• Review Article •

Etiology and management of uveitis-glaucoma-hyphema syndrome: a comprehensive review

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Abstract

● The uveitis-glaucoma-hyphema (UGH) syndrome, initially described in 1978, presents as an iatrogenic complication associated with contact between intraocular implant and ocular tissue. This syndrome encompasses a spectrum of clinical manifestations, including intraocular inflammation, elevated intraocular pressure, and recurrent hemorrhage. Advances in cataract surgery techniques reduced the incidence of early intraocular lens (IOL) dislocation while inversely increased rates of delayed dislocation. The primary etiology of UGH syndrome is IOL subluxation. Weakness of the ciliary zonules or unstable IOL fixation techniques may predispose the eye to iris-lens contact. Other contributing factors include Soemmering's ring formation, abnormal iris and ciliary body anatomy, positional changes, and improper positioning of glaucoma implants. Clinical examination and imaging modalities such as ultrasound biomicroscopy and anterior segment optical coherence tomography supports diagnosis of UGH. Treatment options range from observation and medical therapies to invasive procedures such as laser therapy, IOL repositioning, or replacement. Endoscopy provides direct visualization for identifying causes intraoperatively, aiding in tailored surgical approaches towards minimal intervention. In conclusion, UGH syndrome poses a complex clinical challenge, emphasizing the importance of understanding its etiology, accurate diagnosis, and personalized management strategies to mitigate its impact on visual function and ensure favorable outcomes.

● **KEYWORDS:** uveitis-glaucoma-hyphema syndrome; etiology; management

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INTRODUCTION

Uveitis-glaucoma-hyphema (UGH) syndrome, first described by Ellingson[1] in 1978, is a secondary inflammatory glaucoma that arises as an iatrogenic complication from the contact between intraocular implants and uveal tissue. The incidence of UGH syndrome ranges from 0.4% to 1.6%[2-3]. It features the triad of chronic inflammation, recurrent hyphema, iris transillumination defect, and elevated intraocular pressure (IOP) with occasional iris neovascularization and cystoid macular edema (CME)[2,4]. While it is primarily associated with inappropriately sized anterior chamber intraocular lens (ACIOL), it may also arise from malpositioned or subluxated posterior chamber intraocular lens (PCIOL). The iridolenticular contact leads to uveal tissue disruption, triggering inflammation, blood-ocular barrier breakdown, and microvasculature damage.

Historically, the incidence of UGH syndrome was mainly associated with ACIOLs, which induced mechanical irritation to the iris, ciliary body, and angle structures, resulting in recurring inflammation, increased IOP, and intraocular hemorrhage. Nonetheless, the emergence of modern intraocular lens (IOL) design, with biocompatible materials, polished edges, flexible haptics, and improvements in surgical methods, especially for posterior chamber lenses, has markedly reduced the occurrence of UGH syndrome linked to ACIOLs. Modern IOLs are engineered to reduce intraocular irritation and enhance lens stability. Surgical techniques, including accurate capsular bag implantation, scleral or iris fixation, and sutureless approaches such as the Yamane technique, have diminished the necessity for ACIOLs, further decreasing the risk of UGH syndrome. Furthermore, postoperative treatment,

encompassing vigilant surveillance and prompt correction for lens malposition, has enhanced results.

Notwithstanding the reduction in UGH instances attributed to ACIOLs, modern manifestations of UGH syndrome may still arise, frequently linked to misaligned or subxlued posterior chamber lenses[5]. These cases may present more subtly, with unstable PCIOL fixation, making early detection and management crucial. This review aims to inform doctors about the changing etiology, diagnosis, and management of UGH syndrome, especially for PCIOLs, and the persistent problems despite technological progress.

Methodology A structured literature search was conducted across multiple databases, including PubMed, Embase, and the Cochrane Library, covering articles published from January 1978 (when UGH syndrome was first described) to January 2024. The search utilized terms such as "UGH syndrome", "uveitis-glaucoma-hyphema", "intraocular lens complications", "IOL dislocation", and "iris-lens contact", and relevant articles' reference lists were also reviewed to identify additional studies. Inclusion criteria consisted of original studies, reviews, and case series addressing the etiology, clinical features, diagnostic approaches, or management strategies for UGH syndrome. A total of 363 individual UGH cases from the 121 studies met the inclusion criteria and were included in this review.

ETIOLOGY OF UGH SYNDROME

While the clinical severity of UGH syndrome has diminished with advancements in surgical techniques and implant designs[3], the prevalence of late IOL dislocations and subsequent surgical repairs appear to be on the rise[6]. To gain a deeper understanding of this condition, it is crucial to explore the current concepts and etiologies underlying UGH syndrome.

Late IOLs Dislocations Late dislocation of the IOL, occurring anywhere from several days to decades after surgery, can be triggered by trauma or spontaneous loss of zonular support, particularly in eyes with pseudoexfoliation syndrome, which is very common in various Northern European countries[7]. Over the past decade, there has been a notable increase in late IOL dislocations, with the IOL often remaining within the intact capsular bag[6]. The cumulative risk of IOL dislocation was 0.1% at 5y, 0.1% at 10y, 0.2% at 15y, 0.7% at 20y, and 1.7% at 25y in a large cohort of 14 471 pseudophakic patients[8]. Mönestam's[9] prospective study reported a 2% incidence of dislocation in eyes without pseudoexfoliation and 6% in those with pseudoexfoliation. A study of 60 cases of UGH demonstrated that 57% were associated with dislocated or unstable IOLs due to zonular laxity, with 48% of these being in-the-bag dislocations[2].

This delayed onset may be attributed to a higher volume of cataract surgeries and a trend toward younger patient populations receiving cataract surgeries. Moreover, weakening zonules leading to late IOL dislocations can alter the IOL-iris relationship, potentially increasing IOL contact with uveal tissue[10]. Capsular bag distortion can displace an IOL haptic[11], and all methods of restoring phakia after cataract extraction inevitably involve some contact with uveal tissue, which may trigger UGH-related complications.

IOL Repositioning/Replacement and Fixation Techniques As the frequency of IOL dislocations increases, both vitreoretinal and anterior segment surgeons are increasingly tasked with managing these cases. Numerous studies have compared techniques for IOL repositioning, replacement, and fixation. When inadequate capsular support is present, the IOL may be placed into the ciliary sulcus. Sulcus IOLs are more frequently associated with UGH than in-the-bag IOLs[12]. Moreover, in cases of inadequate capsular support, alternative fixation methods are employed, including suturing the haptics to the iris (iris fixation) or sclera (scleral fixation) or placing the haptics into intrascleral tunnels (intrascleral fixation)[13]. In some instances, the PCIOL may be removed through a limbal incision and exchanged for an ACIOL. A study of 30 UGH cases identified that 50% involved iris-sutured IOLs, 13.3% were associated with sclera-sutured IOLs, and 10% with sulcus-placed IOLs[14].

Unstable sulcus fixation is characterized by the rocking of the IOLs and extrusion of the haptic, which can lead to hyphema[15]. A study in China found that UGH syndrome was observed in all cases of single-piece acrylic IOLs in the sulcus[16]. Proposed mechanisms for this phenomenon include a thicker haptic (compared to 3-piece IOLs) that increases iris-optic contact, the absence of posteriorly angulated haptics, and a small loop-to-loop dimension that increases lens instability[17]. This may be exacerbated by more pronounced chafing between the IOL and iris due to a shallower anterior chamber depth in Asian eyes[18]. The iatrogenic occurrence of UGH syndrome can be minimized by avoiding the placement of single-piece acrylic lenses in the ciliary sulcus.

Unstable In-the-Bag IOL due to Zonular Laxity While UGH syndrome is commonly associated with malpositioning of an IOL or haptic outside of the capsular bag, there are reports of UGH syndrome resulting from iris trauma even when the IOL remains within the bag[19]. Surgical complications often arise due to a floppy iris-lens diaphragm, particularly in eyes with complex pathology such as high myopia[16].

Zhang *et al*[20] proposed a mechanism for in-the-bag UGH syndrome in a patient with pseudoexfoliation syndrome. They suggested that zonular instability leads to subclinical pseudophacodonesis of the haptic-capsule complex, resulting in chafing of the posterior iris. UGH is more commonly associated with square-edge optics than round-edged in the

sulcus in general. Focal capsular fibrosis around square-edged haptics, combined with anteriorly rotated ciliary processes in a plateau iris configuration, creates points of chafing.

UGH Related to Sutures In an interesting case report, a single-piece acrylic IOL was placed in-the-bag with a Cionni endocapsular tension ring (CTR) following a traumatic cataract accompanied by extensive zonular dehiscence[21]. Concurrently, the patient had complete traumatic mydriasis repaired with a pupillary cerclage suture. Despite successful management for over half a decade, the combination of zonular weakness and a broken scleral-fixation suture holding the Cionni ring led to anterior dislocation of the in-the-bag IOL-Cionni CTR complex. With the iris being atonic, movement of the dislocated complex was responsible for localized iris transillumination defects over the Cionni CTR fixation element. To address this, the repair was performed using 9-0 Prolene, recommended by the CTR manufacturer due to reported late suture breakage with 10-0 Prolene[22].

Secondary to a Soemmering Ring Cataract Soemmering ring is characterized by equatorial ringed proliferation of lens epithelial cells within the capsule, which occurs when the capsulorhexis is larger than the IOL optic, allowing adhesion of the anterior and posterior capsules[23]. While Soemmering ring is often clinically insignificant, it can cause capsular swelling and posterior iris irritation, with even potential for acute angle closure[24]. Reported cases have shown that fibrosis surrounding IOL haptics can displace the IOL from the plane of the iris, causing misaligned IOL haptics to chafe the posterior iris surface[21,25]. Although rare, UGH syndrome can occur even with a well-positioned IOL, emphasizing the potential of square-edged haptics to cause chafing, especially in patients with extensive IOL fibrosis.

Glaucoma Implant Malposition Another potential contributor to the expected rise in UGH incidence is the increased utilization of glaucoma drainage devices. The placement of these intraocular devices varies based on the technique and surgeon preference. Common complications following minimally invasive glaucoma surgery or tube shunt surgery include uveitis, CME, and intraocular hemorrhage[26]. Anterior placement of these devices can lead to corneal endothelial issues, while more posterior placement brings the tube into contact with the iris. Hyphema occurs in 14% of Ahmed valve implants[27]. Often, tubes are intentionally placed in the ciliary sulcus, which can precipitate iris contact. Other stents are inserted directly into Schlemm's canal (*e.g.*, iStent trabecular bypass stent[28], Hydrus microstent[29-31], CyPass) or the suprachoroidal space (*e.g.*, EX-PRESS mini shunt[32]).

Abnormal Structure of Iris, Ciliary Body, and IOL Several cases of UGH syndrome associated with abnormal structures of the iris, ciliary body, and IOL have been reported, each with diverse causes. These causes include reversing pupillary block in sulcus-placed PCIOLs[33], bowing of a haptic within the capsular bag due to IOL bending during loading[34], and chafing of the iris by an IOL optic displaced by an iridociliary cyst or iridoschisis[35]. Additionally, iris lymphoma can mimic recurrent hyphema following cataract extraction. This highlights the importance of considering malignant masquerade in patients with UGH syndrome, especially in patients with a history of lymphoma[36].

Trigger: Change in Body Position Clinical observations by our group suggest that patients are more susceptible to intraocular hemorrhage when shifting focus from distance to near vision. Additionally, changes in body position, such as bending over[37] or engaging in intensive facedown yoga sessions[38], have been observed to trigger UGH.

B-ultrasonography conducted in horizontal, sitting, and head-down positions revealed movements of IOLs corresponding to changes in position[39]. While the IOLs made contact with the iris pigment epithelium in sitting and head-down positions, they remained separated in the horizontal position. These dynamic interactions between IOLs and the iris/ciliary body demonstrate that IOL-iris interactions are position-dependent. Chronically intermittent chafing between the IOL and iris in specific head positions may also contribute to UGH syndrome.

DIAGNOSIS

The diagnosis of UGH syndrome does not require the presence of all three eponymous features. Instead, the presence of one or more signs in a patient with suspected contact between the implant and intraocular tissue is sufficient for the diagnosis. However, UGH syndrome diagnosis is often overlooked due to subtle clinical signs such as low-grade CME, occasional bouts of mild vitreous hemorrhage, asymmetry in trabecular meshwork pigmentation, and subtle elevations in IOP. Additionally, these signs may be delayed, sometimes by years after the original surgery, and can recur with long periods of remission.

Clinical examination findings are frequently missed[40]. The assessment of haptic position is often best achieved by viewing the eye tangentially using a slit lamp in a widely dilated eye. Gonioscopy can also provide helpful insights into the evaluation of UGH syndrome as trabecular meshwork pigment is one of the most common signs. Furthermore, gonioscopy allows physicians to rule out neovascular glaucoma and ciliary body tumors which may mimic UGH syndrome.

Ultrasound biomicroscopy (UBM) measurements offer additional insight for detecting IOL malposition and confirming haptic displacement outside of the capsular bag. UBM facilitates early diagnosis by enabling visualization of the exact position of the IOL *in vivo*, guiding treatment strategies such as haptic repositioning, haptic amputation, or IOL removal[41].

Anterior segment optical coherence tomography (AS-OCT) is a non-invasive technique used to determine IOL position and IOL-uveal contact in selected cases of UGH syndrome[42]. AS-OCT allows clinicians to study the location of the IOL, the evidence of contact elements, and the IOL-iris chafing site. The "peephole sign", described by de Simone *et al*[43], indicates the location of the contact point between the IOL implant and the posterior surface of the iris and serves as a useful hallmark for confirming UGH syndrome. Furthermore, AS-OCT provides detailed information such as capsular bag collapse and indirect signs of haptic malposition.

In cases where AS-OCT scans do not show IOL-iris chafing, UBM should be performed, preferably in different head positions, to rule out IOL-ciliary body chafing[42]. Differential diagnosis from hypertensive uveitis can be aided by careful examination of keratic precipitates, the presence of hyphema, and vitreous opacities[4]. Negative bacterial culture results for the IOL exclude the possibility of endophthalmitis caused by hypovirulent bacteria[44].

Endoscopic visualization is a valuable tool for diagnosing UGH syndrome, particularly when traditional imaging or clinical examination is insufficient. Endoscopic ophthalmic surgical systems consist of a fiberoptic camera, light source, laser aiming beam, and diode laser, allowing visualization of structures not readily accessible through standard anterior or posterior chamber viewing. These include the anterior chamber angle, ciliary body and processes, ciliary sulcus space, and anterior retina, especially in cases of opaque media. The endoscope can be introduced through a 2.2 to 2.4 mm clear corneal incision for anterior approaches or a 19-gauge sclerotomy incision for pars plana approaches. Endoscopic techniques have proven particularly useful in diagnosing unexpected causes of UGH syndrome[45], such as vascular lesions at prior pars plana sclerotomy sites, displacement of IOL haptics into the sulcus, inadequate zonular support for IOL repositioning, and synechiae[46]. Additionally, endoscopy enables the visualization of Soemmering's ring, which may push IOL haptics into the posterior iris, contributing to UGH syndrome. These diagnostic capabilities are critical in guiding appropriate surgical management.

MANAGEMENT

Observation and Medication For patients with mild symptoms, observation may be advisable. Minimizing potential triggers of UGH through activity restriction and head elevation has been suggested[47]. Utilizing a Fox shield to prevent nocturnal hand-eye trauma, as suggested by Berger[48], may also be beneficial.

In cases of mild to moderate UGH, conservative treatment with anti-inflammatory and IOP-lowering medications may suffice. Since UGH episodes may be interspersed by long periods of remission, topical therapy can be effective for acute exacerbations. Cycloplegics have shown efficacy in arresting intraocular bleeding in many cases. Initial management of CME with anti-inflammatory drops is recommended, with some eyes achieving long-term remission by tapering to a once-daily non-steroidal anti-inflammatory drug (NSAID) drop[40].

Glaucoma and ocular hypertension may arise in UGH due to the clogging of the trabecular meshwork by blood or pigmentary cells or the destruction of angle structures in case of an angle-supported anterior chamber lens. Elevated IOP can often be managed using standard approaches, however, consideration should be given to avoiding prostaglandin analogs that has the potential to exacerbate inflammatory activity. While anti-vascular endothelial growth factor (anti-VEGF) may be useful to control both inflammation and abnormal iris vasculature, potential complications such as acute and sustained increases in IOP, corneal endothelial damage over time, and risk of endophthalmitis must be considered[49].

Laser Therapy Further management of UGH involves both invasive and minimally invasive options, with laser therapy being a well-established treatment modality. Transscleral cyclophotocoagulation (TS-CPC) has been reported as a possible management option for UGH, particularly when the IOL is slightly tilted[50]. UBM has shown evidence of ciliary body shrinking after TS-CPC treatment, reducing contact with the IOL[51]. Despite its advantage in simultaneously treating elevated IOP and IOL positioning, TS-CPC carries the risk of pain, hypotony, exacerbating intraocular inflammation, and a rare risk of pthisis[52]. Endocyclophotocoagulation is another option, although it carries the rare risk of endophthalmitis due to being an intraocular procedure[53].

Iridoplasty offers a minimally invasive approach that effectively halts bleeding by ablating affected vessels and repositioning contact points between the haptic and iris[54]. In cases involving an EX-PRESS shunt, localized Argon laser iridoplasty successfully shifted the iris away from the shunt tip may resolve inflammation and hyphema[32]. Reliable identification of the iris area requiring treatment can be a logistical challenge, but techniques such as UBM visualization or conjunctival marking during slit lamp examination can aid in accurate laser spot placement[55-56].

Laser therapy can also be applied to manage other conditions associated with UGH syndrome. Reverse pupillary block in sulcus-placed PCIOLs can be effectively managed through laser peripheral iridotomy[33]. Additionally, if the IOL adheres to the iris or ciliary body, preoperative use of Nd:YAG laser to cut the synechiae can facilitate easier removal of the lens and minimize the risk of severe hemorrhage[57].

IOL Repositioning or Exchange UGH syndrome is one of the most common indications for IOL exchange, along

with dislocation of PCIOL and multifocal IOL intolerance[58]. If medical therapy fails or if inflammation threatens retinal or corneal function, IOL repositioning or exchange should be considered[59]. This procedure can be complicated by inflammatory scars, particularly in the anterior chamber angle or posterior to the iris. In such cases, the surgeon may need to amputate the haptics from the optic and remove the lens in a piece-by-piece fashion, rotating the haptic material out of the synechial tunnels to minimize eye trauma. Leaving portions of the haptics in place can be the last resort in case of failure. During vitrectomy for posteriorly dislocated IOLs, all vitreous adhesions to the IOL are removed to reduce vitreous traction on the retina when manipulating the lens back into the anterior chamber.

IOL explantation is often performed in cases with frequent recurrences and those that fail to resolve with medical therapy. Other treatment options include suturing the IOL to the iris, sclera, or capsule[60], IOL haptic amputation, IOL rotation or manipulation[61], placement of a capsular ring to redistribute zonular tension or serial intracameral anti-VEGF injections[62]. While surgical options have the advantage of removing the source of the problem, initial surgery fails to resolve UGH syndrome in up to 23.4% of cases[61]. Additionally, such surgical procedures carry potential risks, including but not limited to infection, retinal detachment, and cystoid macular edema[47]. Combining glaucoma surgery with IOP repositioning may be indicated in certain cases based on the staging of glaucoma and the clinical status of the angle based on gonioscopy.

Endoscopic Visualization Endoscopy plays a pivotal role in the surgical management of UGH syndrome, primarily by facilitating the elimination of contact between the IOL and the iris. This minimally invasive approach helps address a wide range of intraocular complications while minimizing surgical trauma. Endoscopic techniques allow precise removal of IOL haptics[63], identification and treatment of complications such as vascular lesions, and verification of proper haptic positioning or removal. Although using an endoscopic probe carries a risk of intraocular structure damage, this risk does not exceed other intraocular maneuvers during surgical intervention for UGH syndrome. Overall, endoscopic-assisted surgery enhances precision through direct visualization, improves outcomes, and broadens the scope of minimally invasive management options for UGH syndrome.

In conclusion, UGH syndrome presents a complex and multifaceted challenge in ophthalmology, necessitating a comprehensive understanding of its etiology, diagnosis, and management strategies. Throughout this discourse, we have explored various factors contributing to UGH syndrome, from late IOLs dislocations and abnormal iris structures to changes in body position as triggers. The diagnostic process, often complicated by subtle clinical signs and delayed presentations, relies on a combination of clinical examination findings, imaging modalities such as UBM and AS-OCT, and occasionally, endoscopic visualization. Treatment options range from conservative measures like observation and medication to more invasive approaches like laser therapy, IOL repositioning or exchange, and endoscopic interventions. Each modality carries its advantages and risks, emphasizing the importance of individualized patient management guided by the severity of symptoms and underlying pathophysiology. Despite ongoing advancements in surgical techniques and technology, challenges persist, underscoring the need for continued research and collaboration in addressing this intricate syndrome. Overall, a comprehensive approach that integrates clinical expertise, technological innovation, and patient-centered care is essential in effectively managing UGH syndrome and optimizing visual outcomes for affected individuals.

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