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Clot-induced pupillary block and postoperative reverse pupillary block in Uveitis–Glaucoma–Hyphema syndrome associated with late in-the-bag IOL dislocation

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Abstract

Background Uveitis Glaucoma Hyphema (UGH) syndrome results from chafing between an intraocular implant and anterior chamber structures, and it typically presents with anterior chamber inflammation, elevated intraocular pressure and recurrent microhyphema or hyphema. Herein, we report the case of a patient with atypical presentation: iridocorneal angle closure secondary to a clot-induced pupillary block. The same patient developed reverse pupillary block a few weeks after surgery.

Case presentation A 77-year-old Caucasian woman presented with iridocorneal angle closure secondary to a clot induced pupillary block. The bleeding was caused by rubbing between a late in-the-bag dislocated intraocular lens and the iris in pseudoexfoliative syndrome. The diagnosis of UGH was initially delayed due to the unusual presentation. She underwent intraocular lens (IOL) exchange and secondary IOL scleral fixation, and subsequently developed reverse pupillary block. Both pupillary blocks were resolved by laser iridotomy.

Conclusions Uveitis Glaucoma Hyphema syndrome can rarely present with pupillary block, particularly in cases caused by late in-the-bag IOL dislocation in pseudoexfoliation syndrome. This case is unusual due to the occurrence of both pupillary block and subsequent reverse pupillary block in the same patient, highlighting the importance of recognizing these uncommon and atypical mechanisms.

Keywords Uveitis Glaucoma Hyphema syndrome, Blood clot, Acute angle closure, Reverse pupillary block, Iridectomy

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Background

Uveitis–glaucoma–hyphema (UGH) syndrome is an uncommon but potentially sight-threatening iatrogenic complication characterized by intraocular inflammation, elevated intraocular pressure (IOP), recurrent hyphema, and pigment dispersion caused by mechanical irritation of intraocular structures by implanted devices [1]. Intraocular lenses (IOLs) represent the most frequent source of this mechanical chafing, resulting in disruption of the blood–aqueous barrier and secondary inflammatory and glaucomatous sequelae [2]. While early reports primarily implicated rigid anterior chamber and iris-fixated IOLs, contemporary cases are more commonly associated with posterior chamber intraocular lenses (PCIOLs), particularly single-piece acrylic lenses positioned outside the capsular bag [2]. However, UGH syndrome has also been reported in association with scleral-fixated IOLs and, less frequently, with lenses implanted within the capsular bag, especially in the presence of progressive zonular instability [2–7].

The reported incidence of UGH syndrome ranges from approximately 0.4% to 1.6%, and its prevalence may be increasing due to the rising frequency of late IOL dislocation in the aging pseudophakic population [8]. Pseudoexfoliation syndrome (PEX) is a major predisposing factor, characterized by progressive deposition of abnormal fibrillar material within anterior segment structures, leading to zonular weakness, late IOL dislocation, and abnormal lens–uveal contact [9]. These structural alterations increase the risk of mechanical chafing and secondary inflammation.

Pupillary block is a recognized cause of secondary angle closure and occurs when aqueous humor flow from the posterior chamber to the anterior chamber is obstructed, resulting in anterior iris bowing and angle closure. In contrast, reverse pupillary block (RPB) results from a unidirectional valve-like mechanism between the iris and the crystalline lens or IOL, leading to posterior iris bowing and altered aqueous dynamics. RPB has been increasingly described following scleral-fixated IOL implantation. [10–12].

Although UGH syndrome typically presents with chronic inflammation and secondary open-angle glaucoma, acute angle closure is uncommon and not a direct feature of the syndrome. Here, we report a case of UGH syndrome due to late in-the-bag PCIOL dislocation in a patient with pseudoexfoliation syndrome, presenting with massive hyphema. In this case, secondary acute angle closure developed due to clot-induced pupillary block in the setting of intraocular lens instability and evolving UGH syndrome. The case is further distinguished by the postoperative development of reverse pupillary block after scleral-fixated IOL implantation, illustrating a rare sequential spectrum of aqueous dynamic disturbances.

Case presentation

A 77-year-old woman presented to the ophthalmic emergency department of Sant'Orsola-Malpighi Hospital in Bologna (Italy) with acute vision loss and severe ocular pain in her right eye. Her medical history was notable for bilateral uncomplicated cataract surgery performed 10 years earlier, pars plana vitrectomy for a macular hole in the right eye 2 years prior, PEX syndrome, and medically controlled glaucoma. Specifically, the patient was instilling tafluprost and timolol in both eyes; in the right eye, she was also instilling dorzolamide. The axial length was 26.98 mm in the right eye and 25.88 mm in the left eye. At presentation, best-corrected visual acuity in the right eye was no light perception, and intraocular pressure (IOP) was 50 mmHg. Slit-lamp examination revealed diffuse corneal edema, a shallow anterior chamber, grade II hyphema, and a fixed, mid-dilated pupil with a dense clot completely occluding the pupillary aperture. Slit-lamp examination and fundus examination of the left eye were unremarkable, and IOP was 16 mmHg. The patient had no history of ocular trauma and no systemic conditions associated with an increased risk of bleeding. She was not taking anticoagulant or antiplatelet medications and had no known blood dyscrasias or liver disease. Although the patient had type 2 diabetes mellitus, which could potentially contribute to anterior segment neovascularization, there were no signs of rubeosis iridis in the right eye. In addition, examination of the left eye revealed no evidence of diabetic retinopathy. Ocular ultrasonography showed no masses in the vitreous cavity and attached retina. This allowed us to exclude uveal effusion, posterior segment tumors, and other posterior space-occupying lesions.

Anterior segment optical coherence tomography (AS-OCT) confirmed iridocorneal angle closure. See Fig. 1. Based on the clinical and imaging findings, a diagnosis of angle closure secondary to clot-induced pupillary block was suspected. After administration of 500 mL of intravenous mannitol and topical pilocarpine, neodymium-doped yttrium aluminum garnet (Nd: YAG) laser peripheral iridotomy was promptly performed, resulting in immediate deepening of the anterior chamber, reopening of the iridocorneal angle, and marked reduction in IOP, thereby confirming the diagnosis of clot-induced pupillary block.

After partial hyphema resolution over the subsequent two weeks, repeat AS-OCT demonstrated superonasal anterior displacement of the in-the-bag intraocular lens with direct iris–IOL contact, establishing the diagnosis of UGH syndrome secondary to late in-the-bag IOL dislocation. See Fig. 2. Six weeks after initial presentation,

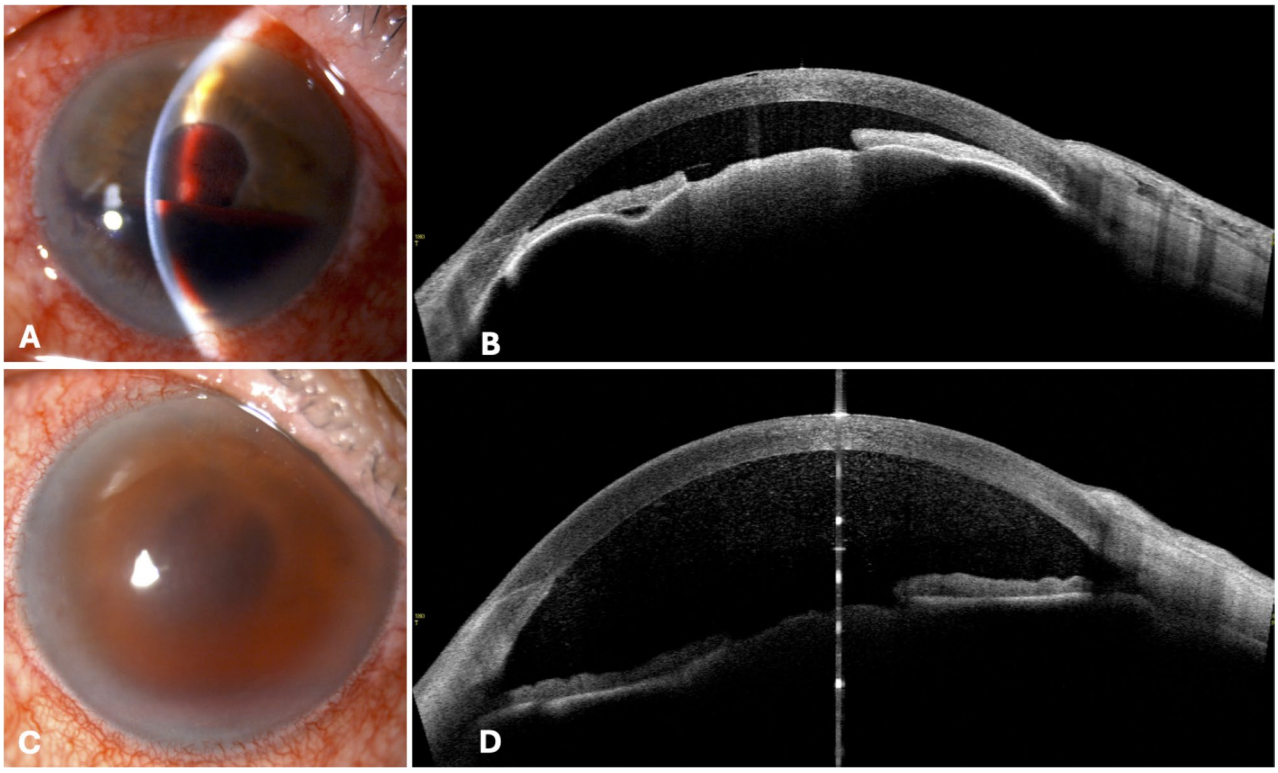

Fig. 1 Clot-induced pupillary block and resolution after laser iridotomy. **(A)** Anterior segment slit-lamp examination of the right eye shows mild corneal edema, shallow anterior chamber, grade II hyphema, fixed pupil and blood clot in the pupillary aperture. **(B)** The same clinical scenario is shown using anterior segment-optical coherence tomography (AS-OCT). Scan section of the AS- OCT is 0°-180°. **(C)** and **(D)** show the resolution of pupillary block after laser iridotomy. Scan section of the AS-OCT is 0°-180°

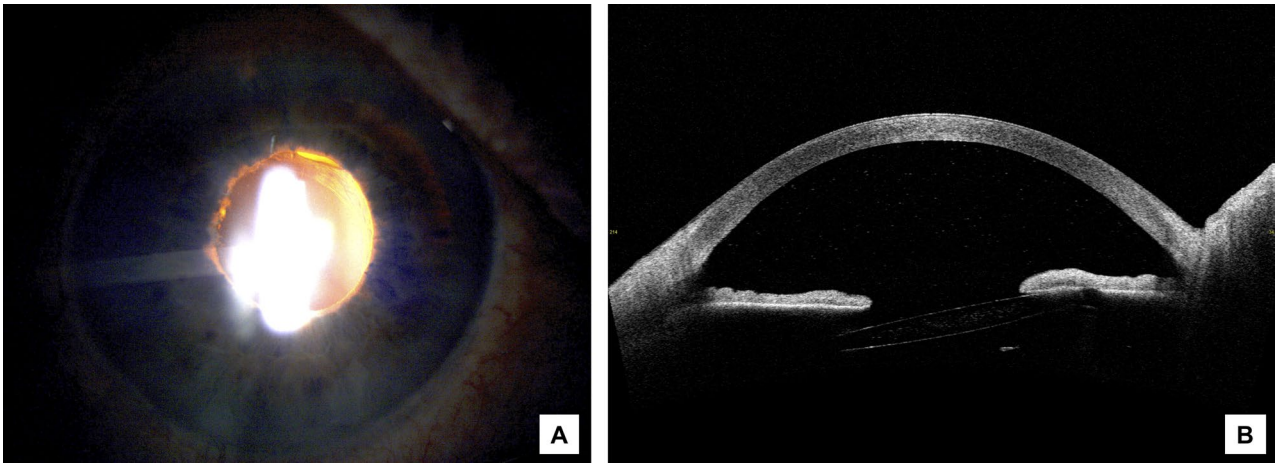

Fig. 2 Diagnosis of UGH syndrome after hyphema resolution. **(A)** Anterior segment slit-lamp examination of the right eye shows iris transillumination defects in the superonasal sector caused by contact with a displaced IOL–capsular bag. A patent iridotomy is also observed at 11 o'clock. **(B)** Anterior segment optical coherence tomography (AS-OCT) shows contact between the IOL and posterior surface of the iris in the superonasal quadrant. Scan section of the AS-OCT is 34–214°

the patient underwent explantation of the dislocated capsular bag–IOL complex and secondary IOL implantation (+8 diopter monofocal hydrophobic three-piece IOL) using the Yamane double-needle flanged intrascleral fixation technique [13]. See Supplementary Video 1.

One week postoperatively, she returned to the emergency room with severe ocular pain and decreased vision. Intraocular pressure was 45 mmHg, and the slit-lamp examination revealed mild corneal edema and a markedly deep anterior chamber. See Fig. 3A. AS-OCT demonstrated pronounced posterior bowing of the iris

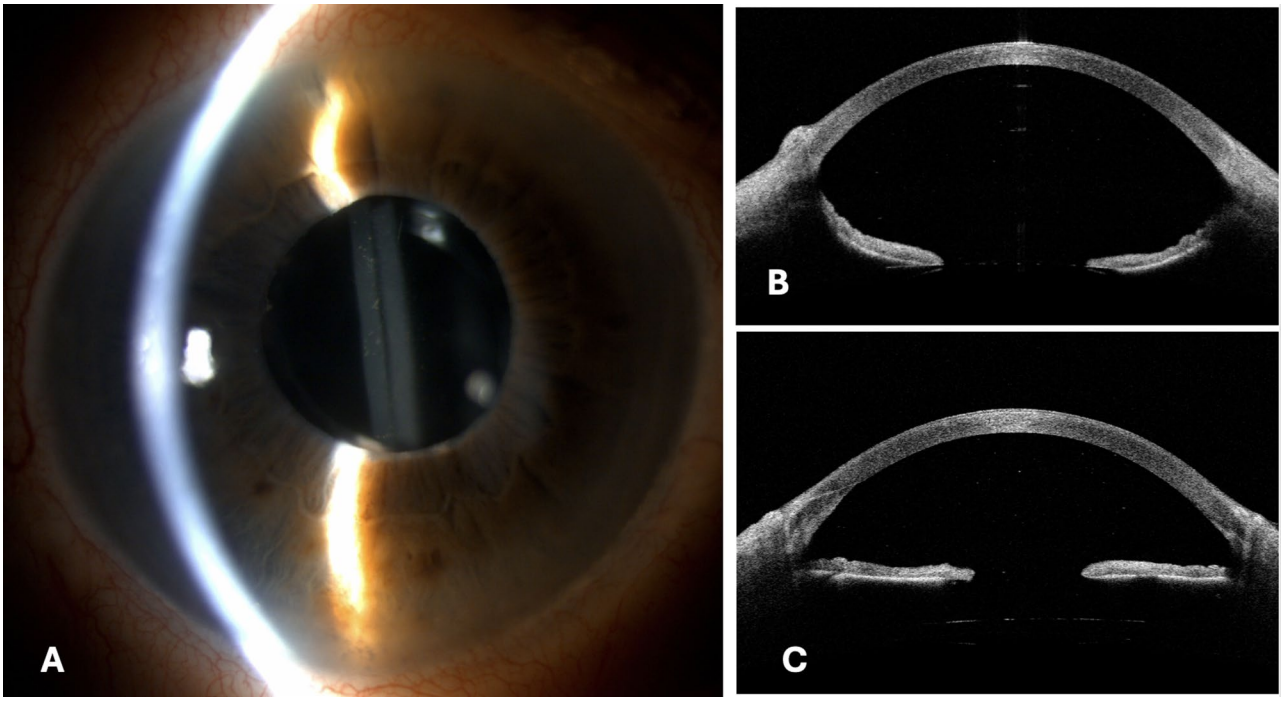

Fig. 3 Reverse pupillary block following surgery and resolution after laser iridotomy. **A**) Anterior segment slit-lamp examination of the right eye shows a very deep anterior chamber and posterior bowing of the iris. There is no diffuse corneal edema because the photo was taken after the administration of 500 mL intravenous mannitol. **(B)** AS-OCT shows a very deep anterior chamber and a contact between the iris and the scleral fixated IOL. Scan section of the anterior segment OCT is 0°-180°, coincides with the horizontal medial section of the eye. **(C)** AS-OCT after repetition of laser iridotomy in the temporal quadrant. Scan section of the anterior segment OCT is 0°-180°, coincides with the horizontal medial section of the eye

with extensive iris–IOL apposition, findings consistent with reverse pupillary block. See Fig. 3B. After administration of 500 mL of intravenous mannitol and topical pilocarpine, a second Nd: YAG laser peripheral iridotomy was performed, resulting in immediate normalization of iris configuration and reduction of IOP to 18 mmHg. See Fig. 3C. At the 6-month postoperative follow-up, best-corrected visual acuity had improved to 20/40, and IOP was 13 mmHg on the same pre-presentation topical anti-glaucoma regimen. The scleral-fixated IOL remained well centered, with no evidence of recurrent hyphema, persistent intraocular inflammation, or recurrent iris–IOL contact.

Discussion and conclusions

Uveitis Glaucoma Hyphema syndrome results from repetitive mechanical contact between an intraocular implant and adjacent uveal structures, leading to disruption of the blood–aqueous barrier, chronic inflammation, recurrent hyphema, elevated IOP, and iris transillumination defects [2]. It was first described by Ellingson as a complication of early rigid anterior chamber intraocular lenses (ACIOLs), and it is now recognized that UGH can occur with virtually all types of IOLs, including PCIOLs placed within the capsular bag [2, 6–8, 14]. Although in-the-bag PCIOL implantation is generally considered anatomically stable, several mechanisms have been proposed to explain UGH in this setting. Foroozan et al. described mechanical iris chafing caused by localized capsular–IOL positional changes associated with an iridociliary cyst [5]. Boutboul et al. reported anterior haptic bowing within the capsular bag resulting in posterior iris indentation [6]. Recently, Zhang et al. proposed two additional mechanisms: (1) PEX-related zonular instability leading to subclinical pseudophacodonesis and secondary posterior iris chafing by square-edged haptics, and (2) plateau iris configuration with focal capsular fibrosis inducing pseudoherniation of haptic segments toward the ciliary processes [15] Collectively, these reports demonstrate that even intracapsular PCIOLs may become pathologic when zonular integrity or anterior segment anatomy is altered.

In the present case, UGH syndrome likely occurred in the context of late in-the-bag IOL dislocation. Late in-the-bag dislocation refers to progressive decentration of the entire capsular bag–IOL complex occurring months to years after cataract surgery as a consequence of zonular weakening [16]. Its reported incidence has increased in recent years, likely reflecting both longer patient survival and cumulative zonular stress over time [16]. Pseudoexfoliation syndrome is the most common predisposing condition, with additional risk factors including

prior vitrectomy, uveitis, trauma, and increased axial length [17, 18]. In our patient, clinically evident PEX and prior pars plana vitrectomy likely contributed synergistically to progressive zonular compromise and eventual IOL–capsular bag complex displacement.

The most distinctive feature of this case was the development of acute angle closure secondary to clot-induced pupillary block. Uveitis Glaucoma Hyphema syndrome typically presents with recurrent microhyphema and secondary open-angle glaucoma; secondary angle closure is uncommon [1, 19]. In this patient, formation of a dense clot at the pupillary aperture completely obstructed aqueous humor flow from the posterior to the anterior chamber, producing a true pupillary block with rapid anterior chamber shallowing and angle closure. The immediate deepening of the anterior chamber following Nd: YAG laser peripheral iridotomy confirmed the mechanism. To our knowledge, acute angle closure due to clot-induced pupillary block as the initial presentation of UGH syndrome has not been previously reported.

Definitive management of symptomatic late in-the-bag IOL dislocation generally requires surgical intervention. Urgent surgery is recommended in cases of sudden symptom onset, marked pseudophacodonesis, or advanced dislocation [16, 20]. In the present case, a sutureless intrascleral fixation approach (Yamane technique) was selected due to its minimal tissue manipulation. This approach achieved excellent visual recovery, with a well-centered IOL and no evidence of recurrent hyphema, persistent intraocular inflammation, or recurrent iris–IOL contact at 6 months of follow-up. However, the patient developed RPB post-operatively, characterized by posterior bowing of the iris and iris–IOL apposition. Reverse Pupillary Block is thought to result from a unidirectional iris–IOL valve mechanism permitting aqueous flow into, but not out of, the anterior chamber [21]. Although its relationship with IOP elevation remains debated, our patient experienced marked pressure elevation. The reason for this phenomenon is unclear, and it remains unknown why RPB is associated with increased IOP in some cases but not others. Our hypothesis is that in our patient, pressure elevation may have been exacerbated by compromised trabecular outflow due to underlying PEX, prior UGH-related inflammation, and recent hyphema, potentially compounded by transient collapse of Schlemm's canal at high IOP levels [22]. This remains a hypothesis and cannot be substantiated at present. Therefore, we believe that further studies are needed to clarify the mechanisms underlying this phenomenon.

The reported incidence of RPB following scleral-fixated intraocular lens implantation varies widely. Schranz et al. reported that this complication may occur in up to one-third of cases of scleral-fixated IOL implantation in the absence of prophylactic iridectomy, with no significant differences observed among different surgical techniques. In contrast, other authors described it as uncommon. This variability underscores the need for more robust epidemiologic data and may have implications for considering prophylactic iridotomy in selected high-risk patients, particularly those with pseudoexfoliation syndrome or compromised aqueous outflow. Therefore, further studies are needed to better clarify the incidence, clinical significance, and associated risk factors of this phenomenon to improve patient management [10].

In summary, this case illustrates an atypical and sequential spectrum of aqueous dynamic disturbances in the setting of late in-the-bag IOL dislocation, with UGH syndrome complicated by massive hyphema and acute clot-induced pupillary block, followed by postoperative reverse pupillary block after scleral fixation. Recognition of these mechanisms is critical in pseudophakic patients with PEX, in whom late IOL instability may manifest with complex and evolving pressure dynamics. Although clot-induced pupillary block and reverse pupillary block have been described separately, their sequential occurrence in the same clinical setting appears to be rarely reported and may complicate diagnosis and management. Therefore, further studies are needed to better define the pathophysiological mechanisms underlying reverse pupillary block, identify associated risk factors and its prevalence following scleral-fixated intraocular lens implantation, and determine whether prophylactic iridotomy in selected patients undergoing scleral-fixated IOL surgery may be beneficial.

Abbreviations

UGH	Uveitis Glaucoma Hyphema
IOP	Intraocular pressure
AC	Anterior chamber
ACIOLs	Anterior chamber intraocular lenses
PCIOLs	Posterior chamber intraocular lenses
PEX	Pseudoexfoliation syndrome
AH	Aqueous humor
PC	Posterior chamber
RPB	Reverse pupillary block
OCT	Optical coherence tomography
AS-OCT	Anterior segment optical coherence tomography

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12886-026-04892-9.

Supplementary Material 1

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Not applicable.

Author contributions

BB collected the patient's information and drafted this manuscript and edited the photos. NV made contribution in collecting the patient's information and to supervision. MM did final approval. AM, AF and LF made critical revision of manuscript. All authors read and approved the final manuscript.

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Data availability
No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate
The study did not require an approval from the Ethics Committee. Written informed consent to participate was obtained.

Consent for publication
Written informed consent was obtained from the participant for the publication of this report and accompanying images.

Competing interests
The authors declare no competing interests.

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