Actinomyces infection in porous polyethylene orbital implant

Abstract • Background: A 60-year-old patient developed actinomycotic inflammation within a porous polyethylene orbital implant which she received following enucleation. • Methods: She had repeated conjunctival exposures with inflammation; the primary implant was removed and replaced with another one. • Results: The anterior two-thirds of the porous implant was infiltrated with numerous actinomycotic granules surrounded by polymorphonuclear cells and necrotic debris. The organisms were demonstrated with Gram stains on the histopathologic preparations and with scanning electron microscopy. Within the zones of inflammation, the polyethylene skeleton of the implant was extensively damaged. • Conclusion: Actinomycetes have been described as causative organisms in conjunctivitis, blepharitis, canaliculitis, dacryocystitis and keratitis, but to the best of our knowledge actinomycotic involvement has never been reported in an infected porous orbital implant.

Introduction

The actinomycetes are gram-positive, filamentous organisms which grow slowly and cause chronic infections. The name, from the Greek aktinos ray and mykes fungus, derives from the fact that its growth within tissues leads to formation of rosettes surrounded by branching filaments of the fungi which resemble radiating rays. Actinomyces israelii and A. bovis are known to cause chronic infections in humans and other mammals. A. israelii is a normal pathogen of the pharyngo-oral mucosa in humans; it can be cultured from the majority of human tonsils [4]. It is usually commensal rather than pathogenic but under certain conditions it can act as a pathogen and grows as small rosettes (“sulfur granules”) leading to purulent, chronic inflammation in many parts of the body [5].

Within the eye and adnexal tissues the most common site for A. israelii infections is the lacrimal drainage system [13, 15]. In long-standing infections, aggregates of sulfur granules are known to form small irregular calcified bodies, dacryoliths, within the canaliculi and lacrimal sac [1, 7]. We recently encountered a case of actinomycotic infection within a porous polyethylene orbital implant at King Khaled Eye Specialist Hospital in Riyadh, Saudi Arabia. Although this organism has been described as a causative pathogen in conjunctivitis, keratitis, blepharitis, canaliculitis, dacryocystitis and orbital cellulitis, to the best of our knowledge it has never been reported in an infected integrated orbital implant [3, 6, 14].

Case report

A 60-year-old woman with phthisis bulbi of the left eye received an 18-mm porous polyethylene (Medpor) implant following enucleation. One month after surgery the patient developed exposure of the implant, measuring 3×3 mm, without any evidence of infection. Repeated smears and cultures of the exposed area were negative. The exposure was repaired with a hard palate mucosal graft, and superior and inferior fornices were deepened and lined with buccal mucosa.

The patient did well for approximately 5 months but then developed exposure of the implant again. At this point the lower fornix was well formed following the buccal graft but the upper fornix
Fig. 1 Biomicroscopic appearance of the socket with exposure of the orbital implant. Markedly hyperemic, edematous conjunctiva surrounds the exposed zone. A thin layer of yellowish purulent discharge is at the junction of the exposed implant material and inflamed conjunctiva (arrowheads).

Fig. 2 Gross appearance of the cut surface of the implant after surgical removal. Slightly elevated, yellow sulfur granules (asterisks) of varying sizes are clearly seen.

Fig. 3 Whole-mount preparation of the implant. Dense fibrovascular ingrowth (dark blue) is depicted posteriorly (arrowheads). The architecture of the implant is misshapen in the anterior two-thirds of the sphere; the polyethylene skeleton is damaged and reveals large, irregular spaces filled with necrotic debris. Masson's trichrome, original magnification x2.

Fig. 4 Light-microscopic appearance of the Actinomyces infection within the implant, with numerous polymorphonuclear cells, necrotic debris, and granules of organisms (G). The interface between the acute inflammatory reaction and the polyethylene (P) skeleton of the implant is marked with arrowheads. Insert: clusters of gram-positive actinomycetes (asterisk). Hematoxylin-eosin, original magnification X100; insert: Gram stain, original magnification X250.

was still scarred and shallow. The conjunctival margin of the exposed area was congested and slightly elevated but there was no discharge or other evidence of infection. The conjunctival cultures obtained from the site of exposure were negative. An exchange of orbital implant was planned but the patient refused further surgery. Two months later, she presented with an enlarged exposure surrounded with hyperemic, edematous conjunctiva and a small amount of yellowish discharge at the margin of the exposed area and in the inferior fornix (Fig. 1). Cultures obtained from the discharge grew gram-positive bacilli resembling Corynebacterium species, coagulase-negative staphylococci and Streptococcus viridans. The patient was placed on gentamicin drops and erythromycin ointment and admitted for orbital implant exchange and socket reconstruction with another hard palate graft.

At the time of surgery the implant was firmly attached posteriorly and had to be dissected from the underlying orbital soft tissues. The rectus muscles were detached from the implant and reattached to a smaller (16-mm) methyl methacrylate implant wrapped with homologous sclera in a “baseball” fashion. Anteriorly, the implant was covered with another hard palate graft. Postoperatively the graft healed well with a deep fornix inferiorly; the shallowness of the superior fornix persisted. No evidence of exposure or conjunctivitis was present 5 months after surgery.

On pathologic examination, the implant was partially covered with irregular fibrous tissue on its posterior aspect. Anteriorly, it was whitish-cream in color with focal hemorrhagic areas. On transverse sectioning, the posterior two-thirds of the sphere was mottled with brown, red, and yellow areas (Fig. 2). On histopathologic examination, fibrovascular ingrowth was dense in the posterior one-third of the implant as demonstrated with Masson's