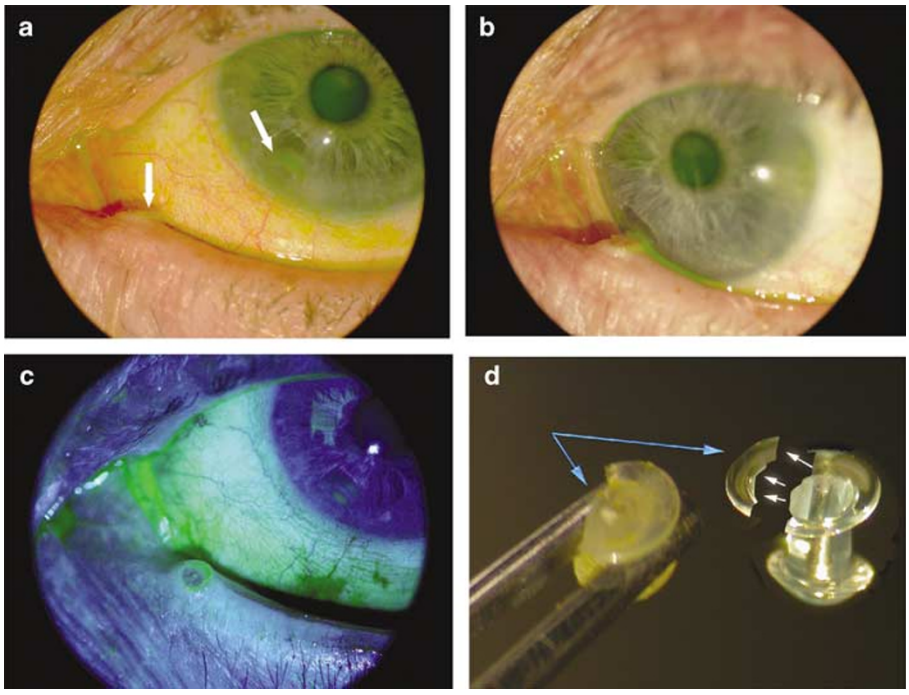

Figure 1 White arrows point to the PP and the corneal ulcer (a), coming in direct apposition on adduction (b). The lower lid is pulled to show PP broken cap (c). The whole plug is shown after removal (d, left), compared to an illustrated new plug with a torn lip (d, right).

diagnosis of PP-associated complications, careful examination of the punctum area is necessary in patients presenting with red eye and a history of PP insertion.

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Eye (2007) **21,** 1437–1438; doi:10.1038/sj.eye.6702952;
published online 17 August 2007

Sir,

Bilateral Group A streptococcal endogenous endophthalmitis following routine gynaecological surgery

When a patient is referred with a sticky, painful eye soon after an operation on a region of the body remote from the eye, and they are systemically unwell, a diagnosis of endogenous endophthalmitis should be considered. If after examining the eye endophthalmitis seems possible then blood cultures should be taken.

Case report

A previously healthy 62-year-old woman with no past ocular history was admitted with abdominal pain and red, painful eyes 2 days following a routine hysteroscopy for investigation of postmenopausal bleeding. The visual acuities were hand movements in each eye and the conjunctivae were injected with an accompanying pseudomembrane formation. Both corneas were opaque and the left was necrotic inferiorly.

Bilateral hypopyons were present, iris detail was obscured and no red reflex could be seen in either eye (Figures 1 and 2). On systemic examination, the patient was confused, pyrexial, septicaemic, and hypovolaemic. A *B*-haemolytic group A streptococcus was isolated on blood culture and a diagnosis of streptococcal bilateral endogenous endophthalmitis was made.

Treatment was commenced with topical ofloxacin and cefuroxime 5%, and oral ciprofloxacin 750 mg bd. Intravitreal amikacin 0.4 mg and vancomycin 1 mg was administered twice within the first 72 h. No organism was identified from the vitreous biopsy specimens. At 3 months following presentation, the right eye

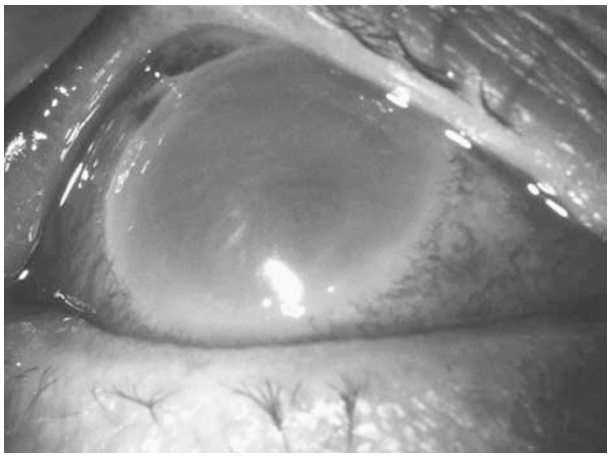

Figure 1 Right eye 1 day after presentation.

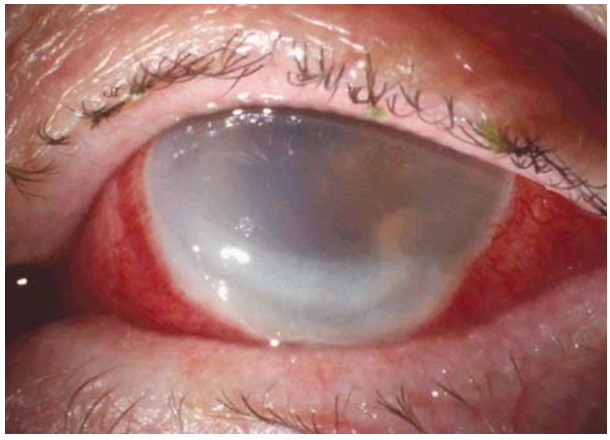

Figure 2 Left eye 1 day after presentation.

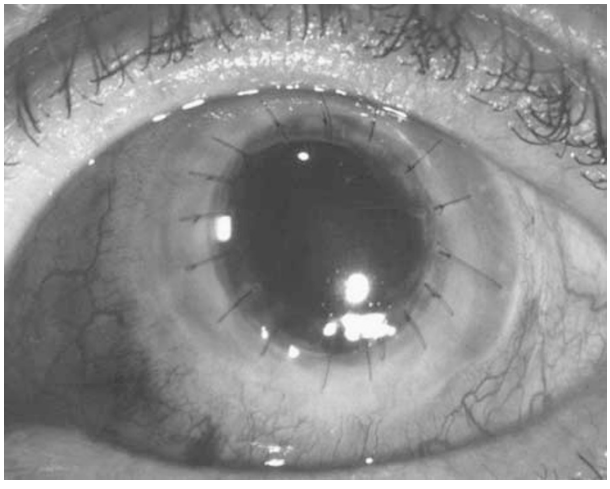

Figure 3 Left eye following anterior segment reconstruction. (For online use: Figure 1 has been uploaded in colour as Figure 4 and Figure 3 has been uploaded in colour as Figure 5.)

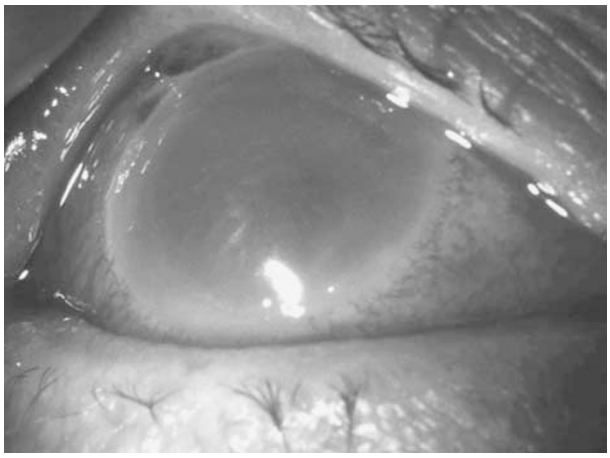

Figure 4 Right eye 1 day after presentation.

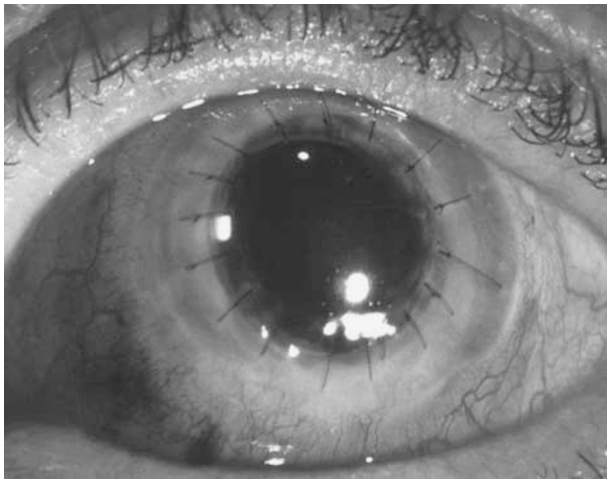

Figure 5 Left eye following anterior segment reconstruction.

had become phthisical and the left eye recovered to 3/60 with anterior-segment reconstructive surgery (Figure 3).

Comment

B-Haemolytic group A streptococcus has been reported to cause endophthalmitis following ocular surgery[1,2] but there are only three other reported cases in the literature of Group A endogenous infection.[3,4] In the largest review of endogenous bacterial endophthalmitis (EBE), 2 out of 267 cases were due to Group A streptococcus infection.[4] Infection occurs haematogenously and blood culture rather than vitreous biopsy is the most frequent means of establishing the diagnosis, which is initially mistaken in the majority of cases.

This is a rare case of streptococcal Group A EBE and the first case of endogenous endophthalmitis to follow gynaecological surgery. We recommend that EBE should be considered in patients who present with painful, red eyes following a surgical procedure and that blood cultures should be taken.

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Eye (2007) **21,** 1438–1440; doi:10.1038/sj.eye.6702956; published online 17 August 2007

Sir,

Reversible tetracycline staining of adult dentition in the treatment of chronic blepharitis

Low-dose oral tetracycline is often used in the management of chronic blepharitis.[1] Tetracyclines are broad-spectrum antibiotics and one of their side effects is discolouration of teeth that occurs inevitably in children.[2] We report an adult patient who developed reversible brown discolouration on her normal dentition after taking oral tetracycline for blepharitis.

Case report

A 54-year-old lady suffered from blepharitis. Because of local allergic reactions, fusidic acid was withdrawn from treatment. She remained symptomatic despite lid scrubs. A course of oral tetracycline 250 mg four times/day was prescribed. After 4 weeks of treatment, she noticed brown discolouration of her incisor teeth (Figure 1, top). The staining was completely removed by abrasive cleansing by dental surgeon (Figure 1, bottom). Prior to this incidence, this patient had not had any regular check-up or cleansing by dentist for over a year.

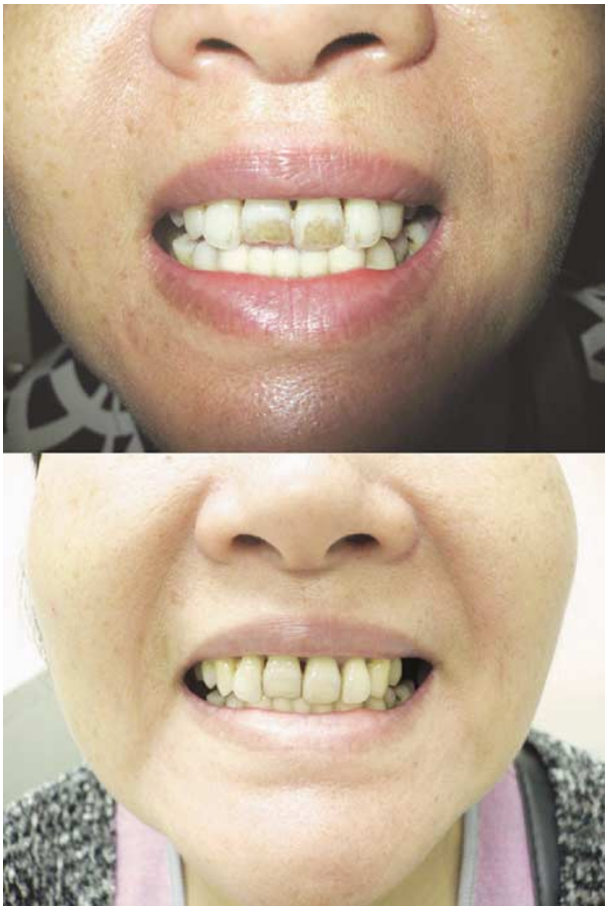

Figure 1 Top: Brown discolouration of teeth after 1 month's oral tetracycline; bottom: brown staining completely removed after cleansing by dental surgeon.

Comment

Chronic blepharitis is frequently associated with sebaceous gland dysfunction, plugging and inflammation of the meibomian glands.[3] Tetracyclines inhibit matrix metalloproteinase expression and bacterial lipase production, with a resultant change in the concentration of inflammatory free fatty acids in the tear film.[1,4] Tetracycline is known to cause permanent discolouration during odontogenesis in children by the formation of insoluble tetracycline–calcium orthophosphate complexes in the dentine and enamel which darken upon exposure to light.[2,5] The relative lack of free calcium protects the erupted permanent adult dentition against tetracycline-induced tooth discolouration. However, minocycline, a tetracycline derivative, has been reported to stain adult dentition in 3–6% of patients taking a daily dose >100 mg for longer than 1 month.[6] Owing to the full reversibility of discolouration in our patient, an 'extrinsic theory' could be one of the plausible mechanisms of staining. The theory states that minocycline is excreted in high concentration in saliva. The drug or its breakdown product forms insoluble salts by chelating with divalent metal ions in saliva and gingival fluid.[7] An alternative mechanism is explained by the attachment of minocycline to the acquired pellicle's glycoproteins. This etches the enamel, and demineralization/remineralization cycles occur. It oxidizes to an insoluble black quinone on exposure to air or from bacterial degradation of the aromatic ring.[8] Since these mechanisms of tooth staining involve excretion of concentrated minocycline in saliva and formation of insoluble black quinone from bacterial degradation of miniocycline, dehydration, poor oral and dental hygiene may be risk factors for the staining. UV radiation is also a possible