Secondary Glaucomas

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**Definition**

- **Glaucoma** - Group of disorders characterized by a progressive optic neuropathy resulting in characteristic appearance of optic disc and a specific pattern of irreversible visual field defects associated with raised intraocular pressure.

- **Secondary Glaucoma** – Group of disorders in which the raised IOP is associated with a primary ocular or systemic disease.
Classification

Depending on the mechanism of rise in IOP –
- Secondary open angle glaucoma
- Secondary angle closure glaucoma

Depending on the causative primary disease –
- Lens - induced glaucoma
- Inflammatory glaucoma
- Pigmentary glaucoma
- Neovascular glaucoma
Classification

- Glaucomas associated with irido – corneal endothelial syndromes
- Pseudoexfoliative glaucoma
- Glaucomas associated with intraocular hemorrhage
- Steroid-induced glaucoma
- Traumatic glaucoma
- Aphakic Glaucoma
- Glaucoma associated with intraocular tumors
- Ciliary block glaucoma
- Post-Operative secondary glaucomas
**Lens – induced glaucoma**

- Raised IOP secondary to a disorder of crystalline lens

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Phacomorphic glaucoma

- **Causes** -
  - Intumescent lens
  - Anterior subluxation or dislocation of the lens and spherophakia
- **Pathogenesis** – Swollen lens pushes iris forwards, obliterating the angle
- **Presentation** – Acute congestive glaucoma and shows features of acute primary angle closure glaucoma
Phacomorphic glaucoma

**Treatment** –

- Medical treatment – Control of IOP by iv mannitol, systemic acetazolamide and topical beta blockers
- Laser iridotomy
- Cataract extraction with implantation of PCIOL
Phacotopic glaucoma occurs when a hyper-mature cataractous lens may subluxate.
PHACOLYTIC GLAUCOMA

Clinical features
- Acute onset of monocular pain and redness
- Gradual reduction in VA
- High IOP
- Conjunctival hyperaemia
- Corneal xerosis
- Chunks of white material seen in the aqueous and anterior lens capsule and corneal endothelium which represent calcium oxalate or cholesterol crystals
- Vitreous opacification was seen (resolved in 12 weeks)
- Cataract – mature and opaque or hypermature; rarely immature
**Phacolytic glaucoma**

- Trabecular meshwork is clogged by the lens proteins, macrophages which phagocytose the lens protein and inflammatory debris
- Deep anterior chamber and aqueous may contain fine white protein particles, which settle down as pseudohypopyon
- Treatment includes medical therapy to lower IOP followed by extraction of hypermature cataractous lens with PCIOL implantation
Lens particle glaucoma

- Usually associated with retained lens material after cataract surgery.
- Trabecular meshwork is blocked by the lens particles floating in aqueous humour.
- Symptoms of acute rise in IOP associated with lens particles in the anterior chamber
- Medical therapy to lower IOP and irrigation – aspiration of the lens particles from the anterior chamber
Lens particle glaucoma
Phacoanaphylactic glaucoma

- Fulminating acute inflammatory reaction due to antigen – antibody reaction
- Granulomatous inflammation in the involved eye
- Preceding disruption of lens capsule by extracapsular cataract extraction, penetrating injury of leak of proteins from the capsule
- IOP is raised due to inflammatory reaction of the uveal tissue excited by the lens matter.
Management includes medical therapy to lower IOP, treatment of iridocyclitis with steroids and cycloplegics and irrigation – aspiration of lens matter from anterior chamber (if required).
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# Open – angle inflammatory glaucoma

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<th>Chronic open – angle inflammatory glaucoma</th>
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Angle - closure inflammatory glaucoma

- Mechanism of rise in IOP –
  - Secondary angle – closure with pupil block
  - Secondary angle – closure without pupil block

- Clinical features – Raised IOP, seclusio papillae, shallow anterior chamber

- Management –
  - Prophylaxis – Local steroids and atropine to prevent formation of synechiae
  - Curative treatment – Medical therapy, surgical or laser iridotomy and filtration surgery
Fuchs’ Heterochromic Iridocyclitis

Unilateral, and its symptoms vary from none to mild blurring and discomfort.

Signs include diffuse iris stromal atrophy with variable pigment epithelial layer atrophy small white stellate KPs scattered diffusely over the entire endothelium. The diagnosis is based on the distribution of KPs, lack of synechiae, lack of symptoms, and heterochromia.

A lighter-colored iris indicates the involved eye. In blue-eyed persons, however, the affected eye may become darker as the stromal atrophy progresses and the darker iris pigment epithelium shows through.

The etiology of Fuchs heterochromic iridocyclitis remains unclear. Associations with ocular toxoplasmosis, herpes simplex virus, and CMV infection have been suggested.

Glaucoma control can be difficult. Abnormal vessels may bridge the angle on gonioscopy.

The prognosis is good in most cases even though the inflammation persists for decades. Because topical corticosteroids can lessen the inflammation but typically do not resolve it. Histological examination shows plasma cells in the ciliary body, indicating that true inflammation occurs.
Posner-Schlossman Syndrome

On exam, the vision can vary from 20/20 to hand motion or light perception depending on the amount of corneal epithelial edema, although the amount of edema is usually mild. The pupil is often slightly dilated or sluggish. The conjunctiva is usually white and quiet, although a mild ciliary flush may be present. There may be small-to-medium, discrete, round, white keratic precipitates on the endothelium, usually in an inferior distribution. The anterior chamber is deep with a mild iritis without significant cell or flare.

The IOP is often markedly elevated, usually 40-50 mmHg. Episodic.
Medical therapy
Initial treatment is directed towards controlling intraocular pressure and decreasing inflammation. Typical first-line therapeutics include topical beta-blockers, alpha-agonists, and carbonic anhydrase inhibitors. Apraclonidine has also been advocated as a first-line agent. Oral carbonic anhydrase inhibitors are sometimes used acutely to lower the IOP quickly.

For control of inflammation a topical steroid drop is the usually used, such as prednisolone acetate 1% QID, given the typically low level of inflammation. Oral NSAIDS such as indomethacin may also be used to avoid a possible steroid-induced glaucoma and for their anti-prostaglandin properties.

Surgical therapy
In cases where the IOP cannot be controlled using maximal medical therapy, surgical therapy may be considered, especially when signs of glaucomatous optic nerve damage or visual field changes appear.

Prognosis
Posner-Schlossman Syndrome has long thought to be a “benign” disease; most patients are treated for attacks and recover without long-term sequelae. However, a number of patients with repeated attacks, even if treated, may show long term glaucomatous changes in the optic nerve and on visual field testing. It is thought that it is the total duration of elevated IOP, not the frequency of attacks, that contributes to the damage.
**Pigmentary glaucoma**

- Clogging up of trabecular meshwork by the pigment particles
- Patients with Pigment Dispersion Syndrome
- Pigment release caused by mechanical rubbing of the posterior pigment layer of iris with zonular fibrils
- **Clinical features** –
  - Young myopic males
  - Glaucomatous features similar to POAG
  - Deposition of pigment granules in the anterior segment
Pigmentary glaucoma

- Gonioscopy – pigment accumulation along the Schwalbe’s line especially inferiorly (Sampaolesi’s line)
- Iris transillumination – radial slit – like transillumination defects in the periphery
- Treatment is exactly on the lines of POAG
Neovascular glaucoma

* Intractable glaucoma results due to formation of neovascular membrane involving the angle of anterior chamber

* **Etiology** – Neovascularization of iris following retinal ischemia, feature of
  - Proliferative diabetic retinopathy
  - Central retinal vein occlusion
  - Sickle – cell retinopathy
  - Rare causes (intraocular tumours and long standing retinal detachment)
Neovascular glaucoma

- Clinical profile –
  - Pre – glaucomatous stage
  - Open – angle glaucoma stage
  - Secondary angle closure glaucoma
- Treatment –
  - Panretinal photocoagulation
  - Anti-Vegf Injections
  - Medical therapy not effective
  - Artificial filtration shunt (Seton operation)
Glaucoma associated with iridocorneal endothelial (ICE) syndromes

- 3 entities –
  - Progressive iris atrophy
  - Chandler’s syndrome
  - Cogan – Reese syndrome

- Presence of abnormal corneal endothelial cells which proliferate to form an endothelial membrane in the angle of anterior chamber
**Clinical features** – Affects middle-aged women
   - Progressive iris atrophy – iris features predominates with corectopia, atrophy and hole formation
   - Chandler’s syndrome – Mild iris changes and corneal edema predominates
   - Cogan – Reese syndrome – nodular and diffuse pigmented lesions of iris, may or may not be associated with corneal changes

**Treatment** –
   - Medical treatment
   - Trabeculectomy
   - Artificial filtration
Pseudoexfoliative glaucoma

- Deposition of an amorphous grey dandruff – like material on the pupillary border, posterior surface of iris and ciliary processes
- Associated with secondary open – angle glaucoma
- Trabecular blockage by the exfoliative material
- Managed on the same lines as POAG
**Glaucoma associated with intraocular hemorrhage**

- **Hyphema and vitreous hemorrhage**

- **Red cell glaucoma** – Associated with fresh traumatic hyphema; caused by blockage of trabeculae by RBCs in patients with massive hyphema; associated with pupil block

- **Hemolytic glaucoma** – Clogging of trabecular meshwork by macrophages laden with lysed RBC debris

- **Ghost cell glaucoma** – Aphakic or pseudophakic eyes with vitreous haemorrhage

- **Hemosiderotic glaucoma** – Sclerotic changes in trabecular meshwork caused by iron from phagocytosed hemoglobin
Glaucoma associated with intraocular hemorrhage

- **Treatment:**
  - Bed Rest or limited activity
  - Shield affected eye
  - Avoid aspirin or NSAIDs
  - Atropine bid to tid
  - Topical Steriods
  - Anti-glaucoma Medications (Avoid carbonic anhydrase inhibitors with sickle-cell)
  - A/C washout.
**Steroid – induced glaucoma**

- Type of secondary open – angle glaucoma which develops following topical or systemic steroid therapy
- **Etiopathogenesis** –
  - Glycosaminoglycans (GAG) theory
  - Endothelial cell theory
  - Prostaglandin theory
- Symptoms similar to POAG
- Prevented by judicious use of steroids and regular monitoring of IOP
- **Treatment** –
  - Discontinuation of steroids
  - Medical therapy
  - Filtration surgery
Traumatic glaucoma

Mechanisms –
- Inflammatory glaucoma due to iridocyclitis
- Glaucoma due to intraocular hemorrhage
- Lens – induced glaucoma due to swollen lens
- Angle – closure glaucoma due to anterior synechiae
- Epithelial or fibrous growth
- Angle recession (cleavage) glaucoma

Management – Medical therapy with topical 0.5% timolol and oral acetazolamide and surgical intervention according to situation
Glaucoma associated with intraocular tumours

- Malignant melanoma, retinoblastoma
- **Mechanisms** –
  - Trabecular block due to blockage by tumour cells
  - Neovascularization of the angle
  - Venous stasis
  - Angle closure due to forward displacement of iris – lens diaphragm
- **Treatment** – Removal of tumor or Enucleation of the eyeball
Rare condition occurring as complication of any intraocular operation also known as Malignant glaucoma

- Patients with primary angle – closure glaucoma operated for peripheral iridectomy or trabeculectomy
- Markedly raised IOP associated with shallow or absent anterior chamber
- Clinical features includes severe pain and blurring of vision following any intraocular operation
**Ciliary block glaucoma**

- **On examination,**
  - Persistent flat anterior chamber
  - Markedly raised IOP
  - Unresponsiveness or even aggravation by miotics
  - Phakic, aphakic or pseudophakic
- **Treatment –**
  - Medical therapy – 1% atropine drops, acetazolamide, 0.5% timolol maleate eye drops and iv mannitol
  - YAG laser hyaloidotomy
  - Surgical therapy
Other Secondary Glaucomas Associated with Surgery

- Raised IOP with deep anterior chamber in early postoperative period
- Secondary angle – closure glaucoma due to flat anterior chamber.
- Secondary angle – closure glaucoma due to pupil block
- Uveitis, Glaucoma, Hyphema (UGH) Syndrome
Other Secondary Glaucomas Associated with Surgery

- Early Postoperative Glaucoma
  - Transient IOP spike usually resolves in one week. Treat IOP>30 mmHg
  - Usual causes retained viscoelastic, lens material, and inflammation.
  - Consider anti-glaucoma medications (avoid prostaglandin agonists due to proinflammatory traits).
  - Increase steroids to address inflammatory causes.
Other Secondary Glaucomas Associated with Surgery

- Secondary angle – closure glaucoma due to flat anterior chamber.
  - Causes - Over filtering Blebs or leaking surgical wounds.
  - Fix cause or reform anterior chamber.
  - Atropine 1% bid
  - Consider aqueous suppressants (Beta-blockers, CAIs)
Secondary angle – closure glaucoma due to pupil block (e.g. Iris Bombe)

Despite multiple laser peripheral iridotomies, iris bombe developed in this patient with chronic iridocyclitis and central posterior synechiae.
Uveitis, Glaucoma, Hyphema (UGH) Syndrome

Surgery to re-position or replace lens usually solves the problem. May need ACIOL or suture fixated lens.
Thank You