

Late Corneal Thinning and Keratitis following Mitomycin-C use in Pterygium Surgery

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ترقق وتهاب القرنية المتأخر بعد استعمال دواء
الميتومايسين (سي) في جراحة الظفرة

رضا شينوي، الكسندر بيزليزيويكس و بدر البرواني

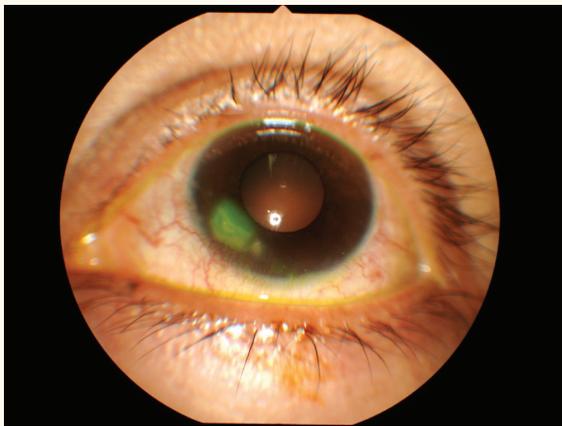


Figure 1a: Left eye – corneal ulcer 2mm from the nasal limbus.

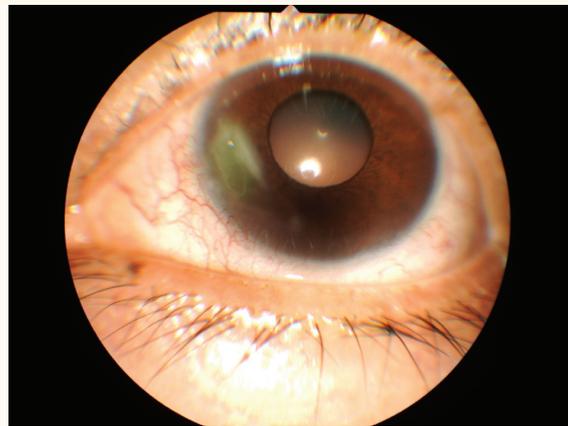


Figure 1b: Left eye – corneal scar temporal to ulcer.

A 28 YEAR-OLD WOMAN PRESENTED TO the Ophthalmology Clinic at the Armed Forces Hospital, Muscat, Oman, with severe pain, redness, photophobia and decrease in visual acuity of her left eye of 1 day's duration. She gave a history of a pterygium excision with intraoperative topical mitomycin-C application (0.1 mg/ml for 5 minutes) in the same eye two years before. There was no other systemic or local disease. She underwent a detailed ophthalmic and systemic evaluation and laboratory examination. The best corrected visual acuity in her right eye was -0.25sph/-0.50cyl /90 = 6/6 and in the left eye -0.25sph/-1cyl 140 = 6/12. Her left eye showed conjunctival and ciliary congestion. There was a linear ulcer on the cornea about 2 mm from the

nasal limbus with a scar immediately temporal to it. There was no surrounding infiltration, but the nasal 4–5mm of cornea was thin between 6 and 11 o'clock [Figures 1a and 1b] The adjacent sclera was normal. The intraocular pressure and fundus of both the eyes was normal. Scrapings and swabs from the ulcer base were sent for microbiological evaluation. The left eye was treated with prophylactic antibiotic drops and artificial tears. The ulcer responded to treatment in a period of 4 weeks, leaving a thin nasal cornea with peripheral limbal vascularisation [Figure 2].

Pterygium is an epithelial hyperplasia accompanied by a fibrovascular growth originating at the corneo-conjunctival junction, from where the modified limbic cells migrate and surpass

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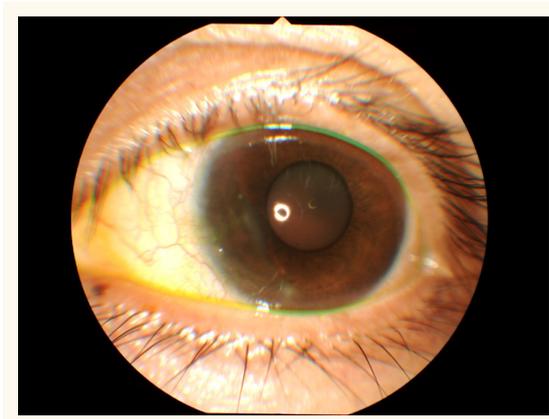


Figure 2: Left eye – 4 weeks after treatment showing no corneal stain/ healed ulcer.

the cornea. It is an active process associated with cell growth, remodelling of the connective tissue, angiogenesis and inflammation, triggered by ultraviolet irradiation. Different investigators have recently emphasised the importance of the limbus and its stem cells in the pathogenesis of the pterygium.^{1,2} Surgery as a treatment for pterygium has been known since 1,000 B.C and has been directed towards excision, prevention of recurrence, and restoration of ocular surface integrity. The recurrence rate has, in the past, been estimated as high as 30–70%, despite adoption of different evolutionary techniques.^{1,2,3} Although irradiation therapy and antimetabolite use have diminished the recurrence rate to 5–12%, complications like secondary glaucoma, cataracts, uveitis, corneal perforation, scleritis, scleral necrosis and secondary endophthalmitis can occur.¹⁻⁵ Mitomycin, isolated from *Streptomyces caespitosus*, is an extremely toxic, non cell-specific alkylating antibiotic with antineoplastic properties that selectively inhibits the synthesis of deoxyribonucleic acid (DNA),

ribonucleic acid (RNA) and protein, and prevents cellular division.¹⁻³ The DNA damage mimics that of ionising radiation and has a radiomimetic effect. The drug has been used in the eye since 1963 for its anti-fibroblast proliferating action, in pterygium and glaucoma surgeries and recently in refractive procedures, both for the management of certain complications and to improve vision after surgery.¹⁻⁵ However, in susceptible individuals, it causes keratocyte depletion leading to delayed wound healing, irrespective of the technique of application.¹⁻⁵ Complications occur either immediately after surgery, or up to 20 years later, and are related to drug concentration, contact time and predisposing ocular factors, highlighting the fact that such patients need long term follow-up.^{3,4,5}

References

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