Sir,

Response to 'The use of Medpor-coated tear drainage tube in conjunctivodacryocystorhinostomy'

Thank you for your kind letter. I am willing to talk about our experience about the exuberant conjunctival overgrowth because of the tube implantation.

First, I would like to thank Dr R Redmond[1] for his dissective study. According to our experience, conjunctival overgrowth was in faith the most common complication in the use of the Medpor-coated tear drainage tube and would cause tube obstruction. In our paper, we had discussed the problem. The reason for the exuberant conjunctival overgrowth is not very clear, and may be related to the porous polyethylene coat of the tube.

About the conjunctival overgrowth, for one thing, to reduce the granulation tissue overgrowth, we should tell the patients in detail how to care for the tube after operation. For another, the hypertrophic tissues can be excised and the stump cauterized to prevent the tissue regrowth. Then it is necessary to suture the conjunctiva around the tube.

If the recurrent conjunctival overgrowth actually happened time after time, removing the tube is the last choice. According to our experience, it is easy to remove the borosilicate glass tube. We first slit the Medpor sheath with a blade or scissors, then removed the glass tube with forceps. During the surgery it is necessary to avoid spalling the glass tube and to completely remove the Medpor sheath.

Reference

1 Redmond R. Correspondence to X Fan *et al* – the use of Medpor coated tear drainage tube in conjunctivodacryocystorhinostomy. *Eye* 2010; **24**: 196.

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Sir,

Cytomegalovirus endotheliitis following fluocinolone acetonide (Retisert) implant

We report a case of cytomegalovirus (CMV) corneal endotheliitis following fluocinolone implant for uveitis.

Case report

A 21-year-old HIV-negative Chinese woman presented to Singapore National Eye Centre with panuveitis and Snellen acuity 6/45 bilaterally. Behcet's disease was diagnosed and systemic immunosuppression commenced. Vision improved to 6/7.5 bilaterally, with relapsing inflammation. Her left eye became quiescent after insertion of fluocinolone acetonide 0.59 mg implant. The non-implanted eye required continued systemic therapy for control. Left phacoemulsification was required 4 months postimplant.

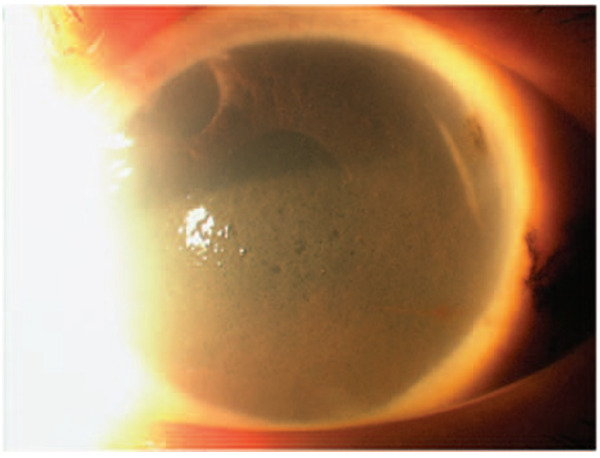

Figure 1 Inferior corneal oedema and keratic precipitates consistent with endotheliitis.

After 14 months, pigmented keratic precipitates and corneal oedema developed inferiorly. Trabeculectomy was performed for elevated intraocular pressure. Corneal oedema increased over the next 9 months (Figure 1). There was no evidence of retinitis (although fundoscopy was impossible once diffused corneal swelling occurred). At 2-year postimplantation, aqueous humour was positive for CMV DNA (3.6×10^4 copies viral DNA per ml) and mRNA by polymerase chain reaction. CMV endotheliitis was diagnosed. Serology for CMV IgG was positive, but negative for IgM and CMV antigen.

Fluocinolone was explanted to reverse local immunosuppression, and systemic valganciclovir commenced. Vitreous was positive for CMV DNA and mRNA. Eight months later, she remained CMV mRNA positive. Corneal oedema persisted despite subsequent insertion of a ganciclovir implant. This eye has only light perception vision.

Comment

Cytomegalovirus has been identified as a cause for anterior uveitis and endotheliitis in immunocompetent patients.[1,2] CMV retinitis can complicate intravitreal triamcinolone administration, and one case of post fluocinolone implant has been reported.[3]

Our patient had CMV endotheliitis, and this is the first report of this complication following fluocinolone implantation. We cannot explain why the cornea was the predominant site of infection in our patient, considering vitreous was positive for CMV. However, histopathological studies of CMV-infected eyes in immunodeficiency showed the presence of virus in iris, ciliary body, and cornea.[4] We suspect that CMV remains latent in the anterior segment in some eyes and reactivates if local immunity is altered. This is supported by the patient's serological findings.

Anterior segment CMV infection can develop in immunocompromised eyes, and clinicians should be

aware of their presentation. Aqueous sampling for CMV should be considered when introducing intravitreal depots of steroid.

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Sir,

Complete external ophthalmoplegia in headache, neurologic deficits, and cerebrospinal fluid lymphocytosis (HaNDL) syndrome

The headache, neurologic deficits, and cerebrospinal fluid (CSF) lymphocytosis (HaNDL) syndrome is characterized by a temporary neurological deficit followed by a severe headache with persistent CSF lymphocytosis. Other features include an increased CSF protein, an increased CSF opening pressure, and a viral prodrome. The exact pathogenesis of HaNDL is unknown.[1] We report a patient with HaNDL syndrome presenting with complete external ophthalmoplegia.

Case report

A healthy 18-year-old woman presented with a 1-week history of headache, nausea, vomiting, and fever. She developed severe visual loss over 4 days followed by acute ophthalmoplegia.

Her best-corrected visual acuity was hand motions, OD, and light perception OS. Humphrey visual fields revealed severe peripheral constriction OU. Her pupils were round and 6 mm; she had no relative afferent pupillary defect. She had bilateral ptosis and no extraocular motility. Vestibulo-ocular reflexes were absent. Anterior chamber examination was normal. Optic discs were swollen with slit haemorrhages in the perimacular area. She had diminished light touch in patchy areas over her upper extremities.

Magnetic resonance imaging (MRI) of the brain and MR venogram of the brain with and without contrast were normal. CSF revealed an elevated opening pressure of 320 mm H20, 172 lymphocytes, 6 red blood cells, protein of 92 mg per 100 ml, and glucose of 55 mg per 100 ml. CSF VDRL and Lyme titres were negative. CSF cultures and PCR for HIV, HSV 1 and 2, and EBV were all negative. CSF IgG synthesis rate and oligoclonal bands were negative. TSH, antinuclear antibody, and acetylcholine receptor antibodies were normal. Single-fibre electromyography was unremarkable.

After CSF drainage with repeated lumbar punctures and receiving i.v. methylprednisolone 1 g/day for 5 days, her symptoms and signs resolved in several weeks, and her final visual acuity was 20/20 OU.

Comment

Blurred vision, photophobia, homonymous hemianopsia, photopsias, cortical blindness, papilledema, and sixth nerve palsies have previously been associated with HaNDL.[2] Although HaNDL is recognized as a distinct entity (IHS 7.8) in the International Headache Society Classification ICHD-II,[3] monophasic cases like this could also represent unusual forms of viral meningitis. In addition to the self-limiting features of headache, dysesthesias, CSF lymphocytosis, and papilledema, this patient presents with transient complete external ophthalmoplegia, a neuro-ophthalmic sign not previously observed in HaNDL. External ophthalmoplegia can also be seen in idiopathic intracranial hypertension (IIH) and usually resolves with lowering of intracranial pressure.[4] Ophthalmoplegia in HaNDL may arise from the effects of increased intracranial pressure and resolve with the lowering of CSF pressure in a similar manner to IIH.

Therefore, transient ophthalmoplegia should be recognized as part of the clinical spectrum of HaNDL syndrome. Although it is an uncommon disorder, ophthalmologists should include HaNDL in the differential diagnosis of any patient with papilloedema and transient neuro-ophthalmic signs.

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