not suture related, was superficial and amenable to routine microbiological work up. However, *Nocardia* may stain irregularly with Gram stain and may fragment into coccobacillary forms leading to a misdiagnosis of *Corynebacterium* spp. Therefore, acid-fast staining should be a part of the protocol under such circumstances. Owing to varying antibiotic susceptibility, it is essential to determine the drug susceptibility of each isolate.[5] The infection resolved on treatment with the antibiotic to which the organism was susceptible. However, under such circumstances complete discontinuation of topical steroids, as in our case, may result in graft failure. So, probably a delicate balance is required between continuing low-dose topical steroids and intensive antibiotics to achieve not only the resolution of the infection, but also to prevent graft failure.

Nocardia should be considered as a possible causative agent in a graft infiltrate, especially under local immunosuppression. Resolution of infection can be achieved by early microbiologic workup and initiation of therapy.

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Sir,

Closure of macular hole following vitrectomy for diabetic tractional macular detachment

Macular holes in proliferative diabetic retinopathy (PDR) may present an unusual surgical challenge when associated with a macular tractional retinal detachment (TRD).[1] We successfully closed such a hole, presumed secondary to TRD, using standard pars plana vitrectomy and membrane peeling, without further intervention specifically addressing the macular hole.

Case report

A 45-year-old insulin-dependent diabetic man was referred with a recent drop in vision of the left eye; best-corrected visual acuity (BCVA) was 6/6 OD and 6/36 OS. Anterior segment examination was unremarkable except for bilateral pseudophakia. Fundus examination revealed postscatter-photocoagulation PDR OU and an extramacular TRD OS. Both eyes underwent additional panretinal photocoagulation. After 2 months, the new vessels regressed; but increasing vitreoretinal traction caused cystoid macular oedema OS. Vitrectomy was advised; but the patient returned only after 4 months, when BCVA had decreased to 2/60 due to macular TRD with a full-thickness macular hole (Figure 1(a)). Optical coherence tomography confirmed the macular hole, and revealed a vitreous membrane bridging the hole (Figure 1(b)). Pars plana vitrectomy, removal of fibrous proliferation, air–fluid exchange, and silicone oil injection were performed without complication. No attempt was made to peel the internal limiting membrane (ILM). After 1 month, left eye had an attached retina and closed macular hole (Figure 1(c)), confirmed by OCT (Figure 1(d)). Silicone oil was removed 6 months later. At 1 year postvitrectomy, significant posterior capsular opacification was noted. After YAG capsulotomy, BCVA improved to 6/36.

Comment

Macular holes in PDR are caused by fibrovascular traction at the edges of a fovea weakened by oedema and

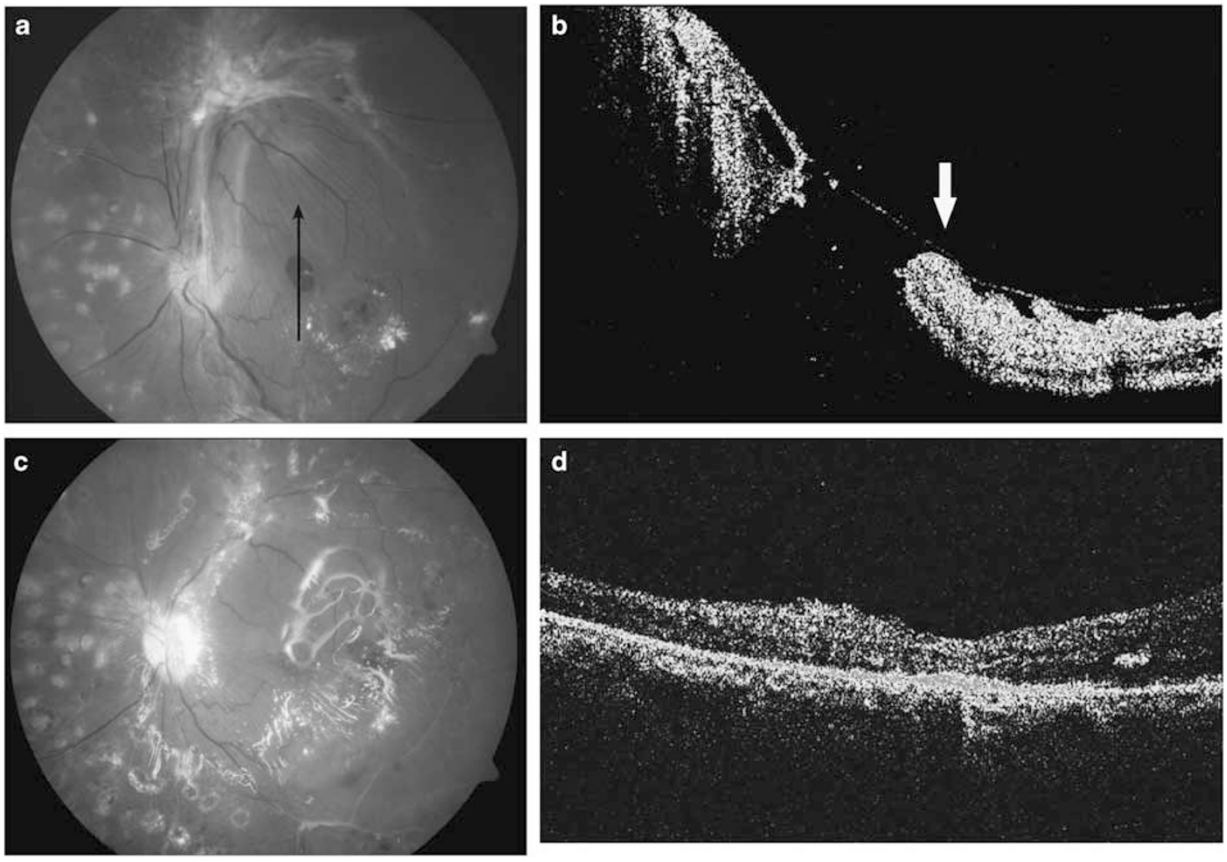

Figure 1 (a) Fundus photograph of the left eye demonstrating a tractional macular detachment and a vertically-oriented oval full-thickness macular hole. The arrow shows the direction of optical coherence tomography (OCT) scan. (b) OCT demonstrating full-thickness macular hole with vitreous bridging the thickened edges of the hole. Only the superior edge (arrow) is everted. The base of the hole is not seen due to retinal elevation in excess of 2 mm. (c) Fundus photograph of the left eye 1 month after vitrectomy demonstrating an attached macula, closure of the macular hole, folds in the internal limiting membrane, residual glial tissue along the superior arcade, and light reflections from the silicon oil interface. (d) OCT of the left fovea 1 month after vitrectomy demonstrating a closed macular hole, minimal central (228 μm) and nasal macular thickening and subfoveal pigment epithelial hypertrophy.

ischaemia. Particularly when eccentric, they may also result from a ruptured cyst of macular oedema, or from sudden dissection of the posterior hyaloid by a premacular haemorrhage. Idiopathic macular holes also occur coincidently with PDR.[1–3] Holes in a TRD typically change its profile from concave to convex.[4] The concave profile of TRD and the vertically-oriented macular hole in this patient pointed to fibrovascular traction, possibly exacerbated by preceding cystic changes. The preoperative OCT demonstrated a tractional membrane bridging the hole, everting its superior edge, and flattening the inferior edge. This was in contrast to the 'pregnant drawbridge' OCT configuration characteristic of idiopathic macular holes.[5] Although we could not rule out inadvertent removal of ILM during membrane removal, it was not specifically attempted.

Though macular hole-closure rates in PDR are comparable to those in nondiabetic eyes, visual outcomes are compromised by coexisting macular oedema, ischaemia, and TRD.[2,4] The visual recovery in this case could also have been influenced by the surgical delay, and toxicity of silicone oil to the retinal pigment pithelium/photoreceptors.[6] We report a good surgical and visual outcome in a rare case of tractional macular hole in diabetic TRD.

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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