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

*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 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**

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.

D Shome, V Jain and S Natarajan

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

*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 (*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.

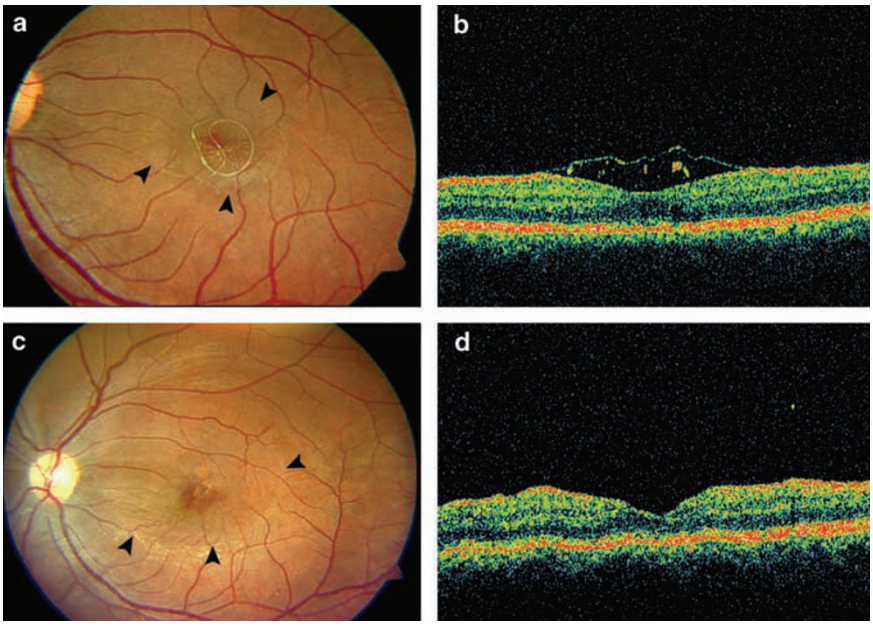


**Figure 1** (a) Fundus photograph of the LE. A 6.8-mm long, non-segmented, tapered worm is seen in front of the central fovea, trapped under the locally detached ILM as indicated by arrowheads. (b) Horizontal 5 mm OCT scan through the left fovea, showing a dome-shaped, lax, undulating membrane corresponding to the clinically suspected ILM detachment; with rapidly shifting hyper-reflective sub-ILM echoes corresponding to the worm trapped underneath. (c) One month after vitrectomy, LE shows a small juxtafoveal area of retinal atrophy superotemporally, representing closed iatrogenic parafoveal retinal hole. The edges of peeled ILM are clearly visible (arrowheads). (d) Post-operative OCT scan in the 'repeat mode' through the LE macula shows normalized foveal contours; atrophic patch was observed only in superotemporal scans (not seen here).

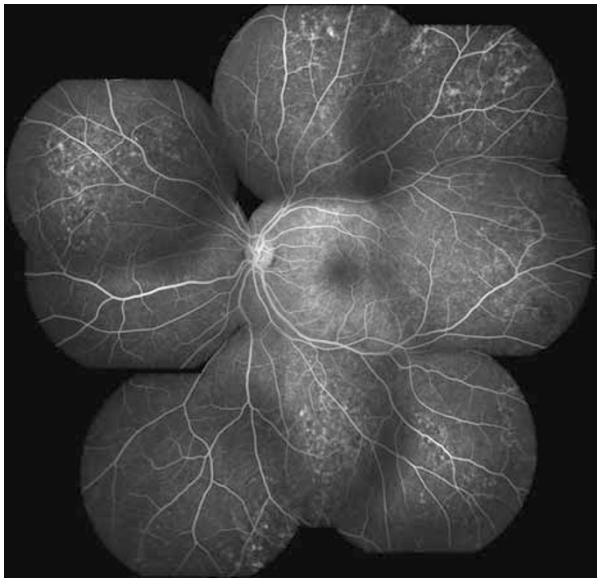

**Figure 2** Composite fluorescein angiogram of the LE, highlighting the classic subretinal worm-tracks of DUSN, leading up to the macula. The worm itself is not visible.

Our patient thus became symptomatic in the usually 'insidious' initial stage.[1] However, subclinical trauma to macula due to incessant flagellations by the worm probably contributed to the intraoperative bleeding and tear. This case presented a management dilemma: the pre-foveal parasite could neither be photocoagulated, nor killed by pharmacotherapy, which failed in absence of intraocular inflammation.[2,3,5] In view of the poor long-term prognosis, we recommend early surgical removal in such a case if photocoagulation is not feasible; even in the presence of good vision and no inflammation.

The authors declare no proprietary/conflict of interest in the manuscript; no funds were obtained for this project from any source.

## References

1 Gass JDM, Braunstein RA. Further observations concerning the diffuse unilateral subacute neuroretinitis syndrome. *Arch Ophthalmol* 1983; **101**: 1689–1697.
2 Cortez R, Denny JP, Muci-Mendoza R, Ramirez G, Fuenmayor D, Jaffe GJ. Diffuse unilateral subacute neuroretinitis in Venezuela. *Ophthalmology* 2005; **112**: 2110–2214.
3 Myint K, Sahay R, Mon S, Saravanan VR, Narendran V, Dhillon B. 'Worm in the eye': the rationale for treatment of DUSN in south India. *Br J Ophthalmol* 2006; **90**: 1125–1127.
4 Yamamoto S, Hayashi M, Takeuchi S. Surgically removed submacular nematode. *Br J Ophthalmol* 1999; **83**: 1088.
5 Souza EC, Casella AM, Nakashima Y, Monteiro ML. Clinical features and outcomes of patients with diffuse unilateral subacute neuroretinitis treated with oral albendazole. *Am J Ophthalmol* 2005; **140**: 437–445.

D Shukla and S Chakraborty

Retina-Vitreous Service, Aravind Eye Hospital and Postgraduate Institute of Ophthalmology, Madurai, Tamil Nadu, India
E-mail: daksh66@gmail.com

*Eye* (2008) **22,** 1198–1200; doi:10.1038/sj.eye.6703074; published online 21 December 2007

Sir,

**Simple method to reduce iatrogenic retinal trauma during vitreous surgery**

Posterior iatrogenic retinal trauma is a known complication of vitreous surgery.[1] We have had two cases where the endoillumination pipe has caused direct retinal trauma during epiretinal or internal-limiting membrane peel. This is a delicate procedure that demands intense concentration, often using a viewing lens that greatly restricts the visible field.

To reduce the likelihood of trauma during this stage of the procedure, we employed the simple use of a length of butterfly tubing (Figures 1 and 2) threaded along the illumination pipe to limit the depth to which the illumination instrument can be inserted into the eye. This butterfly tubing can be adjusted in length as required. This has also been employed during a complex four-port diabetic delamination surgery, where an assistant holds the light pipe. The technique should be particularly useful for trainee retinal surgeons.

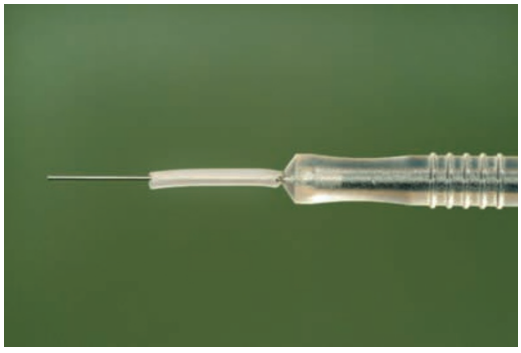

**Figure 1** Butterfly tubing cut to desired length and threaded along endoillumination pipe.

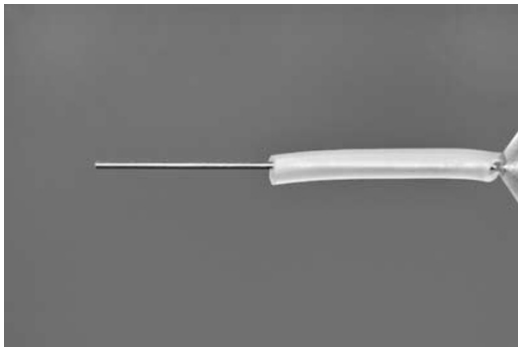

**Figure 2** Butterfly tubing cut to desired length and threaded along endoillumination pipe.

Thankfully, we have not had any similar iatrogenic breaks after employing this device.

No proprietary interests or research funding.

**Reference**

1 Michels RG, Wilkinson CP, Rice TA. *Retinal Detachment*. C V Mosby: St Louis, 1990, p869.

C Goldsmith[1], D Gupta[1] and RL Burton[2]

[1]James Paget University Hospital, Gorleston, UK
[2]Norfolk and Norwich University Hospital, Norwich, UK
E-mail: craig.goldsmith@jpaget.nhs.uk

*Eye* (2008) **22,** 1200; doi:10.1038/sj.eye.6703094; published online 1 February 2008

Sir,

**Intravitreal triamcinolone acetonide as an adjunct in the treatment of severe ocular toxoplasmosis**

Treatment of ocular toxoplasmosis is highly controversial. Results of such treatment depend on host factors, such as age and immune status, as well as on parasite factors.[1] The use of corticosteroids is even more debatable. Although most uveitis specialists agree that corticosteroid therapy without the concurrent use of antimicrobial agents can lead to severe ocular tissue destruction, patients who did well treated with corticosteroids alone have been seen.[1]

Aggressive cases, such as the one reported by Backhouse *et al*, have also been reported. It is important to note, however, that clinical deterioration did not occur immediately after the introduction of oral or intravitreal corticosteroids. On the contrary, in these two circumstances it appears that some improvement was initially observed. Of note, the antimicrobial agent was introduced 1 month following intravitreal triamcinolone acetonide. Taking into account that clinical picture worsening occurred only 2 weeks thereafter (6 weeks following intraocular injection), the temporal relationship strongly suggests that the intravitreal triamcinolone acetonide injection was not directly related to the outcome.

In our opinion, intravitreal corticosteroids should be used with caution in active ocular toxoplasmosis. Clinicians should avoid their use on recalcitrant, rapid worsening cases or those with questionable diagnosis. Intravitreal steroids without appropriate antimicrobial drugs should also be discouraged. On the other hand, there may be a role for them in patients with relatively controlled infection, as those we reported previously on this journal.[2]

**References**

1 Holland GN LX Edward Jackson Lecture Ocular toxoplasmosis: a global reassessment Part II: disease