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Case Report

Severe Corneal Melt Post Trans-Scleral Cyclodiode in a Case of Neovascular Glaucoma Secondary to Coats Disease: A Case Report

Jackson Chee Chea Lee[a] Geoffrey Zhi Peng Chan[a, b, c] Humma Shahid[b]

[a]Lions Eye Institute, Perth, WA, Australia; [b]Centre for Ophthalmology and Visual Science, The University of Western Australia, Perth, WA, Australia; [c]Department of Ophthalmology, Addenbrooke's Hospital, Cambridge University Hospital, Cambridge, UK

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Abstract
A novel case of neurotrophic keratitis and severe corneal melt requiring surgical management is presented 1 month following trans-scleral cyclodiode for Coats disease and neovascular glaucoma. Risk factors contributing to the complication include previous extracapsular cataract surgery, perioperative use of topical non-steroidal anti-inflammatories and dexamethasone/neomycin, as well as other topical drops containing preservatives such as benzalkonium chloride. Meticulous consideration of preoptimization of the ocular surface and rationalization of perioperative eye drop regimes is discussed.

Introduction

Coats disease is a rare congenital idiopathic telangiectasia characterized by lipid exudation and retinal ischaemia with an estimated incidence of 0.09 per 100,000 [1, 2]. Patients may present with advanced unilateral disease, and enucleation may be required in cases with neovascular glaucoma [3].

Studies have demonstrated the use of trans-scleral cyclodiode as an effective and relatively safe technique for management of refractory glaucoma in Coats disease, which has the

Correspondence to:
Jackson Chee Chea Lee, jackson_lcc@live.com

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benefit of tissue preservation [4]. However, rare complications may occur and neurotrophic keratitis post-cyclodiode is reported [5].

We present the first case of severe corneal melt and neurotrophic keratitis precipitated by cyclodiode in the context of treatment for Coats disease and neovascular glaucoma. Significant risk factors for the development of corneal melt in combination with cyclodiode, including the use of topical non-steroidal anti-inflammatories (NSAID) and dexamethasone/neomycin combination therapy are highlighted. Our findings reiterate the importance of assessing preoperative risk factors for developing corneal complications in patients undergoing cyclodiode, with the goal of reducing potentiating factors for the development of corneal disease. The CARE Checklist has been completed by the authors for this case report, attached as online supplementary material (for all online suppl. material, see https://doi.org/10.1159/000534104).

Case Presentation

A 59-year-old Southeast Asian man was referred for management of neovascular glaucoma in a case of unilateral refractory left eye Coats disease. His ophthalmic history included previous left extracapsular cataract surgery (ECCE) for phacolytic glaucoma 10 years prior with pressure normalizing to 14 mm Hg following surgery. This had increased to 53 in the month leading up to referral and was attributed to the development of neovascularization of his iris and angles. He had unremarkable past medical history.

Topical treatment for the left eye included Saflutan 15 μg/mL (Taflotan) nocte, brinzolamide 1%/brimonidine 0.2% (Simbrinza) BD, and Nepafenac 0.1% TDS. He was commenced on oral acetazolamide 250 mg QDS and underwent full left eye panretinal photocoagulation laser to address neovascular disease of the retina. A single intravitreal injection of Eylea was given 1 month prior to presentation.

His best corrected Snellen visual acuity at presentation was 6/4.8 in the right eye and perception of light in the left. Baseline IOP was 7 mm Hg and 48 mm Hg in the right and left eye, respectively. Anterior segment examination of the left eye showed corneal oedema and a large superior corneal incision scar, consistent with previous ECCE (Fig. 1). There was a 3-piece intraocular lens in the capsular bag. Iris rubeosis was present, and gonioscopy confirmed the presence of closed angles and intermittent regions of peripheral anterior synechiae for 360°. Fundus view was compromised due to corneal oedema with prominent retinal exudates. Due to the ongoing pain and the underlying poor visual potential, cyclodiode laser was performed as primary surgical management with an aim to improve comfort.

A left eye trans-scleral cyclodiode was performed under peribulbar anaesthesia. 360° of laser treatment was done, sparing the 3 and 9 o'clock regions of the long ciliary nerve. 1,500–1,900 mW was applied for 1,500 ms, and 10 shots were delivered to each quadrant via cyclodiode G-probe. Dexamethasone/neomycin/polymyxin B (Maxitrol) eye drops QDS were prescribed in addition to his eye drop regime.

One week post-operatively, the corneal epithelium of the left eye remained intact and IOP was reduced to 38 mm Hg with moderate anterior chamber inflammation. At the month post-operative review, the patient was comfortable with a reduced IOP of 14 mm Hg in the treated eye. However, the patient had developed a painless, large central corneal defect with significant 80% stromal thinning (Fig. 2). Corneal sensitivity was significantly reduced. Given the appearance of the ulcer, absence of pain, and subsequent negative corneal scrape result, a diagnosis of neurotrophic keratitis with severe corneal melt precipitated by cyclodiode and potentiated by eye drop toxicity was made.

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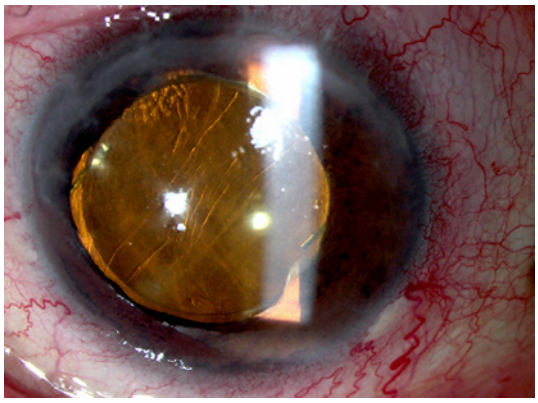

Fig. 1. Anterior segment photograph of the left eye at initial presentation. A large superior corneal wound was present from previous extracapsular cataract extraction. Mild corneal oedema and 360° of peripheral anterior synechiae are visible.

Nepafenac, Maxitrol, Saflutan, and Simbrinza eye drops and oral acetazolamide were stopped and the patient was continued on dexamethasone PF 0.1% BD and preservative-free (PF) latanoprost 50 μg/mL (Monopost) nocte (Monopost was continued in lieu of Saflutan due to its ready availability at the patient's local pharmacy, with both prostaglandins available as preservative free formulations). Two hourly dose of Thealoz Duo (trehalose 3%/sodium hyaluronate 0.15%), topical levofloxacin QDS, and oral doxycycline 100 mg BD were commenced in view of the severe corneal epithelial defect. A Botox ptosis was initially performed. The corneal epithelial defect failed to heal over a 2-week period, so a lateral tarsorrhaphy with multi-layered amniotic membrane transplant (Omnigen) was arranged in theatre to facilitate re-epithelization of the ulcer.

Our patient responded well to temporary lateral tarsorrhaphy with Omnigen transplant, with complete resolution of the neurotrophic defect. A review at 3 months post-cyclodiode revealed a comfortable eye with a stable vision of PL and IOP of 9 mm Hg, and no ongoing corneal defect (Fig. 3).

Discussion

The presence of neovascular glaucoma in Coats disease is a poor prognostic sign [4]. Trans-scleral cyclodiode remains a mainstay of treatment for such cases of refractory glaucoma. Neurotrophic corneal defect is a rare complication of cyclodiode [6]. Mahmood and colleagues [7] reported only 1 case of neurotrophic ulcer among 102 eyes undergoing cyclodiode laser ablation, and this was believed to occur due to inadvertent application of laser to the ciliary nerves.

Johnson [5] explored risk factors for developing neurotrophic corneal defects after ciliary diode, reporting diabetes and chronic eye inflammation as predisposing factors to compromised corneal function. In our patient, multiple risk factors for the development of severe corneal disease were present including the initial 360° application of trans-scleral cyclodiode and the perioperative use of non-PF eye drop formulations containing benzalkonium chloride. Chronic intraocular inflammation in the context of advanced Coats disease as well as additional post-operative inflammation from the cyclodiode procedure may have also contributed to the development of neurotrophic keratitis [8]. Corneal hypoesthesia secondary to previous large corneal incisional ECCE may have also resulted in a predisposition to the development of neurotrophic ulcer, with corneal wounds greater than 5 mm reported to cause corneal hypoesthesia not returning to preoperative baseline states [9].

In addition, the use of topical neomycin, the aminoglycoside component of Maxitrol is well known for its dose-dependent cytotoxicity on epithelial cell function [10]. Independently,

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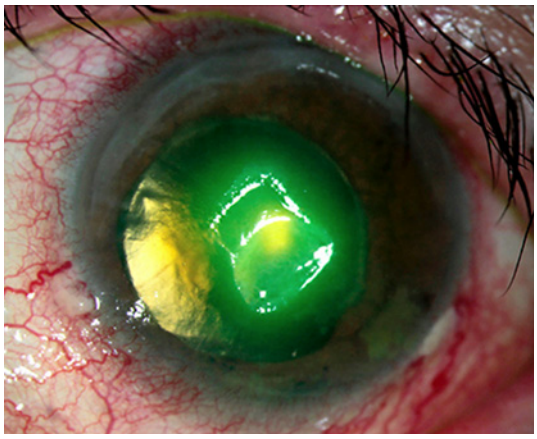

Fig. 2. Anterior segment photograph of the left eye 1 month after trans-scleral cyclodiode. Significant post-operative inflammation and a central corneal melt are demonstrated with fluorescein highlighting a deep stromal component measuring 4 × 5 mm.

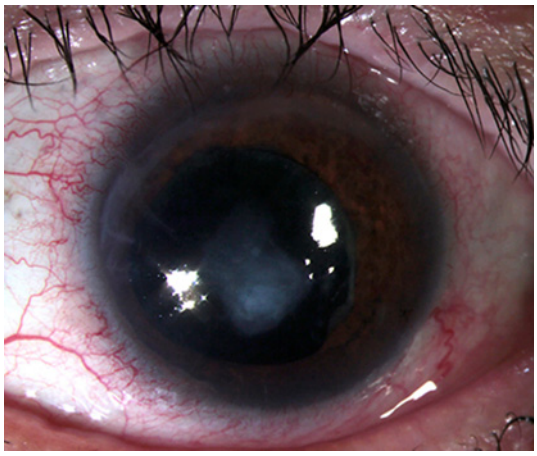

Fig. 3. Left eye 3 months after the initial cyclodiode procedure. A central corneal stromal scar is evident with complete resolution of the previously visualized epithelial defect.

the use of topically applied NSAID has also been implicated, if not causative in corneal melting post-routine cataract surgery [11].

Compared to cyclodiode, MicroPulse trans-scleral laser therapy has a better safety profile and may be the preferred option for intraocular pressure lowering in higher risk eyes [12], with only a few case reports in the literature of neurotrophic keratitis following this alternative treatment [13, 14]. Alternatively, 180° trans-scleral cyclodiode may be considered in higher risk eyes as it has been shown to provide reasonable intraocular pressure control and a low incidence of adverse effects in refractory glaucoma of any cause [15]. We postulate that the combination of non-PF eye drops, higher dosage of cyclodiode, topical NSAID, and dexamethasone/neomycin combination therapy was a powerful potentiator for the development of severe corneal epithelial toxicity, and avoidance of these agents may have prevented the development of severe corneal melt in our patient undergoing cyclodiode laser.

The treatment of the corneal melt and neurotrophic keratitis in our patient posed numerous clinical difficulties. Conservative therapies such as the commencement of PF ocular lubricants, cessation of cornea-toxic medications, and conversion to PF eye drop regimes did improve the status of the ocular surface. Serum eye drops, punctal plugs, Botox ptosis, lateral tarsorrhaphy, and amniotic membrane transplant are well established in the management of neurotrophic ulcers. In this instance, combination of multiple modalities allowed successful resolution of our patient's persistent corneal epithelial defect [16].

Statement of Ethics

This case report conforms to the principles of the Declaration of Helsinki 2013. The subjects provided written informed consent to publish the case and images. Patient identifiers were avoided because of privacy concerns. Ethical approval is not required for this study in accordance with local or national guidelines.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

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Author Contributions

J.L. wrote the case report and performed a literature review of the write-up of the case report. H.S. and G.C. supervised the case report writing and submission. The patient received direct clinical care for H.S. and G.C. J.L., H.S., and G.C. collectively reviewed the manuscript before submission.

Data Availability Statement

Data sharing is not applicable to this article, as no datasets were generated or analysed. Further enquiries can be directed to the corresponding author.

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