

Angle-Closure Glaucoma (PACG)

Primary Angle-Closure Glaucoma (PACG)

Primary angle-closure glaucoma (PACG) is an ophthalmic emergency characterized by closure of the anterior chamber angle due to obstruction of the trabecular meshwork by the peripheral iris, causing a sudden or gradual increase in intraocular pressure (IOP). If untreated, it may lead to irreversible optic nerve damage and permanent blindness.

Pathophysiology

PACG occurs in anatomically predisposed eyes with:

1. Shallow anterior chamber
2. Narrow iridocorneal angle
3. Short axial length (hyperopic eyes)
4. Thick or anteriorly positioned lens

The peripheral iris blocks the trabecular meshwork, preventing aqueous humor drainage.

Mechanisms

1. Pupillary Block (Most Common)

Aqueous humor cannot pass freely from the posterior chamber to the anterior chamber because of increased contact between the lens and iris.

Pressure builds up behind the iris.

The iris bows forward (iris bombe).

The peripheral iris obstructs the trabecular meshwork.

Outflow decreases → IOP rises.

Angle closure usually occurs when the pupil is in a mid-dilated position, because both the sphincter and dilator muscles are partially relaxed, maximizing iris-lens contact.

2. Non-Pupillary Block

These include:

1. Plateau iris configuration
2. Thick peripheral iris
3. Forward rotation of the ciliary body
4. Large or anteriorly positioned lens

These mechanisms narrow or close the angle without a true pupillary block.

Types of PACG

1. Acute PACG

Sudden complete closure of the angle

Markedly elevated IOP (40–80 mmHg or higher)

Ophthalmic emergency

2. Chronic PACG

Progressive formation of peripheral anterior synechiae (PAS)

Gradual angle closure

Slowly progressive optic nerve damage

IOP may be intermittently or persistently elevated (>21 mmHg)

Clinical Features

1. Acute Attack

Symptoms

- | | |
|--------------------------|------------------------|
| a. Severe ocular pain | c. Halos around lights |
| b. Sudden blurred vision | d. Headache |
| | e. Nausea and vomiting |

- f. Abdominal pain
(occasionally)
- g. Photophobia

- 4. Mid-dilated fixed pupil
- 5. Elevated IOP (40–80 mmHg; occasionally >80 mmHg)
- 6. Optic disc edema (early)
- 7. IOP above 50–60 mmHg can produce permanent visual loss within hours if untreated.

Signs

- 1. Red congested eye
- 2. Corneal edema
(cloudy cornea)
- 3. Shallow anterior chamber

2. Prodromal (Intermittent) Attacks

- 1. Mild blurred vision
- 2. Colored halos around lights
- 3. Mild headache
- 4. Eye discomfort
- 5. Episodes last 30 minutes to 2 hours and resolve spontaneously.

Chronic PACG

- 1. Gradual visual field loss
- 2. Optic disc cupping
- 3. Peripheral anterior synechiae
- 4. Mild or absent symptoms
- 5. IOP normal or elevated

Secondary Angle-Closure Glaucoma (SACG)

Secondary angle closure occurs because another ocular or systemic disease pushes or pulls the iris forward.

Causes

1. With Pupillary Block

- a. Cataract (phacomorphic glaucoma)
- b. Posterior synechiae
- c. Aphakia or pseudophakia
- d. Intraocular tumors

2. Without Pupillary Block

- a. Plateau iris syndrome
- b. Neovascular glaucoma
- c. Uveitis
- d. Ciliary body tumors
- e. Choroidal hemorrhage
- f. Choroidal effusion
- g. Malignant glaucoma (aqueous misdirection)
- h. Drug-induced angle closure (Topiramate)

Drug-Induced Glaucoma

Drugs Causing Open-Angle Glaucoma

Corticosteroids:

- 1. Ophthalmic corticosteroids
- 2. Oral corticosteroids
- 3. Inhaled corticosteroids
- 4. Intranasal corticosteroids

Mechanism

Reduced aqueous outflow through the trabecular meshwork



Accumulation of extracellular matrix



Increased resistance to aqueous drainage

↓
Increased IOP

Patients requiring steroids should receive the lowest effective dose with regular IOP monitoring.

Drugs Causing Angle Closure

These drugs produce mydriasis or anterior movement of the iris-lens diaphragm.

Examples:

1. Anticholinergics (Atropine, Tropicamide)
2. Sympathomimetics (Phenylephrine)
3. Antihistamines
4. Tricyclic antidepressants
5. SSRIs
6. Venlafaxine
7. Topiramate
8. Sulfonamides (rare idiosyncratic reaction)

Topiramate mechanism

Ciliochoroidal effusion
↓
Forward displacement of the lens-iris diaphragm
↓
Acute bilateral angle closure
↓
Not due to pupillary block

Management of Acute Angle-Closure Crisis

Goals

1. Rapid reduction of IOP
2. Relieve pain

3. Prevent optic nerve damage
4. Prepare for definitive laser or surgical treatment

Pharmacological Treatment

1. Reduce aqueous humor production

- 1) Beta-blockers (Timolol)
- 2) Alpha₂ agonists (Brimonidine, Apraclonidine)
- 3) Carbonic anhydrase inhibitors
- 4) Acetazolamide (oral/IV)
- 5) Dorzolamide
- 6) Brinzolamide

2. Hyperosmotic Agents: Used when IOP remains very high.

IV Mannitol (1–2 g/kg)

Oral Glycerol (1–1.5 g/kg)

Mechanism: Creates an osmotic gradient that draws water from the vitreous into the bloodstream, rapidly lowering IOP.

3. Miotics

Pilocarpine is used only after IOP has started to fall (usually below 40 mmHg).

Mechanism:

Contracts the sphincter muscle

↓

Pulls the peripheral iris away from the trabecular meshwork

Pilocarpine is not effective at very high IOP, because the ischemic iris sphincter cannot respond. It is not routinely avoided; rather, it is delayed until the pressure falls.

4. Topical Corticosteroids: Reduce ocular inflammation and improve response to therapy.

5. Analgesics and Antiemetics_ Control pain and vomiting.

Non Pharmacological Treatment

Definitive Treatment

Once the cornea clears and IOP is controlled:

Laser Peripheral Iridotomy (LPI) — Treatment of Choice

Creates a small opening in the peripheral iris



Bypasses the pupillary block



Restores aqueous flow



Opens the anterior chamber angle

The fellow eye should also undergo prophylactic laser iridotomy because it is at high risk of developing angle closure.

If laser is not possible: Surgical peripheral iridectomy.