Un-doing All that Good Work! Glaucoma After Vitrectomy and Silicone Oil Injection for the Treatment of Complicated Retinal Detachment

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Abstract

Background: Elevated intraocular pressure is a common complication of pars plana vitrectomy and silicone oil infusion.

Methods: We describe a case of pupil block glaucoma consequent to the occlusion by inflammatory fibrinous membrane formation of an inferior peripheral iridectomy after vitreoretinal surgery and endotamponade with silicone oil in a patient with complex rhegmatogenous retinal detachment.

Results: This report highlights the importance of glaucoma management after this type of surgery.

Conclusion: As in the case described here, much emphasis is placed on the successful re-attachment of the retina in these cases. Delayed detection and/or suboptimal monitoring of optic nerve damage can occur. Glaucoma after pars plana vitrectomy with adjuvant silicone oil can lead to serious glaucomatous optic nerve damage.

Keywords: Glaucoma; Pupil-block.; Silicone oil; Pars plana vitrectomy; Iridectomy; Iridotomy; Retinal detachment

Case Report

Case Presentation

A fifty-two year old bilaterally pseudophakic Caucasian gentleman having a retinal detachment secondary to two retinal breaks superotemporally in his right eye underwent twenty-three gauge pars plana vitrectomy (PPV), endophotocoagulation and perfluoropropane (C3F8) gas insertion. He presented five weeks later with a total retinal detachment in the same eye presumed consequent to a retinal break inferotemporally thought to represent temporal extension of the initial retinal tear beyond the margin of the aforementioned retinopexy. Twenty gauge PPV was performed, cryotherapy was applied to the nasal retina. An encircling band was applied and a further twenty gauge PPV performed wherein the dislocated IOL was extracted, PVR membranes were dissected, an inferior retinectomy was created and Neodymium: Yttrium Aluminium Garnet (Nd: YAG) laser peripheral iridotomy was performed and the IOP declined further to 25 mm Hg.

One week later this gentleman's intraocular lens (IOL) was found to have prolapsed into the anterior chamber, a retinal break was noted inferiorly in the area to which cryotherapy had been previously applied and proliferative vitreoretinopathy (PVR) was obvious at the macula and nasal retina. An encircling band was applied and a further twenty gauge PPV performed wherein the dislocated IOL was extracted, PVR membranes were dissected, an inferior retinectomy was created and endophotocoagulation applied to the margins of the same, an inferior peripheral iridectomy formed and 5000 centistoke SO inserted into the vitreous cavity.

Two months following this, this gentleman attended the Eye Casualty department with a two day history of a red, painful right eye. At examination this gentleman could perceive only hand movements in the right eye and 14 mm Hg in the left eye. The right pupil was mid-dilated. This eye was injected. Corneal oedema was present. The intraocular pressure (IOP) was 40 mm Hg in the right eye and 14 mm Hg in the left eye. Right eye iris bombe was apparent. Non-emulsified SO was visible in the anterior chamber (AC). The inferior peripheral iridectomy had been occluded by a fine inflammatory fibrin membrane. Gonioscopy of the right eye showed SO in an open AC angle circumferentially. Both optic discs looked normal. The diagnosis of pupil block glaucoma was made.

Management

This gentleman received 500 mg Acetazolamide intravenously and g. timolol 0.5% and g. apraclonidine 1% were administered to his right eye in the Eye Casualty Department. IOP was reduced to 30 mm Hg. A Neodymium: Yttrium Aluminium Garnet (Nd: YAG) laser peripheral iridotomy was performed and the IOP declined further to 25 mm Hg. The patient was discharged home to use g. apraclonidine 1% three times daily and g. dexamethasone 0.1% four times daily for the subsequent month. On a third similar presentation to the Eye Casualty Department however this gentleman's IOP failed to respond to medical therapy and an attempt to re-open a peripheral iridotomy.
Nested YAG laser was also unsuccessful. As retinal reattachment had been achieved at this point, the SO was removed from the eye.

PVR recurred in this gentleman’s eye. He underwent twenty gauge PPV with epiretinal membrane peel, removal of PVR, extension of the previous retinectomy, application of 360° endophotocoagulation, creation of a larger inferior peripheral iridectomy and insertion of 5000 centistoke SO. In Figure 1 the large inferior peripheral iridectomy created at this time is shown. This gentleman’s IOP was maintained below 25 mm Hg with the administration of g. apraclonodine 1% three times daily and g. brinzolamide (10 mg/ml) and timolol (5 mg/ml) twice daily postoperatively. Removal of SO was performed six months later. His ocular pressure lowering treatment was subsequently discontinued. This gentleman’s IOP has been less than 20 mm Hg at each of his subsequent reviews. Snellen VA from this patient’s right eye is now 6/60. Unfortunately no improvement in this could be obtained with contact lens correction of his refractive error. As can be seen from Figure 2 the right optic disc is pale and cupped.

Discussion

SO tamponade is now widely used in the surgical management of complex retinal detachment and severe PVR such as might occur following retinal detachment, penetrating ocular trauma or with proliferative diabetic retinopathy (PDR) [1]. Glaucoma following PPV and SO injection remains an important complication of such surgeries with postoperative elevations in IOP occurring in as many as 48% of patients having these procedures [2].

The pathogenesis of glaucoma in eyes after PPV with SO insertion can be difficult to determine [3]. The underlying ocular disease and other aspects of the surgery performed, placement of a scleral buckle for example, may influence aqueous humour production and/or outflow so that in these eyes IOP represents the balance of the complex interplay of many factors. The possible causes for elevations in IOP after PPV and SO infusion are given in Table 1

In these cases elevated IOP is not always related to the presence of SO in the eye.

This report details a case of pupil block glaucoma in a patient after PPV with SO injection. The mechanism by which this occurs is shown in Figure 3. In vitrectomized eyes into which SO has been inserted it can cause secondary angle closure and/or the migration of SO into the AC where it can further limit aqueous humour outflow as in the case we describe here. An inferior peripheral iridectomy is created at the time of PPV and SO insertion in an attempt to maintain the passage of aqueous humour from the posterior to the anterior chamber postoperatively and in doing so prevent the occurrence of pupil block and the movement of SO into the AC [4]. An inferior peripheral iridectomy may however fail to keep the AC free of SO for a number of reasons [5]. These are detailed in Table 2.

The inferior peripheral iridectomy created at the time of PPV and SO infusion may become occluded in up to 32% of patients postoperatively [6]. In the case of the gentleman described in this report an inflammatory fibrin membrane was found to have occluded the inferior peripheral iridectomy. The postoperative treatment of all those who have had PPV and SO insertion should therefore include intensive topical steroid in an attempt to prevent the inflammatory occlusion of their iridectomies.

The Nd: YAG laser can be used to re-open a peripheral iridectomy in such cases. This is a non-invasive and so a relatively low risk procedure that can be carried out in the outpatients department. Failure of the Nd: YAG laser to open a peripheral iridectomy in SO filled eyes is however uncommon. In one study wherein the initial surgical peripheral iridectomy of 18 eyes became occluded it was successfully re-opened by Nd: YAG laser in only 4 eyes or 22% of cases [7]. Using Kaplan–Meyer analysis the authors showed that the probability of such a peripheral iridotomy remaining open at 15 days was only 50% [7].

There are several reasons why the Nd: YAG laser may be unsuccessful in this regard [7]. Cycloplegic agents are routinely used following PPV. If Nd: YAG laser peripheral iridotomy is required to resolve pupil block the pupil is likely to be found dilated with the iris tissue bunched peripherally and so difficult to penetrate with the laser. Any haemorrhage from a laser burst in a SO filled eye is held against the target site by the oil with which it is immiscible and so does not disperse. This makes a second burst of laser at the same site almost futile. Enlargement of a peripheral iridotomy successfully created by the Nd: YAG laser is made difficult by this lack of dispersion of debris from
the site of the peripheral iridotomy with the result that most iridotomies produced by the Nd: YAG laser are small and so prone to closure. Finally the refractive properties of SO may affect the spot size and the energy delivered to the iris by the laser. Nd: YAG peripheral iridotomy then may, at best, be a temporary solution to pupil block. A large surgical peripheral iridectomy likely represents the better solution in eyes from which the removal of SO is undesirable [7].

Pupil block. Absent PI.
Pupil block. Non-patent PI.
Emulsified oil in the AC.
Angle closure. PAS formation.
Surgical procedures used in conjunction with PPV e.g. scleral buckling, endophotocoagulation, cryotherapy
Exacerbation of pre-existing OAG.
Steroid-induced glaucoma.
Inflammatory glaucoma.
Neovascular glaucoma.

Table 1: The Aetiology of Glaucoma following PPV with SO Infusion. PI=Peripheral Iridectomy, AC=Anterior Chamber, PAS=Peripheral Anterior Synechiae, PPV=Pars Plana Vitrectomy, OAG=Open-angle Glaucoma.

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<th>Reason for Failure</th>
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<tr>
<td>Too small.</td>
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<td>Incorrect location. Mid-peripherally and not at the extreme periphery. Involving the pupil margin.</td>
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<td>Occlusion by a fibrin membrane.</td>
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<tr>
<td>Low aqueous production/turnover as can occur when anterior PVR causes ciliary body detachment.</td>
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<tr>
<td>Hypotony causing choroidal detachment which has the tendency to reduce the volume of the vitreous cavity.</td>
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<td>Retinal fibrosis which causes the vitreous cavity to shrink and thereby reduces the volume it can contain.</td>
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Table 2: Reasons for Failure of an Inferior Peripheral Iridectomy.

The role of SO removal for the control of glaucoma after PPV with SO infusion is, as yet, unclear. One group found that SO removal did not affect IOP in 91% of such cases [8]. Others have found that IOP might be controlled in up to 68% of cases by this procedure [9].

The likelihood of controlling glaucoma with the removal of SO may be time dependent. The relatively early removal of oil probably reverses the mechanical blockade of aqueous humour outflow already mentioned allowing for improved IOP control following oil removal. With time SO in the AC can become emulsified; the oil breaks down into smaller droplets. These can be identified at the slit-lamp in the AC or by gonioscopy, in the angle, as in the case we describe here. When extensive, emulsification is manifested by the well-known inverse hypopyon in the AC. Emulsified SO sequestered in the trabecular meshwork can cause macrophage mediated inflammation-trabeculitis [10,11]. This, in turn, may be associated with changes in the collagen network of the trabecular meshwork following the occurrence of which, SO removal may have no effect on lowering IOP. Of course, the removal of SO may be associated with re-detachment in up to 33% of eyes [9].

There is relatively little information available regarding the appropriate management of glaucoma after PPV with adjuvant SO injection. In Figure 4 we have provided a treatment algorithm for consideration when such cases arise. Medical therapy may be successful in controlling glaucoma in 30-78% of cases [1,12]. Topical and/or systemic aqueous suppressants are used commonly in this regard and have been advocated by others for use in cases similar to that described here wherein pupil block glaucoma has necessitated Nd: YAG peripheral iridotomy [3]. As aqueous production might be considered necessary to re-fill the AC and maintain the patency of such an iridotomy we would urge caution in using these agents following laser iridotomy.

Scarring of the conjunctiva after vitreoretinal surgery makes trabeculectomy technically difficult and means the trabeculectomy failure rate is high in patients with glaucoma after PPV and SO injection [2]. Further, if performed superiorly the drainage channel created at trabeculectomy may be blocked by SO while an inferior trabeculectomy which might avoid this problem carries an increased risk of endophthalmitis. IOP control by trabeculectomy was achieved in 4 of 9 eyes in one series 9 and in 3 of 5 eyes in another series wherein it was combined with Mitomycin C (MMC) [1]. Glaucoma drainage devices, the Ahmed valve for example, may be a useful alternative [13]. With the superior placement of these devices SO may again block the drainage channel and/or escape into the subconjunctival space [14,15]. Some success with the inferotemporal placement of these devices has been documented [12]. Transscleral cytophotocoagulation may be the best treatment for eyes with poor visual potential from which it may not be necessary to remove the SO or in which the control of IOP is required primarily for pain relief or
when other glaucoma surgical procedures have failed or are not feasible [1,2].

Figure 3: Pupil block glaucoma in a silicone oil filled aphakic eye. In the absence of a peripheral iridectomy or in the presence of an occluded one, silicone oil can fill the pupillary aperture. Aqueous humour accumulates behind the iris, inferiorly, in the posterior segment. 1. The peripheral iris bows forward and may occlude the drainage angle. 2. The continued accumulation of aqueous may force silicone oil into the AC where it can cause further mechanical obstruction to the drainage of aqueous humour.

Conclusions

Elevated IOP is a common complication of PPV and SO infusion. Consequently, careful postoperative monitoring of IOP is required in patients who have had this type of surgery. Determining the underlying cause for glaucoma in eyes after PPV and SO infusion is crucial for the appropriate management of this condition. Elevated IOP in these cases is not always related to the presence of SO in the eye. Gonioscopy is an integral part of the comprehensive examination of these eyes and can help distinguish between angle closure and the mechanical obstruction by SO of the trabecular meshwork. Depending on the particular pathogenesis of glaucoma in these cases sequential therapeutic measures might include medical therapy to temporize an acute elevation in IOP, Nd: YAG laser peripheral iridotomy, SO removal, trabeculectomy with or without MMC and glaucoma shunt procedures. Transcleral cyclophotocoagulation can be considered in refractory cases or in those where there is poor potential for vision from the affected eye.

This report adds to what is scant pre-existing information on the topic of postoperative elevations in IOP in eyes following PPV and SO insertion and highlights the importance of glaucoma management after this type of surgery. As in the case described here, much emphasis is placed on the successful re-attachment of the retina in these cases. Delayed detection and/or suboptimal monitoring of optic nerve damage can occur. Glaucoma after PPV with adjuvant SO can lead to serious glaucomatous optic nerve damage.
References


