

cured but the epithelial defect persisted. To provide a therapeutic ptosis botulinum toxin was injected into the upper lid. Subsequently the epithelium healed and there has been no recurrence of the defect to date. There is however a residual central stromal scar.

Comment

5-FU is an antimetabolite which interferes with normal cell mitosis. It interferes with the S phase and the G2 phase of the cell cycle which correspond, respectively, with the synthesis of DNA and cellular components required for mitosis.⁸ As corneal epithelial cells are constantly undergoing replication they are particularly susceptible to its toxic effects.

Reduction of dosage has been suggested as a method of reducing complications including potential toxicity to corneal epithelium.⁷ However in this case minimal dosage resulted in toxicity to the cornea.

Diabetes mellitus is associated with impaired healing of corneal epithelium owing to the increased thickness of the basal lamina resulting in impaired organisation of epithelial cells and anchorage to underlying stromal collagen.^{9,10}

This case demonstrates that in patients with compromised corneas even judicious use of very small doses of 5-FU may result in serious corneal

complications. We feel that band keratopathy is a contraindication to subconjunctival injections of 5-FU and the possible delay in healing of epithelial defects in patients with diabetes means that in such cases it should be used with extreme caution and stopped immediately if epithelial defects appear.

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Bilateral streptococcal corneoscleritis complicating β irradiation induced scleral necrosis

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Bacterial corneoscleritis may complicate scleral necrosis induced by β irradiation following pterygium removal.¹² Previous cases have been unilateral. We report a case of severe bilateral corneoscleritis caused by *Streptococcus pneumoniae*.

Case report

A 66-year-old man underwent surgical excision of bilateral pterygia and a course of 2200 cGy of β irradiation in three divided doses to each eye. He remained asymptomatic for 15 years until he complained of irritation in both eyes attributed to calcific plaques at the base of deep scleral ulcers. These were removed and covered by conjunctival flaps. He was given chloramphenicol drops four times daily to both eyes. One week later the patient reattended with a right panophthalmitis and spontaneous perforation (Fig 1), and a large deep medial scleral abscess in

the left eye (Figs 2 and 3). He was referred to our institution and immediately underwent debridement of necrotic sclera of the right eye and injection of intravitreal vancomycin and gentamicin. A complete vitrectomy proved technically impossible, owing to corneal and lens opacification. Urgent Gram staining of sclera from both eyes showed Gram positive diplococci, and cultures ultimately grew *Streptococcus pneumoniae*.

He began intravenous and intensive topical benzylpenicillin and the infection slowly improved. Ultimately a large eccentric right penetrating keratoplasty was necessary to facilitate resolution of the corneal abscess and to reform the anterior chamber following spontaneous perforation (Fig 4). The scleral abscess in the left eye improved without surgical intervention. Visual acuity is perception of light in the right eye due to a dense lens opacity and 6/5 in the left.

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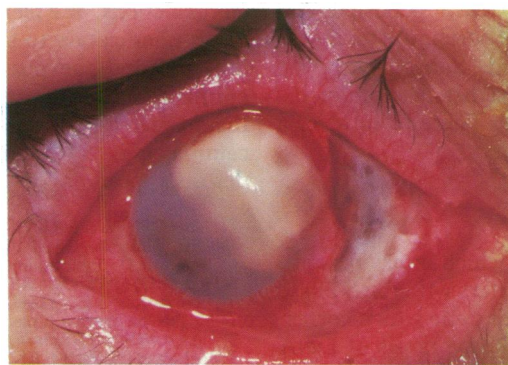


Figure 1 Right eye – panophthalmitis and spontaneous scleral perforation.

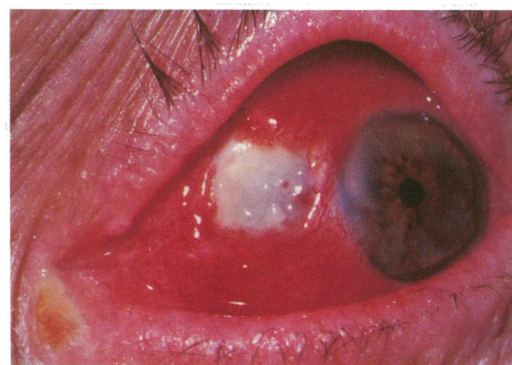


Figure 3 Deep scleral abscess – left eye.



Figure 2 Appearance of patient 1 week after removal of calcific plaques.

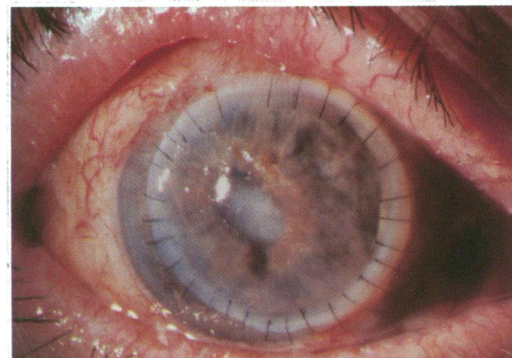


Figure 4 Large eccentric right penetrating keratoplasty performed to remove abscess and reform anterior chamber.

Comment

We have seen 12 cases of infective corneoscleritis after pterygium excision and β irradiation over the past 8 years.³ This is the first case of bilateral involvement. Infection is often precipitated by removal of calcific plaques at the base of scleral ulcers. Usually *Pseudomonas* is responsible though there is one previous report of *Streptococcus pneumoniae* as a cause.²

Visual outcome is poor unless early aggressive measures are adopted in treatment.³ The implications of bilateral infection are serious. Cross infection between the two eyes remains a possibility in this case. We suggest therefore that if plaques are to be removed they should be done so under full aseptic techniques and not on both eyes simultaneously since plaques and ulcer beds are a potential reservoir and nidus for infection. They frequently grow pathogens on culture³ and sterility (by debridement, culture, and disinfection) should be confirmed before ulcers are covered by lamellar grafts or conjunctiva. We routinely use half strength povidone iodine after plaque removal as disinfectant and then maintain patients on chloramphenicol drops for 2 weeks before covering the bare ulcer bed.

A variety of organisms such as *Candida*, *Scedosporium inflatum*, and *Petriellidum boydii* may also be implicated.³ Classically a long

latency of 10–20 years between radiotherapy and infection occurs. Ocular morbidity is severe and prolonged and two eyes have required enucleation. The condition may remain undiagnosed for some time or masquerade as posterior scleritis with serous retinal detachment or as orbital cellulitis.

The anterior sclera is more susceptible to the effect of radiotherapy than posterior sclera.¹ We see late complications from doses as low as 1800–2000 cGy. Our experience is similar to other authors⁴ who report that the risk of endophthalmitis is greatest with severe scleral thinning but scleromalacia is not significantly correlated to dose of radiation. With the risk of scleral necrosis of 4.5%⁴ and a lifelong risk of endophthalmitis following radiotherapy we no longer recommend this treatment. We remove more than 100 pterygia per year in our institution and conjunctival autografting is our treatment of choice to reduce recurrences.

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