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***Streptococcus mutans* endocarditis: beware of the 'diphtheroid'**

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Streptococcus mutans, a major cause of dental caries and endocarditis, is easily dismissed by the inexperienced as a common non-pathogenic skin bacterial contaminant termed 'diphtheroid'.

CASE HISTORY

A man of 62 was admitted after three months of lethargy, malaise, night sweats and weight loss. Type 2 diabetes mellitus had been diagnosed at about the time these symptoms began and, on a home visit, his general practitioner had thought they were due to poor diabetic control. The patient had not experienced chest pain, cough, rigors or arthralgia. His medical history included a Ross procedure (pulmonary valve autograft to aortic position, pulmonary valve homograft) at age 35 for a congenital abnormality of the aortic valve. His medication was gliclazide and aspirin.

On examination he was pyrexial (38.2°C), and tachycardic. The principal abnormal physical sign was a mid-systolic murmur. There was no rash or splenomegaly. Haemoglobin was 11.6 g/L, white cell count $21.6 \times 10^9/L$ (neutrophils 95%), C-reactive protein 72 mg/L, erythrocyte sedimentation rate 94 mm/L, random blood glucose 13.1 mmol/L, HbA1c 9.6%. The initial diagnosis was hyperosmolar non-ketotic hyperglycaemia. Because of his persistent pyrexia, a set of peripheral blood cultures was taken which became positive within 24 h showing Gram-positive rods. Since a biochemical test for the identification

of Gram-positive rods gave no profile, the isolate was reported as 'diphtheroids' and presumed to be a contaminant. However, because of his previous valve replacement and his ongoing fevers the clinicians were advised to take more blood cultures. Three further sets of blood cultures became positive within two days. Gram-positive rods were again seen; and, on review of the original Gram films, the organisms were deemed more likely to be streptococci, possibly *S. mutans*. It was strongly suggested to exclude endocarditis in this patient and to review the state of his teeth. A transthoracic echocardiogram showed a small mobile vegetation on a thickened pulmonary valve. He was then treated for presumed streptococcal endocarditis with gentamicin 80 mg twice daily for two weeks and vancomycin twice daily for five weeks (he was allergic to penicillin). Eight teeth with advanced caries were removed while he was in hospital. His infection markers became normal.

Originally, the laboratory performed a biochemical test (API) for the identification of streptococci, which named the organisms as *Leuconostoc* sp. This was unlikely to be correct since the isolate was vancomycin sensitive whereas *Leuconostoc* spp. are resistant. After two weeks, the reference laboratory confirmed the isolate to be a fully sensitive *S. mutans*.

COMMENT

S. mutans belongs to a group of non-haemolytic Gram-positive cocci which typically appear rod-shaped on acid culture medium but show a streptococcal appearance, in chains, when subcultured into a neutral or alkaline broth. The organism was first described by Clarke in 1924, who noted the variation of morphology with the pH of the medium.¹ On the basis of their rod-shaped appearance when isolated from blood culture medium they can be easily dismissed as diphtheroids—i.e. non-pathogenic skin bacteria. In 1977 Emmerson and Eykyn² drew attention to this hazard, by reporting two cases of *S. mutans* endocarditis in which the organisms had been initially misidentified as diphtheroid contaminants.

Although diphtheroids are the second most common bacterial contaminants in blood cultures, the case presented here illustrates that 27 years on we still fall into the same trap of misidentifying an important pathogen and potentially delaying the diagnosis and treatment of a serious infection.³ Clinicians and microbiology staff need to be critical about the identification of 'diphtheroids' from blood cultures. New technology such as commercial identification kits may even add to the confusion.

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Intravitreal triamcinolone acetonide for refractory diabetic macular oedema

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Diabetic macular oedema is caused by hyperpermeability of the retinal vasculature and results in retinal thickening due to intraretinal fluid accumulation between the inner and outer plexiform layers. The Early Treatment Diabetic Retinopathy Study defined clinically significant macular oedema (CSMO) by the presence of one or more of the following: retinal thickening at or within 500 μm of the centre of the macula; hard exudates at or within 500 μm of the macular centre, if associated with adjacent retinal thickening; retinal thickening greater than or equal to one disc area, any part of which is within one disc diameter of the centre of the macular centre.¹ Focal argon laser photocoagulation is beneficial in non-ischaemic CSMO but less so in diffuse macular oedema.² A treatment now used for macular oedema in various diseases is intravitreal triamcinolone acetonide.

CASE HISTORY

A man of 46 sought advice after two days of bilateral photophobia with red, watery eyes. 8 years before, non-insulin-dependent diabetes had been diagnosed, but he was otherwise well. On examination, unaided visual acuity was 6/18 (improving to 6/6 pinhole) in the right eye and 6/6 in the left. He was diagnosed as having right-sided non-granulomatous uveitis without systemic associations. There were no signs of diabetic retinopathy or cystoid macular oedema. After two weeks of topical steroid treatment the uveitis resolved completely with visual improvement to 6/6 unaided bilaterally. Three months later, right diabetic maculopathy developed. CSMO was absent, so focal laser treatment was not indicated at this stage. Six months later, visual acuity had declined from 6/6 to 6/12 in his right eye

and CSMO was now evident, with macular thickening on ocular coherence tomography. Fluorescein angiography was performed before focal macular argon laser treatment. Three months post-laser there was little angiographic change, and because of the cystoid nature of his macular oedema it was felt that, if his visual acuity decreased, intravitreal triamcinolone should be tried. CSMO then developed in the left eye, and fluorescein angiography indicated that this was suitable for focal laser treatment; however, the right eye showed ischaemic maculopathy (Figure 1) for which laser treatment was thought inappropriate. A month later the patient's unaided visual acuity was 6/24 in the right eye and 6/18 in the left eye, without improvement with a pinhole. He refused laser treatment for the left eye but agreed to intravitreal triamcinolone on the right. 4 mg triamcinolone was injected into the right globe under sterile conditions. After a month visual acuity in this eye had improved to 6/9. On ocular coherence tomography, retinal thickness had lessened from 527 μm to 168 μm (Figure 2). This improvement was still evident six months post-treatment.

COMMENT

Focal macular oedema is characterized by leakage from microaneurysms and dilated capillary segments, whereas in diffuse macular oedema the leakage results from a generalized breakdown of the blood–retinal barrier, with formation of large cystoid spaces. The disappointing results with focal laser photocoagulation in conditions such as ischaemic diabetic maculopathy have prompted evaluation of other approaches. Corticosteroids have been used orally, topically and by periocular injection in the treatment of cystoid macular oedema because of their ability to inhibit the arachidonic acid pathway and

Figure 1 Fluorescein angiogram showing increased size and irregularity (arrows) of the foveal avascular zone

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