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Letters to the Editor

Maxitrol-induced corneal melts after routine cataract surgery in rheumatology patients

Advances in cataract surgery techniques and pathways have now resulted in a predominantly day-case outpatient service. When surgery is uneventful, patients are often not reviewed within the next week but rather are routinely followed up at 1 month.[1] Combination drops containing a steroid and antibiotic are often prescribed for the month following the surgery. In our department, the standard treatment is Maxitrol QID (dexamethasone, polymyxin B sulphate, neomycin); chosen for convenience, cost and compliance. This is a successful management plan in the vast majority of healthy patients.

However, patients with rheumatological conditions are at risk of dry eyes. Corneal and cataract surgery, topical medications, associated inflammation and/or infection can lead to corneal ulceration, melting, thinning and perforation.[2,3] These cases often require systemic immunosuppression with steroids and surgical intervention, such as amniotic membrane or corneal grafting. We wish to report three recent cases of central corneal melting following routine cataract surgery with standard postoperative aftercare to raise awareness about this patient group.

Case 1: A 74-year-old female with Sjogren's syndrome secondary to rheumatoid arthritis underwent routine right phaco-emulsification cataract surgery. Preoperative treatment included celluvisc 1% prn. Postoperative treatment was topical Maxitrol (Alcon, Fort Worth, TX, USA) QID as standard. Six weeks later, she presented with a painful, photophobic right eye. The vision was hand movements (6/24 preoperative). She had been using her Maxitrol as directed. Examination revealed a central corneal epithelial defect with 40% stromal thinning. The diagnosis of central corneal melt was made, the Maxitrol was discontinued, and treatment of 60 mg prednisolone, minims chloramphenicol and hourly lubrication was commenced. The ocular inflammation resolved by day 12 with central corneal scarring, and no improvement in visual acuity.

Case 2: A 65-year-old male with primary Sjogren's syndrome underwent routine left phaco-emulsification cataract surgery. Preoperatively he was using viscotears prn. The surgery was uncomplicated. Postoperative treatment was Maxitrol QID as standard. He presented 2 weeks postoperative with a red, irritable, watery eye. Vision was reduced to 3/60 (6/36 preoperative). There was a large central corneal epithelial defect with approximately 90% stromal thinning. A central corneal melt was diagnosed,

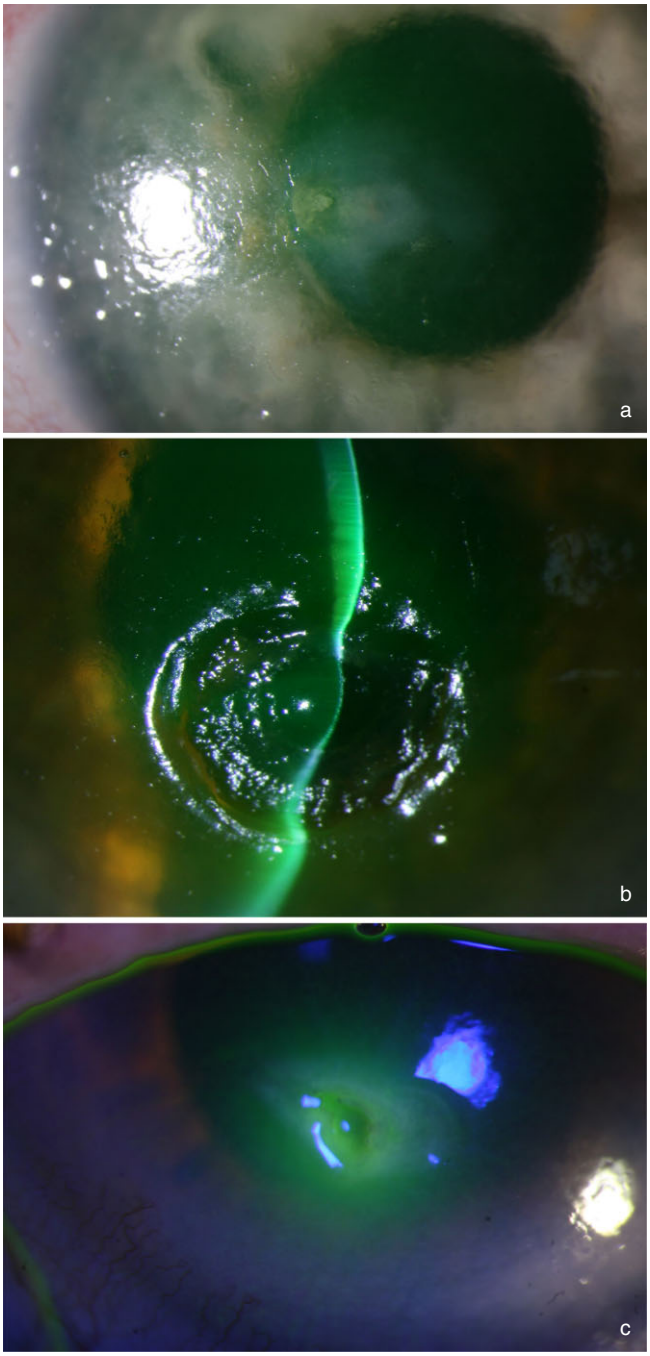

Figure 1. Clinical appearance at time of presentation for three rheumatology patients, each showing central corneal melting following routine use of Maxitrol following standard cataract surgery.

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the Maxitrol was discontinued and treatment started with 60 mg prednisolone, minims chloramphenicol and 2 hourly lubricants. Unfortunately, the cornea perforated and required cyanoacrylate gluing, and a subsequent penetrating keratoplasty.

Case 3: An 82-year-old female with dry eyes and bilateral hip/knee replacements due to osteoarthritis underwent routine left phaco-emulsification cataract surgery. Postoperatively, she used Maxitrol QID as instructed. At 1 month review, vision was 6/60 with a central area of 80% corneal melting. She was treated with oral prednisolone 40 mg, minims chloramphenicol and a bandage contact lens was inserted. She underwent an emergency amniotic membrane graft 3 days later.

Discussion: Corneal perforation has been previously reported in patients with severe dry eyes secondary to rheumatoid arthritis and Sjogren's syndrome following routine cataract surgery.[2–5] Previous literature has suggested the topical steroid was responsible for abnormal wound healing by reducing collagen synthesis.[4] However, it should be noted that the corneal melting process occurred centrally in our patients, rather than at the site of the clear corneal incisions (Fig. 1).

We believe the neomycin component of Maxitrol was responsible for altering the fine balance in the vulnerable ocular surface of these three rheumatology patients. Topical neomycin has long been implicated as the aminoglycoside which has the most significant deleterious effect on epithelial cell function, causing a dose dependent cytotoxicity and cell death.[6] Frequent exposure to neomycin damages the corneal epithelial cells, leaving the stroma exposed to recruitment of inflammatory cytokines, matrix metalloproteinase and collagenase activity and subsequent corneal melting.[6] We acknowledge there were existing comorbid factors (mild dry eyes, ocular surgery, quiescent rheumatological disease) in these patients, and so it is difficult to attribute the corneal melt solely to topical medications. However, it would appear the use of Maxitrol tilted the exquisitely sensitive balance towards pathology.

We highlight that patients with dry eyes and rheumatological diseases are at a higher risk of iatrogenic ocular surface problems following routine cataract surgery.[2–5] These conditions should be identified by the preoperative assessments and highlighted to the operating surgeon as part of the surgical checklist.[1] This is particularly relevant if general cataract lists are centralized or pooled service lists, where the operating surgeon may not be the ophthalmologist who listed the patient. Consideration can then be given to more appropriate, or preservative-free, postoperative medications. We would recommend the avoidance of neomycin-based products in this context.

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Screening phenotypically normal Caucasian Australians for the lysyl oxidase-like 1 gene

Recent work has demonstrated that single-nucleotide polymorphisms within the lysyl oxidase-like 1 (*LOXL1*) gene located on the long arm of chromosome 15 are more common among exfoliation syndrome (XFS) patients.[1] In particular, two coding region single-nucleotide polymorphisms (rs1048661 and rs3825942) were strongly associated with XFS when both of their locations possessed a guanine residue (i.e 'GG' haplotype).[1] When this occurs on both copies of an individual's chromosome 15, the combined GG/GG diplotype is found in approximately 60% of XFS patients and 25% of the general population in both Iceland[1] and Australia.[2] The as yet incomplete understanding of the genetic risk profiling for XFS is highlighted by the presence of different risk alleles at these same *LOXL1* residues in Japanese and South African populations.[3,4]

Given that a genetic test has now been offered in Iceland to screen for XFS, we designed this study to investigate the possible outcome of screening a group of Caucasian Australians for the high-risk GG/GG diplotype who had been found to be phenotypically without any suspicion of glaucoma.

The recruitment of study subjects conformed to the tenets of the Declaration of Helsinki, following ethical approval from the Human Research and Ethics Committee of the Flinders Medical Centre. Our sample was derived from volunteers recruited from local retirement villages, independent living centres and Rotary clubs. Participants were screened for glaucoma in the community and

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