CASE REPORT

The Nd:YAG laser as first-line treatment for fibrin pupillary-block glaucoma following uncomplicated cataract surgery

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Abstract

A 43-year-old lady presented with painful, loss of vision in the right eye 2 weeks after cataract surgery. This is on a background of previous central retinal vein occlusion in the right eye and bilateral diabetic retinopathy. Her past medical history included mixed connective tissue disease for which she is on systemic immunosuppressants. Ocular examination revealed significant corneal oedema, a shallow anterior chamber with an intraocular pressure of 45 mmHg. Intraocular pressure lowering medications were administered and repeat examination identified a fine fibrinous membrane attached to the pupillary margin with a substantial third space that had formed between the posterior edge of the membrane and the anterior lens surface. The fibrin membrane was removed with Nd:YAG laser and intraocular pressure reduced to 12 mmHg. Post-procedural management included hourly dexamethasone 0.1% eye drops for 1 week and cyclopentolate 1%. There was no recurrence in the fibrin membrane.

INTRODUCTION

Fibrin pupillary-block glaucoma describes raised intraocular pressure due to an inflammatory membrane forming completely across the pupillary margin typically after intraocular surgery [1, 2]. This rare phenomenon has been reported after routine cataract surgery and pars plana vitrectomy [1–4].

The exact aetiology of this condition is unknown, however, there are many postulated theories. One theory most relevant to modern cataract and vitrectomy surgery is the predilection for the blood-aqueous barrier to breakdown in patients with concomitant autoimmune disease, e.g. systemic lupus erythematosus, resulting in a heightened post-operative inflammatory reaction [5–7]. We report a case of fibrin pupillary-block glaucoma in a patient with mixed connective tissue disease treated successfully with primary Nd:YAG laser.

CASE PRESENTATION

We describe a case of a 43-year-old woman who presented with sudden loss of vision and severe pain in the right eye 2 weeks after undergoing uncomplicated cataract surgery in this eye. Previous ophthalmic history included a central retinal vein occlusion in 2013 with cystoid macular oedema treated with 15 Ranibizumab intravitreal injections in the right eye and bilateral background diabetic retinopathy. At presentation visual acuity in the right eye was hand movements and −0.02 LogMAR in the left eye. The intraocular pressure was 45 mmHg in the right eye with corneal oedema and a shallow anterior chamber centrally. There was no fundal view of the right eye at the time of presentation. A diagnosis of acute angle closure glaucoma was made and IOP lowering medication commenced: this included intravenous Acetazolamide 500 mg, Guttae. Dorzolamide/Timolol, Guttae.
Apraclonidine 1%, Guttae Dexamethasone 0.1% and a 20% Mannitol intravenous infusion of 250 ml over 30 min. Immediate examination after treatment revealed the corneal oedema had largely cleared and the anterior chamber had deepened. The intraocular pressure had lowered to 28 mmHg. Repeat examination 30 min later revealed the IOP had increased to 43 mmHg and the anterior chamber was shallow again. Furthermore, slit-lamp examination revealed a fine fibrinous membrane was attached to the pupillary margin with a substantial space having formed between the posterior edge of the membrane and the anterior lens surface.

Further topical intraocular pressure lowering medication was administrated and the eye dilated. The decision was made to use the Nd:YAG laser to remove the fibrinous membrane at the pupillary margin using a similar technique when performing a routine posterior capsulotomy.

A total of 167 single burst energy shots using a low energy range of 1.6-1.9 mW was applied to the edge of the fibrinous membrane through an Abraham capsulotomy lens with the coupling agent G. Clinitas 0.2%. Post-procedure G. Apraclonidine 1% and G. Dexamethasone 0.1% were applied to the right eye stat. and she was advised to continue the G. Dexamethasone hourly for 1 week in addition to G. Ketorolac 4 hourly and G. Cyclopentolate 1% 12 hourly to the right eye. At 15 min post-procedure the intraocular pressure in the right eye was 12 mmHg.

At day follow-up, the uncorrected visual acuity had improved to 0.94 LogMAR in the right eye and intraocular pressure was 15 mmHg, the anterior chamber remained deep and the cornea was clear. Fundus examination revealed collateral vessels at the optic disc and some small dot haemorrhages at the macular in keeping with diabetic retinopathy and her previous central retinal vein occlusion.

There is no history of previous uveitis, however, she is under the care of rheumatology for mixed connective tissue disease which is antibody positive for ANA, Anti-CCP, Anti-Ro and Anti-Smith antibodies. She is also treated for hypothyroidism and has a diagnosis of viteligo.

**DISCUSSION**

This case report describes a successful method using the Nd:YAG laser to remove the fibrinous pupillary membrane responsible for pupillary-block glaucoma in an individual with significant autoimmune disease.

A key differential to consider in patients presenting post intraocular surgery with raised pressure and shallowing of the anterior chamber is aqueous misdirection. This is a separate and distinct post-operative phenomenon which is often a diagnosis of exclusion. As with aqueous misdirection, the phenomenon of fibrin pupillary-block glaucoma is more common following pars planar vitrectomy surgery [4], but recently has also been documented in uneventful cataract surgery as in our case.

The development of fibrin pupillary-block glaucoma is dependant on the formation of an inflammatory membrane, which completely occludes the pupil. This results in redirection of fluid into a ‘third space’ cavity between the iris and intraocular lens which leads to bowing of the iris and subsequent shallowing of the anterior chamber with resultant pupil block. The similar anatomical morphology to aqueous misdirection glaucoma may hinder early diagnosis.

In previous years there have been many reports on the formation of a fibrin membrane in the anterior chamber following cataract surgery especially in the early days of posterior chamber lens implantation. Largely, these have been transient, self-resolving after 3 weeks. The theory behind the development of these fibrinous membranes is speculated to be an immunologic reaction, whereby breakdown of the blood-aqueous barrier results in an increased propensity to inflammation [6, 7]. Over the past decade, surgery for cataract extraction has become more sophisticated reducing the frequency of reports of the transient fibrin membrane.

A review of the literature finds there have been few reports of fibrin pupillary-block glaucoma post-cataract surgery in the last 20 years. Thus, owing to its infrequent occurrence there is no general consensus regarding treatment. Khor et al. [2] described a case series of four patients with fibrin pupillary-block glaucoma all treated with Nd:YAG laser peripheral iridotomy and in two cases further removal of the fibrin membrane using Nd:YAG of which one of two cases was successful in removing the membrane. Yoshino et al. [1] described the use of intracameral tissue plasminogen activator to fibrinolyse the membrane and release the pupillary block successfully in a single case. This relies on a further procedure in theatre and logistically may prolong the raised IOP if acquiring theatre space is not expedited or available promptly.

In our case, the use of Nd:YAG laser as the primary treatment of fibrin pupillary block glaucoma was successful. This is likely due to the post-procedure management with high-frequency topical steroids in combination with cycloplegia to reduce the inflammatory response following laser therapy already heightened by an existing diagnosis of mixed connective tissue disease.

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None.

**CONFLICT OF INTEREST STATEMENT**

No conflicts of interest.

**FUNDING**

I declare there are no competing interests nor financial declarations.

**ETHICAL APPROVAL**

There was no official requirement for formal ethical approval in line with the local research department protocol for case reports.

**CONSENT**

Written informed consent was obtained from the patient for publication of this case report and publication of any images. A copy of the consent is available on request.

**GUARANTOR**

Selina Khan.

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