Comment

Atypical manifestations of retinoblastoma (presenting at older age, with unusual features such as pseudohypopyon, hyphaema, or vitreous haemorrhage) can pose diagnosis challenges. Retinoblastoma masquerading as Coats' disease has been described previously.[1] This case is unique because ultrasonography and fluorescein angiography strongly mimicked typical findings in Coats' disease, including lack of calcification, dilated telangiectatic vessels with leakage, and peripheral non-perfusion. Shields and Shields[2] have suggested that the calibre and distribution of retinal blood vessels can help distinguish Coats' from retinoblastoma. In this case, fluorescein angiography was misleading. It is important for ophthalmologists to maintain a high index of suspicion for retinoblastoma in all children with unusual ocular diseases in which the diagnosis is not clear.

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HD Lam[1,2], MA Samuel[1], NA Rao[1] and AL Murphree[1]

[1]Children's Hospital Los Angeles, Doheny Retina Institute, Keck School of Medicine, University of Southern California, Los Angeles, CA, USA
[2]Department of Ophthalmology, UCSF medical Center, San Francisco, CA, USA
E-mail: hhdawnlam@yahoo.com

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Sir,

Reply to Wong *et al*

Wong *et al*[1] claim to have reported the first case of non-tuberculous *mycobacterium* (NTM) infectious crystalline keratopathy in a 'non-traumatized' eye. However, their patient had suffered a dendritic ulcer 6 weeks before and had received topical steroids, two significant risk factors for NTM keratitis. Four cases of corneal ulcer in which NTM were isolated with no predisposing risk factors have been described previously.[2]

Wong *et al*[1] recommend the use of fourth generation fluoroquinolones as a first-line treatment for this condition, although no *in vitro* sensitivity testing was demonstrated for the 0.3% gatifloxacin they used. *Mycobacterium abscessus* is the most resistant NTM, typically showing sensitivity only to clarithromycin, azithromycin, and amikacin, and cannot be distinguished from the closely related but less resistant *M. chelonae* in routine microbiological cultures.[3] DNA sequencing of the suspected pathogen was not carried out in this case, and it is not clear whether repeat microbiological sampling was done when the keratopathy recurred. The true identity of the causative organism is therefore in doubt.

NTM are ubiquitous organisms whose pathogenesis is far from completely understood; a genetic variation in individual susceptibility to infections with these atypical mycobacteria has been described.[4]

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G Clare[1] and S Mitchell[2]

[1]Moorfields Eye Hospital, London, UK
[2]Chelsea and Westminster NHS Trust, London, UK
E-mail: gaclare@doctors.org.uk

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Sir,

Reply to D Shome *et al*

We read with great interest the article by D Shome *et al*, 'Ptosis caused by orbicularis myokymia and treated with botulinum toxin'.

We wish to raise the following points:

The patient did not have a ptosis but blepharospasm, resulting in a decrease in palpebral aperture size in the left eye compared to the right.

The fact that the left upper lid was relatively lower than the right would presumably be a function of increased left orbicularis oculi tone relative to the right.

Furthermore, comparing the two photographs Figure 1a and Figure 2, it would appear that the patient had been squinting, that is, the palpebral apertures on the right eye were also greater in Figure 2 compared to Figure 1a. The authors have not made any attempt to explain this. Was there in fact an element of right

blepharospasm? Did they make electromyographic recordings from the right eye?

How do they explain the increase in right-sided palpebral aperture size between the figures, given that botulinum toxin was only injected around the left eye?

N Malik and N Joshi

Cranio Orbito Palpebral Service, Chelsea & Westminster Hospital, London, UK
E-mail: nabs.100@virgin.net

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Sir,

Reply to Malik *et al*

We wish to thank Dr Malik *et al* for their interest in our article 'Ptosis caused by orbicularis myokymia and treated with botulinum toxin—a case report'.

We agree that this patient did not have actual ptosis. In fact, the first line of the discussion of our published article[1] states 'Overactivity of the orbicularis oculi presents with a reduced palpebral aperture simulating ptosis, due to a disparity between the eyelid closing (orbicularis oculi) and eyelid opening (levator palpebrae superioris) muscles'. The patient's complaint was ptosis and she was referred to us for treatment of ptosis; Dr Malik and Dr Joshi would appreciate that the mention of 'ptosis' was made by us with reference to the appearance of the left eye and not the pathology *per se*.

This case was interesting as there were no obvious fibrillations of the left upper or lower eyelids seen, even on prolonged examination under magnification—so the overactivity of the orbicularis oculi muscle was not readily apparent. Hence, electromyography of the left orbicularis oculi muscle was deemed necessary—this became even more essential when the patient, a very well informed young lady, demanded objective evidence of our diagnosis. The electromyography of the right eye was performed as a control and did not demonstrate the repetitive grouped motor potentials, seen on the left side. Real-time evaluation did not suggest any reduction of the palpebral aperture on the right side. The patient has been under follow-up for greater than 18 months now, with no recurrence in the condition.

Acknowledgements

We thank Dr Malik and Dr Joshi for their comments once again.

Reference

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D Shome, V Jain and S Natarajan

Aditya Jyot Eye Hospital, Mumbai, Maharashtra, India
E-mail: debraj_shome@yahoo.com

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Sir,

Pre-macular nematode in diffuse unilateral subacute neuroretinitis

Diffuse unilateral subacute neuroretinitis (DUSN) is caused by a variety of nematodes, mostly subretinal, frequently leading to panretinal degeneration and visual loss.[1,2] We report the unusual presentation of a large nematode in DUSN.

Case report

A healthy 28-year-old south Indian man presented to us with an unremitting floater in the right eye (RE) for 1 week. Snellen acuity was 6/6 in RE and 6/12 in the left eye (LE). Examination of RE was unremarkable. LE fundus revealed a hyperkinetic non-segmented, 6.8 mm worm, apparently trapped under pre-foveal internal limiting membrane (ILM) (Figure 1a). There were midperipheral tracks of pigmentary degeneration; vitreous was quiet. The patient neither had any previous history of fever, skin rashes or fits nor any treatment for filariasis. Systemic evaluation and laboratory investigations, including a nocturnal peripheral smear (for microfilaria), were negative. Optical coherence tomography (*Stratus*OCT, Carl Zeiss Meditec, Dublin, CA, USA) confirmed the worm's sub-ILM location (Figure 1b). The patient was initially prescribed oral albendazole (400 mg o.d.) and diethylcarbamazine (100 mg t.i.d.). When status quo persisted for a month, vitrectomy was performed with patient's informed consent. Perifoveal capillaries bled during posterior-hyaloid removal. An extrafoveal tear occurred while aspirating the blood-trapped worm. ILM was removed and perfluoropropane–air tamponade used. The worm could not be subjected to parasitological evaluation because it disintegrated during the traumatic aspiration. Post-operatively, the eye remained quiet, with retained preoperative vision, intact macula and minimal juxtafoveal atrophy (Figure 1c and d) for 6 months.

Comment

This case had many unusual attributes: While the subretinal tracks (Figure 2) pointed to trans-retinal migration of the worm as described in DUSN,[1] this is the first OCT-documentation of its sub-ILM location, which facilitated the extraordinary motility of this suspected filarial nematode (endemic in patient's native area). Previously reported nematodes were smaller, slow-moving, and subretinal.[1–4] This worm did not produce the oft-reported intraocular inflammation, macular oedema or visual loss,[1–3] probably because its pre-macular migration and sequestration prevented the deleterious effects of prolonged subretinal movements.