Short communication

Pupillary block acute glaucoma due to acrylic intraocular lens posterior dislocation AFTER Nd:YAG capsulotomy

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A B S T R A C T

Case report: We present the clinical case of a 68-year-old woman who developed a pupillary block acute glaucoma due to vitreous hernia into anterior chamber following posterior dislocation of the intraocular lens one month after an Nd:YAG capsulotomy.

Discussion: We should consider these symptoms in the differential diagnosis of pseudophakic acute glaucoma, especially when visualisation of the anterior chamber is difficult due to corneal oedema and Nd:YAG capsulotomy was done.

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Glaucoma agudo por bloqueo pupilar secundario a luxación posterior de lente acrílica intraocular tras capsulotomía Nd:YAG

R E S U M E N

Caso clínico: Se describe el caso clínico de una mujer de 68 años que presentó un glaucoma agudo secundario a un bloqueo pupilar por herniación vitrea a cámara anterior tras luxación de la lente intraocular a cámara vitrea un mes después de capsulotomía Nd:YAG.

Discusión: Se debe considerar este cuadro clínico en el diagnóstico diferencial del glaucoma agudo secundario, especialmente si existe el antecedente de capsulotomía Nd:YAG y el edema corneal impide una correcta visualización de la cámara anterior.

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Clinic case

Female, 68, who visited the emergency section due to blurred vision in the right eye (RE) with a 24-h evolution. Ocular antecedents included amblyopia in the RE, cataract surgery in the same eye one year earlier with ACR6D hydrophilic single block acrylic intraocular lens (IOL) implant in the capsular sac, without complications. In addition, one month earlier...
she had undergone an Nd:YAG capsulotomy totaling 75 mJ of energy due to progressive visual acuity loss caused by posterior capsule opacification (PCO). In the initial assessment, the best corrected visual acuity (BCVA) was finger counting at 1 m and normal intraocular pressure (IOP), and posterior subluxation of the IOL in the vitreous chamber (Fig. 1) through a broad posterior capsulotomy having a diameter of approximately 4.5 mm. A posterior 20 G pars plana vitrectomy was scheduled to refloat the IOL.

Three days later, while awaiting intervention, the patient visited again the emergency room with pain in the RE, headache and nausea. BCVA was hands movement. She exhibited intense ciliary hyperemia, intense epithelial and stromal edema, folds in Descemet membrane and narrow anterior chamber with iridoendothelial contact in the temporal area. The stromal edema did not allow a clear visualization of the anterior chamber, although the existence of the vitreous was apparent. The pupil was in slight bradichoric miosis. The IOP was of 55 mmHg, with a diagnosis of acute glaucoma due to pupil blockage of uncertain etiology, topical treatment was initiated with timolol+brinonydin (Combigan®) and systemic treatment with metamizol iv (Nolitol®) 1 bleb every 8 h, metochlopramide iv (Primperan®) 1 bleb every 8 h and 20% manitol 1.5 g/kg of weight plus furosemide iv (Seguril®) 1 bleb. After the IOP diminished to 20 mmHg, the corneal edema disappeared exposing the anterior chamber where a vitreous herniation was observed (Fig. 2) which prevented the flow of aqueous humor and caused pupil blockage with hypothalamia (Fig. 3). An Nd:YAG (60 mJ) laser iridotomy was performed, subsequently prescribing treatment with topical 0.5% ketorolac (Acular®).

Three days later 20 G pars plana vitrectomy was performed, refloating the IOL to the anterior chamber with perfluorocarbon. The IOL was explanted with tweezers through a 6-mm corneal incision. In the same surgical operation we implanted a lens with acrylic material and polymethylmethacrylate haptics, sutured to the iris due to the absence of sufficient capsular support. The postoperative period was normal, with good tension controls (Fig. 4).
One month after the intervention, the patient exhibited spontaneous visual acuity of 0.3 and was free of symptoms.

**Discussion**

The patient exhibited acute glaucoma secondary to vitreous pupil blockage after IOL dislocation into the vitreous chamber. The literature describes a similar case involving a silicone IOL. We have not found in the literature any case of the vitreous pupil blockage after posterior dislocation of a single piece acrylic IOL, even though the literature describes posterior dislocation of these lenses after laser Nd:YAG capsulotomy as well as with other type of lenses such as polymethylmethacrylate or silicone.3

PCO is the most frequent complication after cataract surgery, in the area of 10%. The treatment of choice is Nd:YAG laser capsulotomy. However, there is no consensus on the right capsulotomy size, although it is known that with smaller sizes there is a lower risk of retinal detachment and IOL dislocation. Information on its relationship with macular edema is doubtful. We must be careful when performing this technique to utilize the lowest amount of energy and maintaining the size between 2 and 3 mm.

The design of the hydrophilic single block acrylic IOL aimed at maximizing contact with the posterior capsule and thus reduce the risk of PCO. If the capsulotomy is excessively large, the force applied by the lens is redistributed even more towards the posterior capsule, thus facilitating its dislocation into the vitreous.4

The angular closure is defined as the aposition of the peripheral iris to the trabecular mesh, producing a reduction of aqueous humor drainage. The most frequent cause of secondary angular closure is pupil blockage, in which the flow of aqueous humor from the posterior chamber through the pupil is impeded and this generates a pressure gradient between the anterior and posterior chambers that pushes the peripheral iris towards the trabecular mesh, occluding the chamber angle. Pupil blockage can be absolute or relative. Various pupil blockage causes have been described such as lens thickening or anterior dislocation, posterior or myotic synechiae. In pseudophakic patients, a possible presentation of closed angle acute glaucoma is pupil blockage secondary to vitreous dislocation into anterior chamber. The definitive treatment in all cases is peripheral laser iridectomy.

In our case, broad capsulotomy with Nd:YAG laser facilitated spontaneous posterior IOL dislocation (not associated to traumaism) which ruptured the anterior hyaloid membrane allowing the entrance of the vitreous into the anterior chamber through the posterior capsule defect, giving rise to pupil blockage and therefore closed angle acute glaucoma which we resolved with Nd:YAG laser peripheral iridectomy.

This is not a frequent presentation of lens dislocation into the vitreous but we must consider it in the differential diagnostic of pseudophakic acute glaucoma, particularly when corneal edema prevents adequate observation of the anterior chamber.

**Conflict of interests**

The authors have no conflict of interests to declare.

**References**