Sir,
**Recurrent pterygium as a cause of wound dehiscence
in a corneal graft**

Pterygia recur with varying incidence depending on the technique used for excision. We report a case of a recurrent pterygium causing wound dehiscence in a corneal graft after a combined penetrating keratoplasty and pterygium excision.

Case report

A 56-year-old man presented with left pseudophakic bullous keratopathy. He had a history of blunt trauma to the left eye with subsequent complicated extracapsular cataract extraction and anterior chamber intraocular lens implantation. On examination, visual acuity (VA) was hand movement. A nasal pterygium encroaching onto the midperipheral cornea was noted. At surgery, the pterygium was excised prior to trephination using a bare sclera technique. A 7.75 mm diameter corneal graft into a 7.5 mm diameter host was performed and sutured with 10/0 nylon. VA at 6 weeks was 6/36.

At 6 months, VA had reduced to 1/60. There was an aqueous leak at the head of the recurrent pterygium on the graft–host interface (Figures 1 and 2). The pterygium was re-excised with an autoconjunctival graft. The involved graft–host interface was opened, debrided and resutured. Development of intractable glaucoma in the left eye required diode laser cycloablation. A year later, VA was perception of light only. The corneal graft was clear and IOP was 23 mmHg.

Comment

Several techniques are available for the excision of pterygium. The bare sclera technique is simple and quick to perform but has a recurrence rate as high as 60%.¹ Almost all recurrences occur within 1 year, with 50% within 4 months.² An autoconjunctival graft significantly decreases the risk to as low as 2%¹ and this would have been the preferred primary option in this case.

It is postulated that epithelial downgrowth from the recurrent pterygium was the mechanism for the corneal graft wound dehiscence. Unfortunately, a histopathological specimen was not available to confirm this hypothesis. The development of a Seidel-positive fistula at the head of the pterygium suggested the origin of the cells was from the conjunctival epithelium of the pterygium. The development of glaucoma was likely multifactorial including angle closure from peripheral anterior synechiae.

Figure 1 Recurrent pterygium growing over corneal graft–host interface.

Figure 2 Aqueous leak (arrow) at the corneal graft–host interface demonstrated by positive Seidel test.

This case report presents a rare complication of recurrent pterygium causing corneal graft wound dehiscence. Pterygium excision with an autoconjunctival graft could be considered as a separate procedure prior to corneal grafting, allowing time for healing and follow-up of any sign of pterygium recurrence. This principle can also be applied to the management of pterygia prior to other types of intraocular surgery including cataract extraction.

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The authors do not have any conflict of
commercial interest related to the work.

Eye (2006) **20**, 611–612. doi:10.1038/sj.eye.6701928;
published online 3 June 2005

Sir,
**The Indian case of live worm in Diffuse Unilateral
Subacute Neuroretinitis**

There have been many reports of DUSN,¹ but none with
a documented worm from India. We report a case of
DUSN with live worm (size 1500–2000 μ m) from South
India, which was killed by a single shot of laser
photocoagulation with subsequent visual improvement.

Case report

A 40-year-old, apparently healthy man from Kerala state,
where filarial *Burgia malayi* was endemic, presented
with right visual loss of 4 days duration. There was no
history of contact with live animal. Right VA was 2/60
with RAPD and left was 6/6; otherwise there were
unremarkable anterior segment findings. Right fundus
revealed mild vitritis with a glistening white worm
found in the temporal periphery among the diffuse
subretinal tracts, Figure 1a. Full blood counts and stool
examination were normal.

When the patient was scheduled for 532 nm Argon
Green laser treatment on the same day, the worm had
already migrated to the macula. Immediately, after a
single shot of laser (spot size 200 μ m, duration 150 ms,
and power 160 mW) applied to the advancing end of the
worm, it began to migrate to subfovea and further laser
was abandoned, Figure 1b. A combination of
antihelminthics Albendazole 400 mg stat^{2,3} and
Diethylcarbamazepine (100 mg tds for 21 days)⁸ was

Figure 1 (a) Fundus picture of the right eye showing the worm (arrow) among crisscrossing subretinal tracts in the temporal midperiphery at presentation. (b) The same eye showing the migrated worm in the macular region with the arrow indicating the area where the laser was given. (c) The same eye showing the nonmotile dead worm (arrow) with surrounding serous detachment and new crops of evanescent white inflammatory lesions. (d) The same eye showing the resolved inflammatory lesions, 3 weeks postlaser.

started. On the next day, the worm was immobile with
surrounding neuroretinitis, Figure 1c. The patient was
put on oral corticosteroid 40 mg tapering over 21 days.

After 3 weeks, his right vision improved to 6/60. There
were diffuse pigmentary changes in the superior
temporal fundus where the worm was first identified.
However, there was no trace of the worm or of
inflammation, Figure 1d.

Comment

DUSN is a clinical syndrome, caused by a motile, white,
glistening nonsegmented nematode wandering in the
subretinal space. It is characterized by vitritis, papillitis,
and recurrent crops of white evanescent lesions, followed
by severe visual loss, optic atrophy, and diffuse RPE
degeneration.^{4,5} At least two different sizes of the worm
has been recorded: 400–1000 and 1500–2000 μ m. The
precise identity and portal of entry are still a mystery.⁹
The visibility of motile worm is the gold standard in
making the diagnosis of DUSN. However, identifying the
small worm may be difficult and time consuming;
therefore, fundus photography is useful to identify its
location. The oral antihelminthics are ineffective, because
of the impermeability of blood retinal barrier. It is
recommended in those who have severe vitritis and in
whom the worm cannot be easily identified.⁸

In our case, the death of the worm could not be
attributed to oral antihelminthics, because the worm was
found to be dead the next day after the laser. The worm
was identified by its moving advanced end and we