

Angle-Closure Glaucoma: An Insight

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Abstract

Primary angle-closure glaucoma is a serious condition responsible for nearly half of all cases of glaucoma-related blindness globally. The risk is notably higher in women, older adults, and individuals with hyperopia, with the highest prevalence observed in Asia. Ongoing research is exploring the genetic mechanisms behind glaucoma inheritance. Diagnosis is primarily conducted through gonioscopy, often supplemented by anterior segment optical coherence tomography (AS-OCT) and ultrasound biomicroscopy (UBM). Initial treatment usually involves pressure-lowering eye drops. The peripheral iridotomy is frequently performed to alleviate pupillary block, while laser iridoplasty has shown effectiveness for other closure mechanisms like plateau iris syndrome. Phacoemulsification, with or without goniosynechialysis, is an emerging treatment for both cataracts and clear lenses, with its long-term efficacy in early-stage angle closure under investigation. Cyclophotocoagulation, sometimes combined with cataract surgery, is another treatment option. For more advanced cases, trabeculectomy remains a reliable therapeutic approach.

Keywords: Angle-Closure Glaucoma; Pupillary Block; Laser Peripheral Iridotomy; Phacoemulsification; Plateau Iris; Primary Angle Closure

Introduction

Glaucoma is the second leading cause of irreversible blindness globally. It can be classified into Primary angle closure glaucoma (PACG) and Primary open-angle glaucoma (POAG).

About 60.5 million people were affected by POAG and PACG in 2010 [1]. By 2020, this number had rise to 80 million [2]. More than 80% of cases in the United States are of POAG; however, PACG results in severe vision loss [3]. This figure is expected to rise to 111.8 million by 2040 inequitably impacting individuals in Asia and Africa [4]. As per George., *et al.* approximately 11.2 million people are suffering from glaucoma in India aged more than forty years. POAG affects nearly 6.48 million people in India, and PACG affects almost 2.54 million people [5].

Angle-closure glaucoma (ACG) refers to a group of conditions where the closure of the anterior-chamber angle occurs either reversibly (appositional) or through adhesion (synechiae). This angle closure can present in either an acute or chronic form. In the acute form, IOP rises rapidly due to a sudden blockage of the trabecular meshwork by the iris, typically via the pupillary block mechanism [6].

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Primary angle closure disease (PACD) is divided into several categories. The International Society of Geographical and Epidemiological Ophthalmology (ISGEO) classification system classified the patients. Primary angle closure conditions can be divided into three stages. The first stage, known as Primary Angle Closure Suspect (PACS), occurs when there is a potential for the peripheral iris to encounter the posterior trabecular meshwork, which could lead to angle closure. The second stage, Primary Angle Closure (PAC), is characterized by an occludable drainage angle and signs that the peripheral iris is obstructing the trabecular meshwork. These signs include peripheral anterior synechiae (PAS), elevated intraocular pressure (IOP), iris whorling (distortion of the radially oriented iris fibers), glaucomflecken (lens opacities), or excessive pigment deposition on the trabecular surface. The final stage, Primary Angle Closure Glaucoma (PACG), occurs when PAC is accompanied by evidence of glaucoma, such as damage to the optic disc [7]. Secondary angle closure glaucoma occurs when angle closure raises IOP to the point where glaucomatous optic disc damage is generated. This condition is typically brought on by an underlying, distinguishable pathologic aetiology, such as neovascularization or uveitis.

Acute angle closure (AAC) is characterized by a sudden and significant rise in IOP with complete iridotrabecular contact (ITC), while plateau iris syndrome refers to persistent ITC following laser peripheral iridotomy (LPI).

Pathogenesis

PACG

Pupillary block

This is the condition in which inhibition of aqueous flow causes an increased pressure differential between the anterior and posterior compartments of the eye, leading to iris bowing and appositional closure of the angle [8].

The normal flow of aqueous humor moves from the posterior to the anterior chamber of the eye, passing between the posterior iris and the anterior lens. Typically, a pressure differential of about 0.23 mmHg between these chambers facilitates this flow [9]. However, when this pressure difference increases, it can cause the iris to bow forward into a convex shape. This convexity may bring the iris into contact with the trabecular meshwork, potentially blocking the drainage of aqueous humor. This blockage can lead to PAS and increase the risk of developing PACG. Eyes with shallow anterior chambers are more prone to pupillary block due to the proximity of the pupil to the anterior lens capsule. Moreover, research has shown that iris thickness can affect the pressure differential between the anterior and posterior chambers [10,11], with some studies suggesting that darker irides may be more susceptible to pupillary block [12].

Angle crowding

Compression of iris between the trabecular meshwork and another anatomical structure leads to angle closure by the phenomenon of angle crowding [13]. This is most observed in individuals with a plateau iris configuration, a condition that predominantly affects women aged 30-50 years, especially those with hyperopic refractive errors. Kumar, *et al.* estimated that 30% of eyes suspected of PAC were diagnosed with plateau iris using ultrasound biomicroscopy (UBM) after undergoing laser iridotomy. Plateau iris configuration is characterized by a large or forward-positioned ciliary body that pushes the iris root against the trabecular meshwork [14].

Secondary angle closure glaucoma

Anterior lens movement

The lens plays a significant role in the development of primary angle closure (PAC), with lenses positioned more anteriorly contributing to greater iris convexity [3]. This is seen in conditions such as phacomorphic glaucoma with advanced cataracts and choroidal expansion in malignant glaucoma e.g. Ectopia lentis, microspherophakia [15].

Absolute pupillary block

It occurs when the flow of aqueous humor through the pupil is completely obstructed due to 360-degree posterior synechiae, where the iris adheres to a crystalline lens, an intraocular lens, capsular remnants, or the vitreous face.

Without pupillary block

Angle-closure can result either from: (a) the contraction of an inflammatory, hemorrhagic, or vascular membrane in the angle, leading to the formation of PAS, or (b) the forward displacement of the lens-iris diaphragm, often associated with swelling of the ciliary body and anterior rotation. e.g. synechiae closure of the angle by fibrovascular membrane in case of neovascular glaucoma (Figure 1).

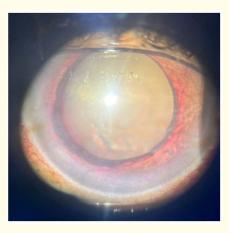


Figure 1: Shows neovascular glaucoma with florid neovascularization of Iris.

Risk factors for primary angle closure glaucoma

Common risk factors for PACD are family history of glaucoma [16], short axial length, feminine gender, [17] older age, Asian heritage, and hyperopia [18,19], Shallow central anterior chamber depth [20,21] thick and anteriorly positioned crystalline lenses [22].

Diagnostic approach

ACG typically presents with intermittent symptoms, including sudden vision changes, or more severe acute symptoms such as eye pain, headache, and associated nausea or vomiting. In contrast, chronic ACG is often found incidentally during a routine eye exam or while being examined for another condition.

So, the patients who are suspected of having ACG, thorough history and ophthalmic examination should be done.

History

When evaluating a patient with PACD, it's important to ask about any past symptoms of intermittent angle closure, such as blurred vision, halos around lights, eye pain, headaches, or eye redness. These symptoms can occur on their own or may be triggered by pharmacologic factors, like the ingestion or transdermal absorption of anticholinergics, or the use of mydriatic eye drops [23,24]. Reviewing the patient's family history may help identify relatives who have experienced AAC [25]. Additionally, the ophthalmologist should inquire about any systemic medications the patient is using that might lead to ciliary body edema and angle narrowing, such as sulphonamides (including topiramate), or drugs with adrenergic or anticholinergic effects, like ipratropium bromide, salbutamol inhalers, and phenothiazines [26-28].

Examination

Regardless of refractive status, hyperopic eyes and eyes with short axial length, particularly in older phakic individuals, have narrower anterior chamber angles and are more likely to develop PACD.

During or AAC, the affected eye may display changes in the size and shape of the pupil, often appearing oval, asymmetric, or middilated. Pupil reactivity can be reduced or entirely absent during the event. Additionally, a relative afferent pupillary defect (RAPD) may be present, either due to the increased intraocular pressure (IOP) or because of asymmetric optic nerve damage. During a slit-lamp examination, conjunctival hyperemia, particularly in acute cases, and narrowing of both the central and peripheral anterior chamber depth. Anterior chamber inflammation may be present, suggesting a recent or ongoing attack, and corneal swelling, with microcystic and stromal edema, is common in acute situations. Iris abnormalities, such as diffuse or focal atrophy, posterior synechiae, abnormal pupillary responses, irregular pupil shape, and a mid-dilated pupil, can also be observed, often pointing to a recent or current attack (Figure 2).



Figure 2: Shows mid-dilated fixed pupil with diffused iris atrophy in acute on chronic angle closure glaucoma.

Additionally, lens changes like cataract formation, anterior lens displacement, and the presence of glaukomflecken, which are localized anterior subcapsular lens opacities, may be detected.

Diagnosing anatomic narrow-angle or angle-closure primarily relies on gonioscopy, which remains the gold standard for evaluating the anterior chamber angle. The optimal technique for performing gonioscopy involves examining a dark room, using a small, dim light source that is just bright enough to visualize the angle structures. This careful approach is necessary because even a slight increase in light can open an appositionally closed angle in approximately one-third of cases, potentially leading to a misdiagnosis. [29,30]

Dynamic, compression, or indentation gonioscopy is crucial for distinguishing between appositional and synechial closure. By gently applying pressure on the cornea with the goniolens, the iris is pushed back, allowing the examiner to see if the angle can be opened further. If the angle remains closed despite the pressure, synechial closure is present. Additionally, this maneuver can be therapeutic, as it can help break acute angle closure attacks by forcing fluid into the peripheral areas, thereby opening sections of the angle that are appositionally closed.

When the angle width is less than 20° or the posterior, normally pigmented, trabecular meshwork is visible for less than 90° of the angle circumference, the angle is said to be occluded [31].

The classification schemes used in gonioscopy to measure the anterior chamber angle, which is important for glaucoma diagnosis and treatment. Each system is summarized as follows [32]:

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Angle Grading and Classification Systems	
A. Scł	neie System (R)
•	0 : The entire angle is visible, with a wide ciliary body band.
•	I: The last roll of the iris partially obscures the ciliary body.
•	II: No structures posterior to the trabecular meshwork are visible.
•	III: The posterior portion of the trabecular meshwork is not visible.
•	IV: No structures posterior to Schwalbe's line are visible.
Pigm	entation: Graded from 0 (no pigmentation) to 4 (heavily pigmented).
B. Sha	affer System (R)
•	0 : The angle is closed or slit-like.
•	1: Extremely narrow angle, ≤10 degrees.
•	2 : Narrow angle, approximately 20 degrees.
•	3 : Open angle, approximately 20-35 degrees.
•	4 : Wide open angle, approximately 35-45 degrees.
C. Spa	aeth System (R)
•	Level of Iris Insertion:
•	A: Anterior to the trabecular meshwork.
•	B : Anterior to the posterior limit of the trabecular meshwork.
•	C : Posterior to the scleral spur.
•	D : Into the mid-ciliary body face (with the anterior ciliary body band visible).
•	E: Posterior ciliary body (with a wide band of the ciliary body visible).
•	Angle Width: Measured in degrees from a line tangential to the trabecular meshwork to a line tangential to
the iri	is surface one-third of the way from the periphery, ranging from 0 to 40 degrees.
•	Curvature of the Iris:
•	r : Regular configuration with no significant forward or backward arching.
•	s: Steep or forward bowing (convex) curve.
•	q : Queer or posterior bowing (concave) curve.
Pigm	entation: Graded from 0 (no pigment) to 4 (heavy pigmentation).

Table

Imaging modalities

Ultrasound biomicroscopy (UBM) is an advanced imaging technique that produces detailed cross-sectional images of the eye's anterior segment, including the anterior vitreous. This method can penetrate opaque structures, allowing visualization of areas that are otherwise hidden during a standard clinical examination, such as the ciliary body.

Anterior segment OCT (AS-OCT), A diode light source is used instead of sound in AS-OCT to produce highly detailed images of the cornea, angle region, and anterior ciliary body, like those obtained with ultrasound biomicroscopy (UBM) [33]. However, unlike UBM, AS-OCT has limitations in imaging structures located posterior to the iris plane due to shadowing from the pigmented iris and light scattering from the sclera (Figure 3).

Differential diagnosis

When diagnosing PACS or PAC, it's important to remember that these conditions are typically bilateral. If a wide-open angle is observed in the fellow eye, it suggests a different diagnosis other than PACD.



Figure 3: AS-OCT- shows pupillary block glaucoma.

Anterior pulling mechanisms such as neovascularization of the iris, epithelial or fibrous ingrowth, descemetization of the anterior chamber angle as seen in iridocorneal endothelial (ICE) syndrome, contracture of inflammatory precipitates, Axenfeld-Rieger syndrome, or PAS following anterior segment surgery can also contribute to angle closure. Posterior pushing mechanisms are another cause and may result from retinal conditions that push the lens-iris diaphragm forward.

This can occur due to ciliary body edema or uveal effusion secondary to central retinal vein occlusion, pan-retinal photocoagulation, scleral buckle placement, or certain medications like topiramate or sulphonamides. Other posterior pushing mechanisms include intraocular gas or silicone oil causing forward displacement of the lens-iris diaphragm, persistent fetal vasculature, chronic or hemorrhagic serous choroidal detachment, intraocular tumors due to mass effect or direct angle invasion, retinopathy of prematurity, and aqueous misdirection, also known as malignant glaucoma.

The primary goals in managing PACD are to reverse or prevent the angle-closure process, control IOP, and prevent optic nerve damage.

Management goals

The management goals for a patient with PACD are to:

- Reverse or prevent the angle-closure process.
- Control IOP.
- Prevent damage to the optic nerve.

Acute attack of ACG

The immediate objective in managing an acute episode is to relieve symptoms and lower IOP, which is usually accomplished through medical therapy. The primary role of medical therapy in managing acute angle closure attacks is to lower IOP, alleviate pain, and clear corneal edema in preparation for an iridotomy. The following medications are commonly used, provided the patient has no contraindications:

• Topical medications

- Beta blockers
- Selective alpha agonists
- Carbonic anhydrase inhibitors
- Miotics (e.g., pilocarpine 2%)
- These can help break an early angle-closure attack but may be ineffective if the iris is already ischemic. High-concentration miotics (e.g., pilocarpine 4%) should be avoided due to the risk of forward displacement of the iris-lens diaphragm.

- Prostaglandin analogues: These are generally unreliable in acute attacks due to their slow onset of action.
- Hyperosmolar agents (e.g., 5% sodium chloride): These help clear corneal edema.
- Prednisolone 1%: This reduces inflammation.
- Systemic medications
- **Carbonic anhydrase inhibitors**: Oral acetazolamide reaches maximum IOP reduction in 2 4 hours, lasting for 6 8 hours. Intravenous acetazolamide begins reduction of IOP within 2 minutes, with peak effects at 10 - 15 minutes. In acute cases, a 500 mg dose of oral acetazolamide should be given if the patient is not vomiting. If vomiting is present, intravenous acetazolamide is preferred.
- Osmotic agents:
 - Mannitol: It can reduce IOP by 30 mm Hg or more within 30 minutes. The recommended intravenous (IV) dose is 0.5 1.5 g/ kg body weight in a 15% or 20% solution, administered at 3 to 5 mL/minute. Frail patients or those with cardiac conditions may develop complications such as circulatory overload, pulmonary edema, congestive heart failure, or electrolyte imbalances. Rapid reduction in cerebral volume may lead to subdural hematomas, so IV mannitol should be administered under hospital supervision.
 - **Glycerol**: Typically administered as a 50% solution at 1 to 1.5 g/kg body weight, it reduces IOP within 10 to 30 minutes. Glycerol should be avoided in diabetics due to the risk of ketoacidosis from the increased caloric load.
 - **Isosorbide**: This is available as a 45% solution, this agent has similar effects to glycerol but is safe for diabetics as it is not metabolized. The recommended dose is 1 to 1.5 g/kg body weight. Although less common, oral osmotic agents can also lead to subdural hematomas, with headache and gastrointestinal upset being common side effects.

Paracentesis

Paracentesis may be performed in acute settings to reduce IOP quickly, particularly when medications are slow to take effect or an iridotomy cannot be performed immediately. However, it is technically challenging in a phakic eye with a shallow chamber and carries risks such as permanent damage to the cornea, lens, and iris. Severe complications like endophthalmitis and choroidal hemorrhage may occur due to rapid pressure drops. Additionally, the effects of paracentesis are often short-term, as the ciliary body will resume producing aqueous humor, leading to a rise in IOP.

Laser iridotomy

Laser iridotomy should be performed as soon as possible on the affected eye and prophylactically on the contralateral eye to prevent future episodes of acute angle closure glaucoma. Complications of laser iridotomy may involve a rise in intraocular pressure (IOP), laser-induced burns to the cornea, lens, or retina, and delayed corneal edema. Other possible issues include the formation of posterior synechiae, hyphema, iritis, and visual disturbances triggered by light exposure [34].

Chronic angle closure glaucoma

Laser peripheral iridotomy (LPI)

It reduces the chronic disease's pupillary block component and may stop synechiae closure and increase the rise of IOP [35]. However, its effects on IOP management could not endure long, particularly in eyes where glaucomatous optic neuropathy has already set in. Often, additional medicine or surgical intervention is required [36].

Iridoplasty

Contraction burns are applied to the peripheral iris using long duration, low power, and a large spot size. This causes the iris stroma to contract, physically pulling the iris away from the drainage angle to help open it. In cases of acute angle closure, iridoplasty has been

shown to be both effective and safe for short-term reduction of IOP [37,38]. It is the preferred procedure for managing plateau iris syndrome, particularly when the angle does not open, and IOP remains elevated despite a successful peripheral iridotomy. In chronic cases of angle closure, iridoplasty may also help reduce the formation of PAS [39].

Cataract extraction

The surgical removal of the lens from an eye with a crowded anterior chamber can help open the angle and potentially prevent or reduce the formation of PAS. A study found that early phacoemulsification was more effective than LPI in preventing an increase in IOP after an acute angle closure event had been medically managed [40]. In the chronic phase, when a patent laser iridotomy and medical treatment have not successfully controlled intraocular pressure (IOP), lens extraction performed several months after the initial attack has been shown to lower IOP and reduce the reliance on IOP-lowering medications [41,42]. Recently, the EAGLE Study demonstrated the efficacy of clear lens extraction combined with intraocular lens implantation in treating PACG and PAC patients with excessive IOP (\geq 30 mm Hg at diagnosis).

Goniosynechialysis (GSL) with/without lens extraction

GSL is a surgical procedure used to strip peripheral anterior synechiae (PAS) from the trabecular surface, thereby restoring the flow of aqueous humor to the trabecular meshwork. Progression of cataracts, corneal damage due to endothelial cell loss, fibrin exudation, and mild bleeding are among the complications associated with GSI [43]. Combining GSL with lens extraction and phacoemulsification often results in significant visual improvement after surgery. This combined approach is more effective than GSL alone in controlling intraocular pressure (IOP), with studies reporting success rates between 85% and 100% in maintaining normal IOP and reducing the need for additional follow-up procedures [44,45].

Trabeculectomy

Trabeculectomy, a longstanding and primary treatment for glaucoma, has demonstrated reliable long-term effectiveness in managing IOP. Bevin., *et al.* reported a success rate of 79% in controlling IOP across various glaucoma subtypes [46].

Cyclophotocoagulation

In 1950, Bietti introduced cyclocryotherapy, a procedure that employs a probe cooled to -80°C to destroy the ciliary body epithelium, stroma, and vasculature. This destruction leads to a reduction in the production of aqueous humor and, consequently, a decrease in IOP.

The clinical utility of cyclocryodestruction is limited by complications such as hypotony, phthisis, hyphema, choroidal detachment, and retinal detachment. In recent years, diode lasers have been used for cycloablation, offering better tissue penetration and absorption [47]. Although diode lasers provide a safer alternative, they still carry risks, including hypotony, vision loss, corneal edema, pupil atony or distortion, and cystoid macular edema [48].

Glaucoma drainage implants

Glaucoma drainage implants are often more effective than filtering procedures in treating neovascular glaucoma and ICE syndrome because these conditions frequently involve the growth of a fibrovascular membrane over the sclerostomy site, leading to bleb failure [49].

Fellow-eye management

The fellow eye of a patient with AAC needs thorough evaluation due to its high risk of experiencing a similar event. If the chamber angle is anatomically narrow, a prophylactic LPI should be performed as soon as possible, as nearly half of the fellow eyes in AAC patients may develop acute angle closure within five years [50].

Conclusion

PACG is a major contributor to blindness worldwide. Although it accounts for only 26% of glaucoma cases, it is responsible for half of all instances of glaucoma-related blindness. The primary cause of angle-closure glaucoma is pupillary block, with anterior lens movement-often due to aging, cataract development, or changes in the posterior segment-being a significant contributing factor.

Treatment typically begins with IOP-lowering topical medications, such as aqueous suppressants and prostaglandins. Procedural intervention usually starts with peripheral iridotomy, which can positively influence disease progression, especially in the early stages, reducing progression rates to 0 - 16% compared to the 19 - 35% rate in untreated eyes. However, when LPI is performed in the later stages of the disease, additional medical or surgical treatments, like trabeculectomy or goniosynechialysis, are often needed. In recent years, lens extraction has emerged as a promising surgical option for lowering IOP and reducing the extent of PAS in the eye. Post-phacoemulsification, 65% of patients with PAC and cataracts achieve normal IOP without the need for medication.

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