Secondary Glaucomas

By David Luellwitz, D.O
Mercy Health System
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 haemorrhage
- Steroid-induced glaucoma
- Traumatic glaucoma
- Glaucoma-in-aphakia
- Glaucoma associated with intraocular tumours
- Ciliary block glaucoma
Raised IOP secondary to a disorder of crystalline lens

<table>
<thead>
<tr>
<th>Lens induced glaucoma</th>
<th>Lens induced secondary angle closure glaucoma</th>
<th>Phacomorphic glaucoma</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Lens induced secondary open angle glaucoma</td>
<td>Phacotopic glaucoma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Phacolytic glaucoma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Lens particle glaucoma</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Phacoanaphylactic glaucoma</td>
</tr>
</tbody>
</table>
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 iridotomony
- Cataract extraction with implantation of PCIOL
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

- 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
Phacoantigenic 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.
Phacoantigenic glaucoma

- 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).
# Glaucomas due to uveitis

<table>
<thead>
<tr>
<th>Non specific inflammatory glaucoma</th>
<th>Open – angle inflammatory glaucoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Specific hypertensive uveitis syndrome</td>
<td>Angle – closure inflammatory glaucoma</td>
</tr>
<tr>
<td></td>
<td>Fuchs’ uveitis syndrome</td>
</tr>
<tr>
<td></td>
<td>Glaucomatocyclitic crisis</td>
</tr>
</tbody>
</table>
## Open – angle inflammatory glaucoma

<table>
<thead>
<tr>
<th>Mechanism of rise in IOP</th>
<th>Acute open – angle inflammatory glaucoma</th>
<th>Chronic open – angle inflammatory glaucoma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trabecular clogging, trabecular oedema and prostaglandin – induced rise in IOP</td>
<td>Chronic trabeculitis and trabecular scarring</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Clinical features</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Features of acute iridocyclitis associated with raised IOP with open-angle of anterior chamber</td>
<td>Raised IOP, open angle, no active inflammation but signs of previous episode of uveitis present</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Management</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Treatment of iridocyclitis and medical therapy to lower IOP by use of hyperosmotic agents, acetazolamide and beta – blockers eye drops</td>
<td>Medical therapy Trabeculectomy Cyclodestructive procedures</td>
</tr>
</tbody>
</table>
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
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 ischaemia, 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
- 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
Glaucoma associated with iridocorneal endothelial (ICE) syndromes

- **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 oedema 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 haemorrhage**

- **Hyphaema and vitreous haemorrhage**
- **Red cell glaucoma** – Associated with fresh traumatic hyphaema; caused by blockage of trabeculae by RBCs in patients with massive hyphaema; associated with pupil block
- **Haemolytic 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
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 by 0.5% timolol maleate
- Filtration surgery
**Traumatic glaucoma**

* Mechanisms –
  - Inflammatory glaucoma due to iridocyclitis
  - Glaucoma due to intraocular haemorrhage
  - 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 – in aphakia

- 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
- Undiagnosed pre – existing primary open – angle glaucoma
- Steroid – induced glaucoma
- Epithelial ingrowth
- Malignant glaucoma
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** – Enucleation of the eyeball
Ciliary block glaucoma

- Rare condition occurring as complication of any intraocular operation
- 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
Thank You