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Case Series

Pseudophakic Acute Angle Closure Glaucoma Case Study

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Introduction

Pseudophakic Acute Angle Closure Glaucoma is an uncommon condition compared to phakic AACG. It might occur weeks, months or, in rare cases, years after cataract surgery [7]. The posterior chamber Intraocular lens plays a role in either:

- Causing pupillary block : this leads to iris bombé resulting in AACG [1]
- Changing the anatomical setting of the lens complex, ciliary processes and anterior vitreous face leading to Aqueous Misdirection Syndrome (Malignant Glaucoma) [2].

The aim of this article is to report a case of a pseudophakic patient with no history of glaucoma presenting with AACG four years after his cataract surgery. This didn`t respond to medical or laser treatment. Anterior pars plana vitrectomy and removal of lens-capsule complex was the successful management plan.

Case Presentation

A 87 years old male patient presented complaining of sudden drop of vision in the right eye accompanied by mild headaches. He had bilateral cataract surgery 4 years ago.

No other relevant ocular or systemic medical or surgical history. No history of glaucoma or significant hypermetropia or myopia.

On examination, visual acuity in the right eye was hand motion, in the left was 6/6 on Snellen chart. The intraocular pressure was 45 mmhg in the right eye, and 12 mmhg in the left one.

Both eyes looked white with no congestion.

Anterior chamber examination, in the right eye showed typical AACG signs: Hazy cornea with epithelial oedema, Very shallow AC with no significant cells count, Mid-dilated non-reactive pupil, No signs of neovascularization, PCIOL centered in place with no sign of posterior synechiae. There was mild posterior capsular opacity, Fundoscopy, Although the view was hazy, there was no evidence of retinal detachment or choroidal effusion, the optic disc looked healthy. Left eye examination was unremarkable.

A and B Ultrasound scan was done which showed no abnormalities such as choroidal effusion. UBM access was unavailable as the patient presented over the weekend.

Management

Initially the patient had was commenced on 500 mg PO Acetazolamide stat along with topical Atropine, Timolol, Latanoprost, Brinzolamide, Brimonidine and Maxidex.

Urgent YAG capsulotomy and anterior hyaloidotomy were performed centrally in addition to single YAG peripheral iridotomy despite the fact of poor visuality. These procedures led to IOP dropping to 32 mmhg and AC noticed to considerably deepen. Furthermore, headache resolved, and patient sent home with full treatment to be seen in the next day. Unfortunately, headaches came back in the following morning and IOP was over 60mmhg. Examination showed a hazy cornea with no funds view. AC was very shallow with irido-cornea touch. 500 mg Acetzolamide IV was given and IOP dropped to 44mmh.Then followed by IV mannitol which dropped the IOP to 20 mmhg. However, anterior chamber remained collapsed with no change of irido-corneal touch. Patient

was referred urgently to the Vitreal retinal team, He underwent anterior vitrectomy with removal of PCIOL with entire capsule as the complex became unstable. IOP is controlled medically after the surgery and the patient is still due for further follow ups to have another AC IOL later.

Discussion

The presented case is of a rare occurrence in which a pseudophakic patient with no history of glaucoma presented 4 years after the surgery with an episode of acute glaucoma with evident element of Malignant Glaucoma. In addition, there was an element of pupillary block according to the operating team who also noticed a thick Soemmering ring.

UBM assessment is essential to confirm the diagnosis.

Malignant glaucoma (Aqueous Misdirection)

The precise cause in not known. It is believed that abnormal anatomic relationship between the ciliary processes, crystalline or intra-ocular lens, and anterior hyaloid (along with possible thickening or abnormal permeability) causes posterior diversion of aqueous flow.

The classic picture is of elevated IOP that resists medical intervention or peripheral iridotomy. The anterior chamber typically becomes progressively shallower, and the IOP suddenly rises without a clear posteriorly pushing force such as a choroidal effusion or suprachoroidal hemorrhage [3]. B-scan to rule out masses in the posterior segment or choroidal haemorrhages or effusions is necessary. UBM (ultrasound biomicroscopy) showing an anterior rotation of the ciliary processes is considered pathognomonic for malignant glaucoma [4]. Treatment should start with cyclopegia (to deepen the anterior chamber and cause the posterior movement of the lens/iris diaphragm) and IOP lowering agents. YAG PI would follow with YAG capsulotomy/anterior vitreotomy (disruption of anterior hyaloid surface). Key factor is the size of PI and doing both capsulotomy and PI at the same site. If a peripheral iridotomy is not performed at the same site as the capsulotomy/anterior vitreotomy, a well-established pathway between the vitreous cavity and the anterior chamber may not be created [5]. Elevated IOP and a shallow anterior chamber may persist despite medical and laser treatment. Pars plana vitrectomy will be required and often resolves the process.

Pseudophakic angle-closure/pupil block

The etiology of the late onset presentation (as in the presented case) could be caused by a thick developed Soemmering ring. Soemmering ring is formed by the proliferation of the capsular remains of some retained lens epithelial cells. Three suggested mechanisms by which the Soemmering ring causes pupillary block [6]: 1) Soemmering-capsule-IOL complex causes increased thickness in the equatorial zone leading to iris bombé without detectable posterior synechiae. A successful PI would relieve the block and deepen the AC [6]. 2) Extended Soemmering ring pushes iris in apposition to the anterior chamber angle leading to irido-corneal touch. Capsulotomy through PI would resolve it [6].

Soemmering content causes pupillary block by acting as a one way valve to prevent anterior-posterior chamber pressure balance. PI would equalise the two chambers balance [6].

PI could be performed in multiple sites to avoid aqueous loculation from undetectable septums between posterior iris surface and anterior portion of Soemmering ring [6].

Conclusion

- Angle closure in pseudophakic eye is uncommon. Pupillary block is a common cause of angle closure in pseudophakic eye. Malignant Glaucoma is a serious cause that needs to be addressed promptly.
- B scan and UBM play crucial rules in demonstrating etiology.
- Proper diagnosis and prompt intervention are essential to the successful resolution of pseudophakic acute glaucoma.
- Disrupting the anterior hyaloid face and posterior lens capsule by laser and surgical treatment are the keys to success of malignant glaucoma treatment [3].

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