bernard Prendergast³ and Daniel P. Webster²

Correspondence
Daniel P. Webster
danwebster@doctors.org.uk

¹Department of Acute General Medicine, John Radcliffe Hospital, Oxford OX3 9DU, UK

²Department of Microbiology, John Radcliffe Hospital, Oxford OX3 9DU, UK

³Department of Cardiology, John Radcliffe Hospital, Oxford OX3 9DU, UK

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We report what we believe is the first reported case of *Streptococcus mutans* endocarditis complicated by vertebral discitis. The case is particularly interesting and topical as it occurred in a patient with pre-existing cardiac valvular disease who had recently had a dental procedure without antibiotic prophylaxis following a dramatic shift in the UK guidelines.

Case report

A 67-year-old man presented with a 3 week history of general malaise, night sweats, anorexia and 6 kg weight loss. Over the preceding 2 months, he had developed lower back pain of increasing severity, without lower limb weakness, paraesthesia or incontinence. His past medical history included mitral valve prolapse with associated moderate regurgitation diagnosed 3 years previously and several dental procedures, the most recent of which entailed drainage of a dental abscess 3 months prior to admission. Following the diagnosis of mitral valve disease, antibiotic prophylaxis with oral clindamycin (as the patient had previously developed a rash with oral amoxicillin) had been administered before all dental procedures. However, following a recent change in the infective endocarditis (IE) prevention guidelines in the UK, this had been omitted for the most recent procedure. There was also a history of hypertension and benign prostatic hypertrophy and the patient's regular medications included ramipril, simvastatin and solifenacin. On examination, the patient was afebrile, there was a pansystolic murmur radiating to the axilla and no peripheral stigmata of IE. There was lumbar spinal tenderness and neurological examination of the legs was normal. Blood tests revealed a haemoglobin of 11.8 g dl⁻¹ and white cell count of 16.24×10^9 l⁻¹ (neutrophils 75 %). C-reactive protein was 99 mg l⁻¹. Plain X-ray of the lumbar spine showed degenerative changes only but T2-weighted magnetic resonance imaging showed increased signal at the L5/S1 disc space and adjacent endplates, highly suggestive of discitis (see Fig. 1). Transthoracic echocardiogram showed a thickened mitral valve with bileaflet

prolapse, moderate mitral regurgitation and a mass attached to the posterior leaflet. Subsequent transoesophageal imaging confirmed these findings, the mass being highly suggestive of a large vegetation. The diagnosis of IE complicated by vertebral discitis was made.

Five sets of blood cultures were taken over 3 days and became positive within 48 h of incubation. The initial Gram stain showed a Gram-positive staining rod (see Fig. 2), occasionally forming chains. The cultures grew *Streptococcus mutans* identified by the API 20 Strep identification kit (bioMérieux) in our laboratory and confirmed by the Health Protection Agency UK Streptococcal Reference Laboratory. MICs for penicillin (0.032 mg l⁻¹) and ceftriaxone (0.094 mg l⁻¹) were determined by E-test (AB Biodisk).

Taking into account the history of allergy to penicillin, the patient was treated with intravenous ceftriaxone (2 g once daily for 6 weeks) and gentamicin (80 mg twice daily for the first 2 weeks).

After 2 weeks of therapy, the white cell count had normalized and the C-reactive protein had fallen from 99 to 15 mg l⁻¹. There were no cardiac symptoms, no haemodynamic compromise and serial echocardiography demonstrated reduced size of the vegetation with no increase in the severity of mitral regurgitation. His lumbar back pain had improved and the patient was able to mobilize comfortably. He was discharged to complete the remaining 4 weeks of intravenous ceftriaxone as outpatient parenteral therapy followed by 6 weeks of oral clindamycin (300 mg four times daily). He made a good recovery and repeat blood cultures following completion of antibiotic therapy were sterile.

Abbreviation: IE, infective endocarditis.

Fig. 1. T2-weighted magnetic resonance imaging of the lumbar spine showing increased signal at the L5/S1 disc space and adjacent endplates, highly suggestive of discitis.

Discussion

S. mutans is a commensal of the oral cavity and considered a primary cause of dental caries. The organism can survive in the bloodstream and has been reported as a cause of IE (Jung *et al.*, 2009). Several virulence factors have been identified for IE including a fibronectin-binding protein

Fig. 2. Gram stain of blood cultures showing pelomorphous Gram positive-staining organisms.

(AtlA autolysin) (Jung *et al.*, 2009), serotype-specific polysaccharides associated with phagocytosis resistance (Nakano & Ooshima, 2009) and cell surface protein antigen c, which is involved in platelet aggregation (Matsumoto-Nakano *et al.*, 2009).

Cells of *S. mutans* are non-haemolytic Gram-positive cocci that typically appear rod-shaped in acid media and can be mistaken as contaminating 'diphtheroids'. They show a more streptococcal appearance (cocci in chains) when subcultured on neutral media (Schelenz *et al.*, 2005). Conventional laboratory identification techniques can misidentify them and 16S rRNA gene sequencing may be required for formal identification (Petti *et al.*, 2005; Schelenz *et al.*, 2005).

Dental procedures may be associated with transient bacteraemia. However, there is no clear evidence that the prior administration of antibiotic prophylaxis is either effective or ineffective in preventing IE, or whether the potential harms and costs of prophylaxis outweigh any potential benefit (Oliver *et al.*, 2008). Additionally, there is evidence that cumulative exposure to bacteraemia is significantly greater from everyday procedures (such as tooth brushing) when compared to isolated invasive dental procedures (Roberts, 1999). Following literature reviews, the American Heart Association (AHA), UK National Institute for Health and Clinical Excellence (NICE) and European Society of Cardiology (ESC) published new guidelines for the prevention of IE (in 2007, 2008 and 2009, respectively) (Habib *et al.*, 2009; Richey *et al.*, 2008; Wilson *et al.*, 2007). Although all agree that the benefits of prophylaxis are unproven, the NICE guidelines recommend no prophylaxis for dental procedures for *any* patients 'at risk' of IE, whereas the AHA and ESC guidelines continue to recommend prophylaxis for 'high risk' patients with the poorest outcomes from IE (Richey *et al.*, 2008; Wilson *et al.*, 2007).

To our knowledge, this is the first reported case of *S. mutans* IE complicated by discitis. There was a clear temporal association with a dental procedure at an infected site which, following a dramatic change in guidelines, was not accompanied by antibiotic prophylaxis. The current UK NICE guidelines do not recommend antibiotic prophylaxis for *any* dental procedure whereas, in contrast, for gastro-intestinal or genito-urinary tract procedures prophylaxis is recommended at a site where infection is suspected (Richey *et al.*, 2008). In light of this case, we believe that there is an argument for recommending prophylaxis for dental procedures where infection is involved. Additionally, the question of whether our patient should receive antibiotic prophylaxis for future dental procedures remains contentious, particularly while there is discrepancy between national and international guidelines. However, following this experience, we are recommending continuing antibiotic prophylaxis for future dental procedures in our patient.

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