

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?

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Eye (2008) **22**, 1197–1198; doi:10.1038/sj.eye.6703059; published online 20 June 2008

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 article¹ 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

- 1 Shome D, Jain V, Natarajan S. Ptosis caused by orbicularis myokymia and treated with botulinum toxin: a case report. *Eye* 2007; **21**: 444–445.

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Eye (2008) **22**, 1198; doi:10.1038/sj.eye.6703060; published online 21 March 2008

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 (*StratusOCT*, 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.