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Sir,

Trochlear displacement by orbital plexiform neuroma: a novel mechanism causing superior oblique underaction

We present signs of superior oblique underaction in a patient with orbital plexiform neuroma with radiological evidence of trochlear displacement as the primary cause of underaction. We postulate mechanisms that account for the features seen on ocular motility examination in this particular tumour.

We present unusual signs in a case of unilateral medial orbital plexiform neuroma in a 22-year-old white male with neurofibromatosis type 1.

The patient presented with significant mechanical ptosis of the left upper lid associated with a rubbery tumour in the superomedial orbit. MRI scanning showed a large plexiform neuroma in the left superomedial orbit causing ptosis by anteroinferior displacement of the tarsal plate. He underwent uncomplicated levator dehiscence repair to elevate the upper lid with deliberate avoidance of the tumour. He made a good recovery, with a good cosmetic and excellent functional result.

It had been noted that examination of eye movements revealed striking features of left inferior oblique overaction, with gross hyperdeviation of the left eye on right gaze (Figure 1). There was no associated diplopia as the bulk of tumour at the nasal bridge obscures the view from the left eye on right gaze (Figure 2). Of note, the patient was not concerned by his eye movement abnormalities (for the reason outlined above), and was purely interested in correction of his ptosis for functional purposes.

Comment

It was initially thought that the plexiform neuroma was inducing a left fourth nerve palsy; however, the trochlear nerve enters the superior oblique muscle approximately one-third the way along its length from its origin at the body of sphenoid, above the tendinous ring, and the trochlea. The MRI scan shows that the tumour does not extend much further posteriorly than the trochlea (Figure 3).

Additionally, the trochlear nerve enters the muscle from the orbital aspect, rather than the side adjacent to the ethmoid and frontal bone. The tumour is not seen on the orbital side of the muscle (Figure 4). The MRI scan shows the tumour extending posteriorly into the orbit and beneath the trochlea, detaching it from the frontal bone. The trochlea is seen at the angle of the superior oblique tendon. Comparison with the contralateral anatomy shows distinct posterior displacement of the trochlear complex with the trochlea being attached to the tumour rather than the frontal bone. The amount of displacement does not, however, appear consistent with the degree of superior oblique underaction.

Plexiform neuromas have unusual mechanical properties, with firm, rubbery characteristics. It may be that in addition to inducing displacement of the trochlea, the tumour also acts like an elastic band, allowing the trochlea to travel posteriorly into the orbit upon contraction of the superior oblique muscle, and pulling the complex forward again during relaxation. This would ensure two things:

1. The force of contraction is not transmitted to the globe, inducing the appearances of inferior oblique overaction (superior oblique underaction).
2. The tendon-muscle complex does not undergo contracture. Contracture of the complex would eventually partially reverse the effects of trochlear displacement by taking up the slack in the tendon.

Posterior displacement of the trochlea complex also redirects the superior oblique tendon, increasing its

Figure 1 Eye movements demonstrating left superior oblique underaction on right gaze simulating inferior oblique overaction. Notice that tumour bulk effectively occludes left eye on right gaze.

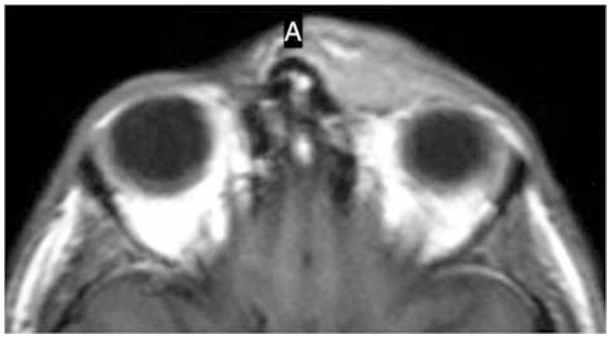

Figure 2 Axial MRI showing plexiform neuroma at medial canthus extending posteriorly into orbit.

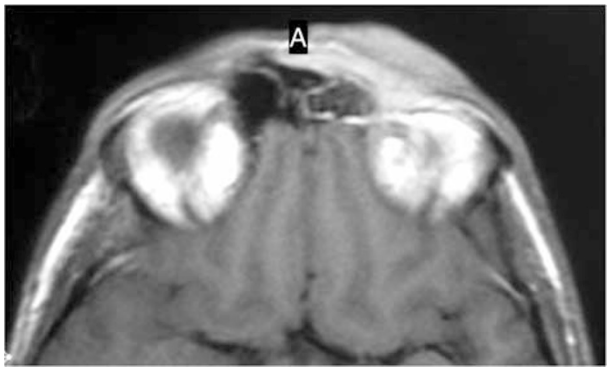

Figure 3 Axial MRI showing posterior displacement of the left trochlea, with increase in angle between the two limbs of the superior oblique tendon.

incyclotorsion vector and reducing the depression vector (Figure 4). This acts synergistically to increase the observed ocular motility abnormality.

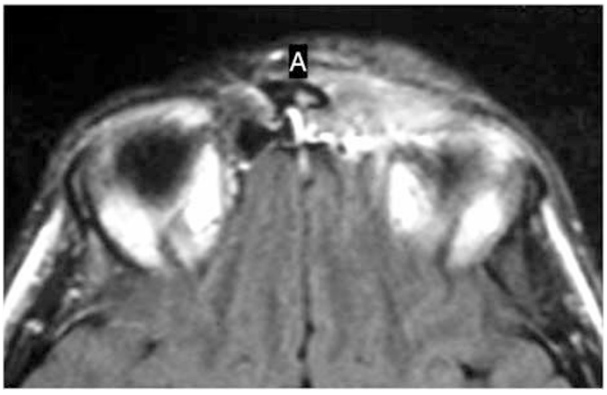

Figure 4 Axial MRI. Note that there is no posterior invasion of the orbit by the tumour but that the trochlea has been displaced posteriorly and laterally.

While numerous case reports and case series of fronto-orbital mucocoeles and other tumours exist, this case is the first report of trochlear displacement by this rare orbital tumour. It is interesting to consider that the mechanical properties of the tumour substance may influence the forces it exerts and has exerted upon it by surrounding tissues.

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Sir,

Outcomes of macular hole surgery

We note with interest that Jaycock *et al*,[1] in their report on the outcomes of macular hole surgery, found no evidence of ICG retinotoxicity. This is based on their findings that patients who underwent indocyanine green (ICG) assisted internal limiting membrane (ILM) peel had better anatomical and visual outcomes than both the ILM peel group as a whole or those who had no ILM peel. As they noted there are many publications on the subject of ICG toxicity to the retina, often with contradictory conclusions. We have previously reported our experience with ICG-assisted ILM peel.[2] Our patients achieved a high rate of anatomical hole closure but visual results were disappointing. As a result of these findings we performed an audit of macular hole surgery in our department. This confirmed the poor visual outcome with ICG-assisted ILM peel described in our paper and recommended we discontinue ICG use and use Trypan blue as an alternative. Our current practice is to use membrane blue (DORC International bv, Zuidland, Holland) for all cases requiring ILM peel. Recently, we have reaudited our macular hole outcomes in light of this change in practice.

The anatomical success and visual improvements of both our initial and repeat audit are summarised in Table 1. The high rate of anatomical hole closure with ICG remains with membrane blue but without the adverse effect on visual outcome we experienced with ICG. No other aspects of surgery have changed between the two audits with the vast majority of the surgery performed by the same two vitreoretinal surgeons. We believe our audit results confirm the potentially toxic effect of ICG on the retina. As discussed in our initial paper the concentration of ICG may be the main factor influencing functional outcome. This appears to be supported by the recent paper by Jaycock *et al*, as they found no evidence of toxicity with a 0.05% solution of ICG while our adverse outcomes followed use of a 0.5% solution. In light of our audit finding we will continue to use membrane blue to assist in ILM peeling but ICG does appear to be safe in low concentrations.

Table 1 Anatomical and functional outcomes following macular hole surgery

	Initial audit		*Reaudit*	
	All cases (n = *123*)	*ICG cases only* (n = *21*)	*All cases* (n = *27*)	*Membrane blue cases only* (n = *17*)
Anatomical success (%)	85	92	81	100
Visual improvement (Snellen-converted logMAR)	0.48	0.03	0.4	0.41

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Sir,

Report of a novel lobular chorioretinal dystrophy

Atrophy involving the choroid and retina is a consequence of infective, inflammatory, or degenerative