



Recurrent iris bombé after laser peripheral iridotomy in a pseudophakic, vitrectomised eye: pupillary block, aqueous misdirection, or both?

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ABSTRACT

Both pupillary block and, especially, aqueous misdirection syndrome (AMS), are infrequent complications after pars plana vitrectomy (PPV). Posterior synechiae (PS) formation can occur after PPV or phacovitrectomy, especially with a previous history of diabetic retinopathy, laser photocoagulation, or advanced degree of cataract. However, secondary angle closure due to PS after PPV has rarely been reported. AMS in vitrectomised eyes has been linked to the remnant of an intact anterior hyaloid or, less frequently, to the formation of a fibrinous pupillary or retro-irideal membrane as a result of chronic inflammation. Unlike in AMS, a patent peripheral iridotomy (PI) is expected to reverse the pupillary block and restore the anatomy of the anterior chamber (i.e. iris bombé) in the absence of peripheral anterior synechiae. Here, we report a case of recurrent iris bombé after laser PI two months after combined phacovitrectomy surgery.

INTRODUCTION

Transient elevations of intraocular pressure (IOP) are one of the most common complications after pars plana vitrectomy (PPV). However, pupillary block and, especially, aqueous misdirection syndrome (AMS), are both rarely described following PPV. The formation of posterior synechiae (PS), when circumferential, can cause seclusio pupillae, leading to anterior bowing of the peripheral iris stroma (iris bombé) and secondary acute or chronic angle closure. AMS is characterized by increased intraocular pressure due to apposition of the ciliary processes to the anterior vitreous and subsequent diversion of aqueous into the vitreous cavity. If the anterior hyaloid face has reduced permeability to aqueous, the fluid is trapped within the vitreous cavity, resulting in increased vitreous pressure and axial anterior chamber (AC) shallowing despite a patent peripheral iridotomy (in contrast to seclusio pupillae)¹. AMS has more commonly been described in pseudophakic eyes, possibly because the wider horizontal

diameter of a pseudophakic bag is more likely to get in contact with the ciliary processes. However, there are very few cases reported of AMS after PPV^{2,3}.

Here, we present a peculiar case of recurrent angle closure with iris bombé despite laser peripheral iridotomy (PI) two months after combined phacovitrectomy surgery.

CASE REPORT

A 37-year-old Afro-Caribbean male presented to the eye emergency department complaining of left eye pain, nausea and vomiting since the previous night. He had a recent history of left phacovitrectomy seven weeks earlier for proliferative diabetic retinopathy (PDR) with recurrent vitreous haemorrhage (VH) refractory to both medical and laser treatment. The surgery had been performed uneventfully, with intact posterior capsule, air tamponade at the end, and no complications in the immediate postoperative course.

Right eye examination was unremarkable, with deep AC and quiet pseudophakia. Examination of the left eye revealed diffuse corneal epithelial oedema and peripheral iris bulging with shallow AC (deeper centrally). Left visual acuity was hand motion, and intraocular pressure (IOP) was 72 mmHg with Goldmann applanation tonometry. The patient had no previous

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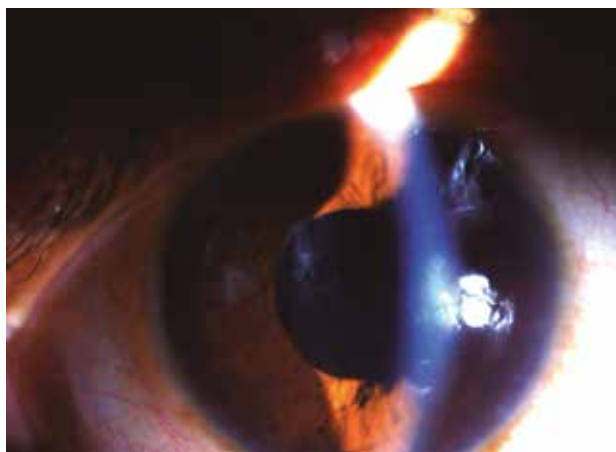


FIGURE 1. Slit-lamp photograph of the left eye showing peripheral iris bulging with deep anterior chamber centrally.



FIGURE 2. Visualization of the pupillary margin separate from the intraocular lens complex on dynamic gonioscopy (2-mirror Magna View lens).



FIGURE 3 AND 4. CASIA anterior segment optical coherence tomography images of the left eye showing recurrent iris bombé before widening of peripheral iridotomy (PI) (Figure 3), and complete anterior chamber deepening and patent PI one day after PI enlargement (Figure 4).

history of angle closure or narrow angles. No recent changes in his systemic medication were reported, and no other risk factors for secondary angle closure were identified.

One gram oral acetazolamide and topical medication with dorzolamide/timolol, apraclonidine 1%, dexamethasone and cyclopentolate 1% were given, and one hour later, the IOP reduced to 60 mmHg, with subjective improvement and clearer media. Angle closure was evident on gonioscopy, with 360 degrees iridocorneal contact (ICC). The pupil was fixed, but not dilated, and the iris looked bombé, although no PS were seen at the pupillary margin, either from a frontal view or through dynamic gonioscopy (Figures 1 and 2). Nd:YAG laser PI was performed, with significant deepening of the AC afterwards. One hour later, the IOP reduced to 12 mmHg, the corneal oedema had resolved, the retina was flat with no choroidal ef-

fusions, both optic nerves looked healthy, the AC was uniformly deep, and the angles were wide open in both eyes (Shaffer grade 3), with no peripheral anterior synaechiae or angle rubeosis. The patient was discharged with a tapering course of dexamethasone, as well as dorzolamide/timolol and atropine 1% eye drops to be continued bd for three days.

Examination was stable at twenty-four and ninety-six-hours follow-up. However, one week later (four days after discontinuing the eye drops), the patient presented again with progressive left eye pain and blurred vision starting that day, with IOP 45 mmHg and almost 360 degrees peripheral ICC despite patent PI. CASIA anterior segment optical coherence tomography (Tomey, Nagoya, Japan) showed peripheral iris bulging resembling iris bombé (Figure 3), although again no clear PS were evident. A component of aqueous misdirection was suspected, and Nd:YAG

laser anterior hyaloidotomy was performed in the mid periphery through the intraocular lens. However, no changes in the AC configuration occurred. Enlargement of the PI was then performed, and immediate AC deepening was noted, followed by progressive IOP reduction. The patient was given oral acetazolamide 250 mg bd only for that day, and maximal topical IOP-lowering medication, together with atropine 1% bd, was prescribed upon discharge. Twenty-four hours later, the IOP was 10 mmHg, with patent PI, deep AC and normal iris configuration (Figure 4). The patient remained stable on successive visits with the above treatment. One month later, all drops were stopped and follow-up was arranged two weeks afterwards, with stable findings and IOP 20 mmHg off medication.

DISCUSSION

Raised IOP is a frequently encountered complication after PPV. Around 15% to 56% of patients develop a transient IOP elevation within a few days after combined phacovitrectomy⁴. However, late postoperative IOP elevations of more than 30 mmHg after PPV in patients with no previous history of ocular hypertension or glaucoma are rarely reported, and only a low percentage of these cases converts to glaucoma. Secondary angle closure following PPV can be the result of, either pupillary block (mostly secondary to PS formation) or, less frequently, AMS. Both mechanisms can develop acutely or progressively, and the time to presentation may vary depending on the underlying cause.

The incidence of PS formation after PPV, especially if combined with phacoemulsification, has been reported to be between 6.1% and 30%, and main associated risk factors include preoperative PS, gas or oil tamponade, single-piece intraocular lens implantation, postoperative AC fibrin deposition, previous PDR, amount of endophotocoagulation, and long duration of surgery leading to increased inflammation⁵. However, secondary angle closure due to PS has much more rarely been reported. In a large retrospective case series of 493 eyes undergoing uneventful combined phacovitrectomy, Raj et al.⁴ reported a 1.82% incidence of postoperative secondary angle closure due to PS (95% CI: 0.64-3%), and the only significant risk factors identified were systemic diabetes with PDR, and advanced degree of cataract. However, it must be noted that, in all these eyes, silicone oil was used, and a single laser PI was enough to resolve the angle-closure attack and reverse the iris bombé. In our patient, the

only tamponade used was air, and the angle closure recurred despite an apparently patent PI, which led us to consider a coexistent AMS in the differential.

AMS after PPV has rarely been reported in the literature. Despite the precise pathophysiology is still unclear, and there may be several confounding factors, such as the use of gas or, especially, silicone oil tamponade, it has been postulated that the remnant of some intact anterior hyaloid, if abnormally impermeable to aqueous, would increase the posterior vitreous pressure and lead to AMS in predisposed eyes². Other authors have proposed that the formation of a pupillary or retro-irideal fibrin membrane should also be considered as a potential culprit in these cases, especially in eyes at higher risk of inflammation, suggesting argon laser membranectomy, and even intracameral tissue plasminogen activator, as complementary strategies to the conventional Nd:YAG iridozonulohyaloidotomy^{3,6,7}.

In our case, although we could not see any fibrotic membrane secluding the pupil, or any clear PS along the pupillary margin, the presence of some fibrinous or other obstructive inflammatory debris behind the PI site could not be ruled out, and may actually explain the success of the PI enlargement in resolving the problem. However, coexistent AMS in our patient would have produced a clear anterior displacement of the entire lens-iris diaphragm (not seen at any time), despite the presence of PS. The iris bombé configuration, together with a deep central AC, point towards a pupillary block mechanism secondary to some PS formation beyond the pupillary margin as the most likely diagnosis. It is also known that fibrin production is temporarily increased after laser PI and, therefore, rapid reformation of PS may occur, especially in an Afro-Caribbean patient with multiple inflammatory risk factors (uncontrolled PDR, previous photocoagulation). The time to presentation (7 weeks post-op) may also favour a pupillary block mechanism. In Raj et al.⁴ series, the interval to secondary angle closure was 2-8 weeks, which is when PS become firmly adherent to the anterior capsule and lead to iris bombé. Furthermore, the prolonged treatment with topical atropine may have helped break the retro-irideal adhesions potentially present after combined surgery in an eye with previous episodes of inflammation.

There are several learning points from this case which deserve special emphasis. Firstly, long-term treatment with cycloplegic agents after PPV and, especially, phacovitrectomy, is an essential measure to

decrease the risk of PS formation and secondary angle closure. Secondly, prompt identification of risk factors is mandatory to individualize management, and patients at higher risk of inflammation (i.e. PDR, previous photocoagulation, chronic inflammation, Afro-Caribbean ethnicity) may need more frequent postoperative follow-up. Careful IOP monitoring in the acute postoperative period is important, even if no gas or silicone oil were used. Finally, despite the non-visualization of PS at the pupillary margin, pupillary block always needs to be ruled out in all cases of secondary angle closure after PPV/phacovitrectomy, especially in eyes with inflammatory risk factors. A thorough examination to identify secluding membranes should be undertaken, and a well-sized PI should be performed before considering less likely causes, such as AMS.

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