Figure 2 Adult nasolacrimal duct bypass tube (unflanged) of Chandler type 1 removed during dacryocystorhinostomy. The tube was 3 mm in diameter and 32 mm long, with a silk fixation suture still in place.

soft tissues around the inferior meatus. The malposition of the nasolacrimal intubation was confirmed at subsequent dacryocystorhinostomy, when the unflanged silicone tube of 3 mm outside diameter and 32 mm length was removed (Fig 2). The patient has been symptom free since surgery.

Comment

This clinical case provided a rare opportunity to demonstrate the actual course of a lacrimal bypass tube designed for placement within the nasolacrimal duct. The intended position for this and similar bypass tubes, such as those of Chandler¹ or Metaireau,^{2,3} is within the nasolacrimal duct; this case raises the issue as to how many *actually* lie in this location – an issue that might merit further investigation.

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Simultaneous retinal and optic nerve lesions in toxoplasmosis: the advantages of magnetic resonance imaging

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It is extremely rare for an optic nerve lesion to occur simultaneously with the reactivation of toxoplasmic retinochoroiditis.^{1–4} We report on a patient in whom the occurrence of an active optic nerve lesion was further confirmed using magnetic resonance imaging (MRI). This is the first clinical report of such an association.

Case report

A healthy 22-year-old man without HIV risk factors presented with right retro-ocular pain exacerbated by eye movements. Two days later he noted decreased vision in that eye. There had been two episodes of painless visual impairment in his right eye 1 and 4 years previously, leaving him with a paracentral scotoma. The first episode was diagnosed elsewhere as optic neuritis and the second as toxoplasmosis.

Examination of the right eye revealed visual acuity 6/12, right relative afferent pupillary defect, 1+ cells in the anterior chamber, and 2+ cells in the vitreous on slit-lamp examination. Visual fields showed a superior arcuate defect (Fig 1A) and a slightly blurred disc and two areas of active choroidoretinitis on funduscopy (Fig 1B). Examination of the left eye was entirely normal with a visual acuity of 6/4.

MRI of the optic nerves with STIR sequences⁵ revealed a 15 mm long lesion in the mid-portion of the right optic nerve (Fig 1C), non-adjacent to the globe. The lesion enhanced after gadolinium injection, indicating the presence of active inflammation.⁶ Brain MRI was normal.

The blood count, erythrocyte sedimentation rate (2 mm/h), serum angiotensin converting enzyme, autoantibodies, viral, and syphilis serologies were all normal, together with

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Figure 1 Findings in the patient at presentation. (A) Fundus photography, right eye. Hazy view of the fundus due to the vitritis. Two foci of active inflammation are seen paramacular nasal and papillomacular inferotemporally. (B) Right superior arcuate visual field defect on Bjerrum tangent screen. (C) MRI, orbital coronal STIR (TR 1500/40/150) image, 5 mm thickness. The optic nerves and surrounding muscle cone are demonstrated and there is increased signal in the right optic nerve (arrow). Images were obtained using a 0.5 T system (Picker International).

Fig 1A

Fig 1C

Fig 1B

examination and serological testing of the cerebrospinal fluid. Initial *Toxoplasma* titres were dye test 125 IU. Active toxoplasmosis was diagnosed clinically and, owing to the location of the retinal lesions, the patient was started on oral prednisone, pyrimethamine, sulphadiazine, and folic acid supplementation. Two weeks later the right ocular pain had disappeared, visual acuity had

improved to 6/7.5, and intraocular inflammation had decreased. The visual field defect (Fig 2A) and fundus examination were unchanged. The *Toxoplasma* dye test titre was now 250 IU. Two months later visual acuity in the right eye was 6/5 and intraocular inflammation had subsided. The visual field defect persisted but on fundus examination the retinal lesions were inactive (Fig 2B). Six months later, the situation was unchanged and the *Toxoplasma* dye test titre was now 30 IU.

Comment
Toxoplasmosis is a common cause of ocular infection producing mostly retinitis or retino-

Fig 2A

Fig 2B

Figure 2 (A) Goldmann visual field, right eye, 2 weeks after starting treatment, showing a superior arcuate scotoma. (B) Fundus photograph, right eye, 2 months after the onset of symptoms. The vitreous is clear and the retinal lesions are inactive.

choroiditis and less frequently papillitis or optic neuritis. Our patient presented with simultaneous reactivation of two areas of choroidoretinitis, characteristic of toxoplasmosis, which could be related to the loss of visual acuity and the visual field defect. However, the painful eye movements and the afferent pupillary defect were more suggestive of optic nerve disease.^{7,8} This was confirmed by an MRI examination which revealed an active inflammatory lesion in mid-portion of the right optic nerve, separate from the retinal lesions. Such optic nerve lesions occur in demyelinating disease,⁹ in granulomatous disease and in Leber's hereditary optic neuropathy,¹⁰ which is usually situated more posteriorly.

However, the optic nerve lesion is more likely to have resulted from an active toxoplasmic lesion, particularly as there were no other features of demyelination. *Toxoplasma gondii* has been isolated from the optic nerves at post mortem in an infant with congenital toxoplasmosis,¹ a patient receiving long term steroids,⁴ and in two patients with AIDS.^{2,3} Rarely, the cysts are associated with inflammation and demyelination of the optic nerve.¹¹

The present report is unique in describing an immunocompetent adult who, in addition to active retinal toxoplasmosis, had a simultaneously active retrobulbar optic nerve lesion on MRI. This is the first in vivo report of such an association, raising the possibility that optic nerve lesions may be more common in cases of typical toxoplasmic retinochoroiditis than previously considered, and that MRI may be valuable if visual function does not correlate with

the clinical presentation or with the topography of the retinal lesions.

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Focal electroretinogram and visual field defect in multiple evanescent white dot syndrome

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Multiple evanescent white dot syndrome (MEWDS) is characterised by decreased visual acuity, white dots at the level of retinal pigment epithelium (RPE), scotoma, including enlargement of the blind spot, and visual return without treatment.^{1,2} Although electrophysiological data, such as reduced a-wave in electroretinogram (ERG) and reduced early receptor potential,² indicate that the disease primarily affects the RPE and the outer segments of photoreceptors, optic nerve involvement has been reported to be a possible cause of the scotoma.³ On the other hand, several reports^{4,5,6,7} suggest that a disease known as 'acute idiopathic blind spot enlargement (AIBSE)',⁸ in which the enlargement is

caused by retinal dysfunction, and MEWDS are the same clinical entities. We recorded focal electroretinographs in two patients with MEWDS to study the nature of the scotoma.

Case reports

CASE 1

A 32-year-old woman noticed a central scotoma in the left eye, and was examined 6 days after the onset. Corrected visual acuity was 1.2 right eye and 0.2 left eye. Fundus examination of the left eye revealed multiple white lesions deep in the retina and fluorescein angiography demonstrated