Unilateral ↑ IOP associated with:

--Intraocular inflammation

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--Secondary glaucoma

*We are going to look at unilateral elevated IOP in each of these clinical scenarios*
Unilateral ↑ IOP associated with:

--Intraocular inflammation

Generally speaking, do you expect an inflamed eye to be hypertensive, or hypotensive?

--Corneal endothelial abnormality

--PPMD--ICE

--Increased episcleral venous pressure

--CCF--SVC syndrome--Sturge-Weber--Orbital inflammation--cellulitis--pseudotumor--TED

--Secondary glaucoma
Unilateral ↑ IOP associated with:

--Intraocular inflammation

*Generally speaking, do you expect an inflamed eye to be **hypertensive**, or **hypotensive**?*

**Hypotensive**

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--CCF--SVC syndrome--Sturge-Weber--Orbital inflammation--cellulitis--pseudotumor--TED

--Secondary glaucoma
Unilateral ↑ IOP associated with:

--Intraocular inflammation

Generally speaking, do you expect an inflamed eye to be hypertensive, or hypotensive?

Hypotensive

Why hypotensive?

--Secondary glaucoma
Unilateral ↑ IOP associated with:

--Intraocular inflammation

Generally speaking, do you expect an inflamed eye to be **hypertensive**, or **hypotensive**?  
**Hypotensive**

--Corneal changes

Why hypotensive?  
Ciliary-body inflammation leads to decreased aqueous production, with subsequent low IOP.

--Increased episcleral venous pressure

--Secondary glaucoma
Unilateral ↑ IOP associated with:

--Intraocular inflammation

Generally speaking, do you expect an inflamed eye to be hypertensive, or hypotensive?
Hypotensive

Why hypotensive?
Ciliary-body inflammation leads to decreased aqueous production, with subsequent low IOP. That said, certain uveitic entities are notorious for elevated IOP, thus rendering it an important clue re the etiology of the inflammation.

--Secondary glaucoma
Unilateral ↑ IOP associated with:

**Intraocular inflammation**

- A very general condition
- A somewhat specific condition
- A specific eponymous condition
- Another specific eponymous condition

**Corneal endothelial abnormality**

**Increased episcleral venous pressure**

**Secondary glaucoma**
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--Secondary glaucoma
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
  - Increased episcleral venous pressure
  - Secondary glaucoma

Both of these present with unilateral AC cell and ↑ IOP. How does one differentiate between them?
Intraocular inflammation

- Anterior uveitis
- Trabeculitis
- Fuchs heterochromic iridocyclitis
- Posner-Schlossman

- Corneal endothelial abnormality

- Increased episcleral venous pressure

Unilateral ↑ IOP associated with:

Both of these present with unilateral AC cell and ↑ IOP. How does one differentiate between them? The severity of the AC reaction is an important clue. In order for a ‘simple’ anterior uveitis to cause ↑ IOP, the inflammatory reaction must be quite severe.

Secondary glaucoma
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
- Increased episcleral venous pressure
- Secondary glaucoma

Both of these present with unilateral AC cell and ↑ IOP. How does one differentiate between them? The severity of the AC reaction is an important clue. In order for a ‘simple’ anterior uveitis to cause ↑ IOP, the inflammatory reaction must be quite severe. In contrast, the cell associated with trabeculitis can be quite mild.
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman
--Corneal endothelial abnormality

--Increased episcleral venous pressure

--Secondary glaucoma

What etiology should come to mind with this?
Herpesvirus infection, especially HSV and VZV
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--Secondary glaucoma

What etiology should come to mind with this? Herpesvirus infection, especially HSV and VZV
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- Corneal endothelial abnormality

- Increased episcleral venous pressure

- Secondary glaucoma

What four anterior uveitis etiologies are notorious for elevating IOP?
1) Herpesvirus (all forms)
2) Toxoplasmosis
3) Sarcoidosis
4) Syphilis

(hints soon forthcoming…)

(Etiologies other than these two, that is)
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--Secondary glaucoma

What four anterior uveitis etiologies are notorious for elevating IOP?
1) Herpesvirus (all forms)
2) this one is a family of infectious agents
3) 
4)
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--Secondary glaucoma

What four anterior uveitis etiologies are notorious for elevating IOP?
1) Herpesvirus (all forms)
2)
3)
4)
What four anterior uveitis etiologies are notorious for elevating IOP?
1) Herpesvirus (all forms)
2) this one is a specific bug
3) 
4)
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--Secondary glaucoma

What four anterior uveitis etiologies are notorious for elevating IOP?
1) Herpesvirus (all forms)
2) Toxoplasmosis
3) 
4)
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- Corneal endothelial abnormality

- Increased episcleral venous pressure

- Secondary glaucoma

What four anterior uveitis etiologies are notorious for elevating IOP?

1) Herpesvirus (all forms)
2) Toxoplasmosis
3) this one is a common noninfectious entity
4)
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--Secondary glaucoma

What four anterior uveitis etiologies are notorious for elevating IOP?
1) Herpesvirus (all forms)
2) Toxoplasmosis
3) Sarcoidosis
4)
Unilateral ↑ IOP associated with:

**Intraocular inflammation**
- **Anterior uveitis**
- Trabeculitis
- Fuchs heterochromic iridocyclitis
- Posner-Schlossman

**Corneal endothelial abnormality**

**Increased episcleral venous pressure**

**Secondary glaucoma**

---

What four anterior uveitis etiologies are notorious for elevating IOP?
1) Herpesvirus (all forms)
2) Toxoplasmosis
3) Sarcoidosis
4) This one is another specific bug
What four anterior uveitis etiologies are notorious for elevating IOP?

1) Herpesvirus (all forms)
2) Toxoplasmosis
3) Sarcoidosis
4) Syphilis

Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--Secondary glaucoma
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
- Increased episcleral venous pressure
- Secondary glaucoma

What four anterior uveitis etiologies are notorious for elevating IOP?
1) Herpesvirus (all forms)
2) Toxoplasmosis
3) Sarcoidosis
4) Syphilis

Which of these can present with granulomatous-appearing KP?
All of them
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--Secondary glaucoma

What four anterior uveitis etiologies are notorious for elevating IOP?
1) Herpesvirus (all forms)
2) Toxoplasmosis
3) Sarcoidosis
4) Syphilis

Which of these can present with granulomatous-appearing KP?
All of them
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- Corneal endothelial abnormality

- Increased episcleral venous pressure

- Secondary glaucoma

What four anterior uveitis etiologies are notorious for elevating IOP?
1) Herpesvirus (all forms)
2) Toxoplasmosis
3) Sarcoidosis
4) Syphilis

Which can present with stellate KP?

All but sarcoid (and don’t forget about FHI)
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--Secondary glaucoma

What four anterior uveitis etiologies are notorious for elevating IOP?

1) Herpesvirus (all forms)
2) Toxoplasmosis
3) Syphilis
4) Sarcoidosis

Which can present with stellate KP?
All but sarcoid (and don’t forget about FHI)

FHI causes stellate KP too
Unilateral ↑ IOP associated with:

-- Intraocular inflammation
  -- Anterior uveitis
  -- Trabeculitis
  -- Fuchs heterochromic iridocyclitis

-- Corneal endothelial abnormality

-- Increased episcleral venous pressure

-- Secondary glaucoma

**What is the usual treatment regimen for anterior uveitis accompanied by increased IOP?**

-- Topical steroids

-- Cycloplegia

-- Anti-infectives if bug is known

-- +/- ocular hypotensives
<table>
<thead>
<tr>
<th>Intraocular inflammation</th>
<th>Posterior uveitis</th>
<th>Anterior uveitis</th>
<th>Trabeculitis</th>
<th>Fuchs heterochromic iridocyclitis</th>
<th>Posner-Schlossman</th>
</tr>
</thead>
<tbody>
<tr>
<td>Corneal endothelial abnormality</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

- Increased episcleral venous pressure

- Unilateral ↑ IOP associated with:
  - Intraocular inflammation
    - Anterior uveitis
    - Trabeculitis
    - Fuchs heterochromic iridocyclitis
    - Posner-Schlossman
  - Corneal endothelial abnormality

**What is the usual treatment regimen for anterior uveitis accompanied by increased IOP?**

- Topical steroids
- Cycloplegia
- Anti-infectives if bug is known
- +/- ocular hypotensives

- Secondary glaucoma
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
- Increased episcleral venous pressure
- CCF—SVC syndrome—Sturge-Weber
- Orbital inflammation
  - Cellulitis
  - Pseudotumor
  - TED
- Secondary glaucoma

What is the usual treatment regimen for anterior uveitis accompanied by increased IOP?
- Topical steroids
- Cycloplegia
- Anti-infectives if bug is known
- +/- ocular hypotensives

What two classes of topical hypotensives should probably be avoided?
- Miotics
- Prostaglandin analogues/prostamides

Both are potentially inflammogenic.
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--Secondary glaucoma

What is the usual treatment regimen for anterior uveitis accompanied by increased IOP?
--Topical steroids
--Cycloplegia
--Anti-infectives if bug is known
--+/- ocular hypotensives

What two classes of topical hypotensives should probably be avoided?
--Miotics
--Prostaglandin analogues
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
- Increased episcleral venous pressure
- CCF
- SVC syndrome
- Sturge-Weber
- Orbital inflammation
  - Cellulitis
  - Pseudotumor
  - TED
- Secondary glaucoma

What is the usual treatment regimen for anterior uveitis accompanied by increased IOP?
- Topical steroids
- Cycloplegia
- Anti-infectives if bug is known
- +/- ocular hypotensives

What two classes of topical hypotensives should probably be avoided?
- Miotics
- Prostaglandin analogues

Why should these be avoided?

Unilateral ↑ IOP associated with:
What is the usual treatment regimen for anterior uveitis accompanied by increased IOP?
- Topical steroids
- Cycloplegia
- Anti-infectives if bug is known
- +/- ocular hypotensives

What two classes of topical hypotensives should probably be avoided?
- Miotics
- Prostaglandin analogues

Why should these be avoided?
Both are potentially inflammogenic
What is the usual treatment regimen for anterior uveitis accompanied by increased IOP?

- **Topical steroids**
- **Cycloplegia**
- **Anti-infectives if bug is known**
- +/- ocular hypotensives

In what way might steroid therapy complicate the IOP issue?

Recall that some pts are steroid responders, meaning their IOP increases secondary to steroid therapy. Thus, when a uveitis pt (under treatment) develops increased IOP, it could mean either:

- a) their uveitis is not well controlled, and therefore the steroid should be increased;
- b) they are a steroid responder, and their steroid should be decreased (or stopped).

Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
- Increased episcleral venous pressure
- Secondary glaucoma

Secondary glaucoma
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--Secondary glaucoma

What is the usual treatment regimen for anterior uveitis accompanied by increased IOP?

--Topical steroids
  --Cycloplegia
  --Anti-infectives if bug is known
  --/+ ocular hypotensives

In what way might steroid therapy complicate the IOP issue?

Recall that some pts are steroid responders, meaning their IOP increases secondary to steroid therapy. Thus, when a uveitis pt (under treatment) develops increased IOP, it could mean either:

a)

b)
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
- Increased episcleral venous pressure
- Secondary glaucoma

What is the usual treatment regimen for anterior uveitis accompanied by increased IOP?

- Topical steroids
  - Cyclopentolate (Cyclomed)
  - Anti-infectives if bug is known
  - +/- ocular hypotensives

In what way might steroid therapy complicate the IOP issue?

Recall that some pts are steroid responders, meaning their IOP increases secondary to steroid therapy. Thus, when a uveitis pt (under treatment) develops increased IOP, it could mean either:

a) their uveitis is not well controlled, and therefore the steroid should be increased; or

b)
### Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
- Increased episcleral venous pressure
- Secondary glaucoma

### What is the usual treatment regimen for anterior uveitis accompanied by increased IOP?

- **Topical steroids**
  - Cycles
  - Anti-infectives if bug is known
  - +/- ocular hypotensives

### In what way might steroid therapy complicate the IOP issue?

Recall that some pts are **steroid responders**, meaning their IOP increases secondary to steroid therapy. Thus, when a uveitis pt (under treatment) develops increased IOP, it could mean either:

a) their uveitis is not well controlled, and therefore the steroid should be **increased**; or

b) they are a steroid responder, and their steroid should be **decreased** (or even stopped).
What is the usual treatment regimen for anterior uveitis accompanied by increased IOP?

- **Topical steroids**
- Cycloplegia
- Anti-infectives if bug is known
- +/- ocular hypotensives

In what way might steroid therapy complicate the IOP issue?

Recall that some pts are **steroid responders**, meaning their IOP increases secondary to steroid therapy. Thus, when a uveitis pt (under treatment) develops increased IOP, it could mean either:

a) their uveitis is not well controlled, and therefore the steroid should be increased; or
b) they are a steroid responder, and their steroid should be decreased (or even stopped)

So how do you know whether steroids are the solution to, or the cause of, ocular hypertension in uveitis?
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--Secondary glaucoma

What is the usual treatment regimen for anterior uveitis accompanied by increased IOP?

--Topical steroids
  --Cycloplegia
  --Anti-infectives if bug is known
  -- +/- ocular hypotensives

In what way might steroid therapy complicate the IOP issue?

Recall that some pts are steroid responders, meaning their IOP increases secondary to steroid therapy. Thus, when a uveitis pt (under treatment) develops increased IOP, it could mean either:

a) their uveitis is not well controlled, and therefore the steroid should be increased; or
b) they are a steroid responder, and their steroid should be decreased (or even stopped)

So how do you know whether steroids are the solution to, or the cause of, ocular hypertension in uveitis?

As a rule, it takes at least 2 weeks before steroid-induced ocular hypertension occurs. Thus, elevated IOP within this time frame indicates inadequate uveitis control, and the steroid dose should be increased.
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
- Increased episcleral venous pressure
- Secondary glaucoma

What is the usual treatment regimen for anterior uveitis accompanied by increased IOP?

- Topical steroids
  - Cycloplegia
  - Anti-infectives if bug is known
  - +/- ocular hypotensives

In what way might steroid therapy complicate the IOP issue?

Recall that some pts are **steroid responders**, meaning their IOP increases secondary to steroid therapy. Thus, when a uveitis pt (under treatment) develops increased IOP, it could mean either:

a) their uveitis is not well controlled, and therefore the steroid should be **increased**; or

b) they are a steroid responder, and their steroid should be **decreased** (or even stopped)

So how do you know whether steroids are the solution to, or the cause of, ocular hypertension in uveitis?

As a rule, it takes at least 2 weeks before steroid-induced ocular hypertension occurs. Thus, elevated IOP within this time frame indicates inadequate uveitis control, and the steroid dose should be increased.

On the other hand, if IOP spikes **after** the uveitis is controlled, consideration should be given to tapering the steroid and/or adding an aqueous suppressant to the treatment regimen.
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --**Fuchs heterochromic iridocyclitis**
  --Posner-Schlossman

--Corneal endothelial abnormality

*What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)*
Unilateral ↑ IOP associated with:

---Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --**Fuchs heterochromic iridocyclitis**
  --Posner-Schlossman

---Corneal endothelial abnormality

*What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)*
Heterochromia iridis, cataract, and stellate KP

---Increased episcleral venous pressure
---CCF--SVC syndrome--Sturge-Weber--Orbital inflammation--cellulitis--pseudotumor--TED

---Secondary glaucoma
---Lens-related: Phacolytic, phacomorphic--PXS--PDS
Unilateral ↑ IOP associated with:

FHI: Heterochromia
Unilateral ↑ IOP associated with:

FHI: Note the cataract
Unilateral ↑ IOP associated with:

FHI: Stellate KP
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)

Heterochromia iridis, cataract, and stellate KP

Is the affected eye the darker eye or the lighter eye?
Unilateral ↑ IOP associated with:

**--Intraocular inflammation**

- Anterior uveitis
- Trabeculitis
- **Fuchs heterochromic iridocyclitis**
- Posner-Schlossman

**Corneal endothelial abnormality**

What exam findings comprise the ‘classic triad’ of FHI? *(Hint: Elevated IOP is not one of them.)*

**Heterochromia iridis**, cataract, and stellate KP

Is the affected eye the **darker eye** or the **lighter eye**?

The lighter (with one exception)
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

Is the affected eye the darker eye or the lighter eye? The lighter (with one exception)

What is the exception; ie, under what circumstances is the darker eye the one with FHI?
In individuals with light-blue eyes…
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)

Heterochromia iridis, cataract, and stellate KP

Is the affected eye the darker eye or the lighter eye?
The lighter (with one exception)

What is the exception; ie, under what circumstances is the darker eye the one with FHI?
In individuals with light-blue eyes…the iris atrophy stemming from the FHI process will make visible the darkly-pigmented epithelium of the posterior iris, thus making the eye appear darker
Unilateral ↑ IOP associated with:

--Intraocular inflammation
   --Anterior uveitis
   --Trabeculitis
   --Fuchs heterochromic iridocyclitis
   --Posner-Schlossman

--Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

The KP in FHI have a couple of other notable characteristics—what are they?
--
--
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --**Fuchs heterochromic iridocyclitis**
  --Posner-Schlossman

--Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and **stellate KP**

The KP in FHI have a couple of other notable characteristics—what are they?
--They may be interconnected by lines described as ‘**two words**’.
--
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and **stellate KP**

The KP in FHI have a couple of other notable characteristics—what are they?
-- They may be interconnected by lines described as ‘lacy tendrils’
--
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)

Heterochromia iridis, cataract, and stellate KP

The KP in FHI have a couple of other notable characteristics—what are they?
  --They may be interconnected by lines described as ‘lacy tendrils’
  --They are diffusely scattered (as opposed to being concentrated in Arlt’s triangle, as is the case in most anterior uveitides)
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --**Fuchs heterochromic iridocyclitis**
  --Posner-Schlossman

--Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and **stellate KP**

The KP in FHI have a couple of other notable characteristics—what are they?
--They may be interconnected by lines described as ‘lacy tendrils’
--They are diffusely scattered (as opposed to being concentrated in Arlt’s triangle, as is the case in most anterior uveitides)
Unilateral ↑ IOP associated with:

FHI: Stellate KP. *Note the diffuse distribution*
Unilateral ↑ IOP associated with:

---Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

---Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

The KP in FHI have a couple of other notable characteristics—what are they?
--They may be interconnected by lines described as ‘lacy tendrils’
--They are diffusely scattered (as opposed to being concentrated in Arlt’s triangle, as is the case in most anterior uveitides)

Where/what is Arlt’s triangle?

Arlt’s triangle
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and **stellate KP**

The KP in FHI have a couple of other notable characteristics—what are they?
--They may be interconnected by lines described as ‘lacy tendrils’
--They are diffusely scattered (as opposed to being concentrated in Arlt’s triangle, as is the case in most anterior uveitides)

Where/what is Arlt’s triangle?
It’s an equilateral triangle with its apex at the corneal center and base near the inferior border of the cornea.
Unilateral ↑ IOP associated with:

KP (in sarcoidosis) concentrated in Arlt’s triangle
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --**Fuchs heterochromic iridocyclitis**
  --Posner-Schlossman

--Corneal endothelial abnormality

*What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)*
Heterochromia iridis, cataract, and stellate KP

*How common is glaucoma in FHI?*
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How common is glaucoma in FHI?
It develops in about 25-50% of cases
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --**Fuchs heterochromic iridocyclitis**
  --Posner-Schlossman

--Corneal endothelial abnormality

*What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)*
Heterochromia iridis, cataract, and stellate KP

*How common is glaucoma in FHI?*
It develops in about 25-50% of cases

*Is the angle in FHI glaucoma open, or is it closed?*
Unilateral ↑ IOP associated with:

- **Intraocular inflammation**
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- **Corneal endothelial abnormality**

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How common is glaucoma in FHI?
It develops in about 25-50% of cases

Is the angle in FHI glaucoma open, or is it closed?
Open
Unilateral ↑ IOP associated with:

---Intraocular inflammation

---Anterior uveitis
---Trabeculitis
---**Fuchs heterochromic iridocyclitis**
---Posner-Schlossman

---Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

**How common is glaucoma in FHI?**
It develops in about 25-50% of cases

Speaking of the angle in FHI…It has two characteristics that are unusual, and may aid in making the diagnosis:

---
---

Is the angle in FHI glaucoma open, or is it closed?
Open
Unilateral ↑ IOP associated with:

- **Intraocular inflammation**
  - Anterior uveitis
  - Trabeculitis
  - **Fuchs heterochromic iridocyclitis**
  - Posner-Schlossman

- Corneal endothelial abnormality

*What exam findings comprise the ‘classic triad’ of FHI?* (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

*How common is glaucoma in FHI?*
It develops in about 25-50% of cases

*Speaking of the angle in FHI...It has two characteristics that are unusual, and may aid in making the diagnosis:*
  - Despite the chronic nature of the iridocyclitis in FHI,
    - four words (and an abb.) never develop
  - Neovascularization of the angle (NVA)
    - three words (and an abb.) is common, but does not lead to angle closure
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How common is glaucoma in FHI?
It develops in about 25-50% of cases

Speaking of the angle in FHI…It has two characteristics that are unusual, and may aid in making the diagnosis:
-- Despite the chronic nature of the iridocyclitis in FHI, peripheral anterior synechiae (PAS) never develop
-- Neovascularization of the angle (NVA) is common, but does not lead to angle closure
Unilateral ↑ IOP associated with:

Fuchs heterochromic iridocyclitis. Fine vessels (arrows) are seen crossing the TM. This NVA is not accompanied by a fibrovascular membrane and does not result in PAS formation and subsequent secondary angle closure.
Unilateral ↑ IOP associated with:

- **Intraocular inflammation**
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How common is glaucoma in FHI? It develops in about 25-50% of cases

Speaking of the angle in FHI...It has two characteristics that are unusual, and may aid in making the diagnosis:
-- Despite the chronic nature of the iridocyclitis in FHI, *peripheral anterior synechiae (PAS)* never develop
-- **Neovascularization of the angle (NVA)** is common, but does not lead to angle closure

The NVA vessels in FHI rupture easily, and it not uncommon for FHI pts to develop a small hyphema when the paracentesis wound is made at the start of cataract surgery. **What is the eponymous name for this classic finding?**
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How common is glaucoma in FHI? It develops in about 25-50% of cases

Speaking of the angle in FHI... It has two characteristics that are unusual, and may aid in making the diagnosis:
-- Despite the chronic nature of the iridocyclitis in FHI, peripheral anterior synechiae (PAS) never develop
-- Neovascularization of the angle (NVA) is common, but does not lead to angle closure

The NVA vessels in FHI rupture easily, and it not uncommon for FHI pts to develop a small hyphema when the paracentesis wound is made at the start of cataract surgery. What is the eponymous name for this classic finding? Amsler’s sign
Unilateral ↑ IOP associated with:

--- Intraocular inflammation
  --- Anterior uveitis
  --- Trabeculitis
  --- Fuchs heterochromic iridocyclitis
  --- Posner-Schlossman

--- Corneal endothelial abnormality

**What exam findings comprise the ‘classic triad’ of FHI?** *(Hint: Elevated IOP is not one of them.)*
Heterochromia iridis, cataract, and stellate KP

**How common is glaucoma in FHI?**
It develops in about 25-50% of cases

**What is the etiology of FHI?**
Unilateral ↑ IOP associated with:

- **Intraocular inflammation**
  - Anterior uveitis
  - Trabeculitis
  - **Fuchs heterochromic iridocyclitis**
  - Posner-Schlossman

- **Corneal endothelial abnormality**

**What exam findings comprise the ‘classic triad’ of FHI?** *(Hint: Elevated IOP is not one of them.)*
Heterochromia iridis, cataract, and stellate KP

**How common is glaucoma in FHI?**
It develops in about 25-50% of cases

**What is the etiology of FHI?**
It is uncertain at this time. Four infectious entities have been suggested: protozoan, virus, virus, and **virus**.
Unilateral ↑ IOP associated with:

**Intraocular inflammation**
- Anterior uveitis
- Trabeculitis
- **Fuchs heterochromic iridocyclitis**
- Posner-Schlossman

**Corneal endothelial abnormality**

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How common is glaucoma in FHI?
It develops in about 25-50% of cases

What is the etiology of FHI?
It is uncertain at this time. Four infectious entities have been suggested: Toxoplasmosis; HSV; CMV; and rubella.
Unilateral ↑ IOP associated with:

**Intraocular inflammation**
- Anterior uveitis
- Trabeculitis
- *Fuchs heterochromic iridocyclitis*
- Posner-Schlossman

**Corneal endothelial abnormality**

*What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)*
Heterochromia iridis, cataract, and stellate KP

*How common is glaucoma in FHI?*
It develops in about 25-50% of cases

*What is the etiology of FHI?*
It is uncertain at this time. Four infectious entities have been suggested: Toxoplasmosis; HSV; CMV; and rubella. As of now, the preponderance of the evidence points to **rubella** but it remains unproven.
Unilateral ↑ IOP associated with:

---Intraocular inflammation

---Anterior uveitis
---Trabeculitis
---Fuchs heterochromic iridocyclitis
---Posner-Schlossman

---Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How common is glaucoma in FHI?
It develops in about 25-50% of cases

What is the etiology of FHI?
It is uncertain at this time. Four infectious entities have been suggested: Toxoplasmosis; HSV; CMV; and rubella. As of now, the preponderance of the evidence points to rubella, but it remains unproven.
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchsheterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How common is glaucoma in FHI?
It develops in about 25-50% of cases

What is the etiology of FHI?
It is uncertain at this time. Four infectious entities have been suggested: Toxoplasmosis; HSV; CMV; and rubella. As of now, the preponderance of the evidence points to rubella, but it remains unproven.

Who is the typical pt?
Unilateral ↑ IOP associated with:

---Intraocular inflammation
  ---Anterior uveitis
  ---Trabeculitis
  ---Fuchs heterochromic iridocyclitis
  ---Posner-Schlossman

---Corneal endothelial abnormality

**What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)**
Heterochromia iridis, cataract, and stellate KP

**How common is glaucoma in FHI?**
It develops in about 25-50% of cases

**What is the etiology of FHI?**
It is uncertain at this time. Four infectious entities have been suggested: Toxoplasmosis; HSV; CMV; and rubella. As of now, the preponderance of the evidence points to rubella, but it remains unproven.

**Who is the typical pt?**
A middle-aged adult
Unilateral ↑ IOP associated with:

---Intraocular inflammation
  ---Anterior uveitis
  ---Trabeculitis
  ---**Fuchs heterochromic iridocyclitis**
  ---Posner-Schlossman

---Corneal endothelial abnormality

*What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)*
Heterochromia iridis, cataract, and stellate KP

*How common is glaucoma in FHI?*
It develops in about 25-50% of cases

*What is the etiology of FHI?*
It is uncertain at this time. Four infectious entities have been suggested: Toxoplasmosis; HSV; CMV; and rubella. As of now, the preponderance of the evidence points to rubella, but it remains unproven.

*Who is the typical pt?*
A middle-aged adult

*Is there a gender predilection?*
Unilateral ↑ IOP associated with:

**Intraocular inflammation**
- Anterior uveitis
- Trabeculitis
- **Fuchs heterochromic iridocyclitis**
- Posner-Schlossman

**Corneal endothelial abnormality**

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How common is glaucoma in FHI?
It develops in about 25-50% of cases

What is the etiology of FHI?
It is uncertain at this time. Four infectious entities have been suggested: Toxoplasmosis; HSV; CMV; and rubella. As of now, the preponderance of the evidence points to rubella, but it remains unproven.

Who is the typical pt?
A middle-aged adult

Is there a gender predilection?
No
Unilateral ↑ IOP associated with:

--- Intraocular inflammation
  -- Anterior uveitis
  -- Trabeculitis
  -- Fuchs heterochromic iridocyclitis
  -- Posner-Schlossman

--- Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How well does FHI respond to steroid therapy?

A middle-aged adult

Is there a gender predilection?
No
Unilateral ↑ IOP associated with:

- **Intraocular inflammation**
  - Anterior uveitis
  - Trabeculitis
  - **Fuchs heterochromic iridocyclitis**
  - Posner-Schlossman

- Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How well does FHI respond to steroid therapy?
Rather poorly--AC cell is notoriously difficult to eradicate in FHI (as is the vitreous cell which frequently occurs)

A middle-aged adult

Is there a gender predilection?
No
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --**Fuchs heterochromic iridocyclitis**
  --Posner-Schlossman

--Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How well does FHI respond to steroid therapy?
Rather poorly--AC cell is notoriously difficult to eradicate in FHI (as is the vitreous cell which frequently occurs)

If the inflammation fails to respond to topical steroids, should more aggressive therapies be pursued?

Is there a gender predilection?
No
Unilateral ↑ IOP associated with:

**Intraocular inflammation**
- Anterior uveitis
- Trabeculitis
- **Fuchs heterochromic iridocyclitis**
- Posner-Schlossman

**Corneal endothelial abnormality**

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How well does FHI respond to steroid therapy?
Rather poorly--AC cell is notoriously difficult to eradicate in FHI (as is the vitreous cell which frequently occurs)

If the inflammation fails to respond to topical steroids, should more aggressive therapies be pursued?
Generally no. In fact, most pts require no anti-inflammatory tx of any sort (including steroids).

Who is the typical pt?
A middle-aged adult

Is there a gender predilection?
No
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How well does FHI respond to steroid therapy?
Rather poorly--AC cell is notoriously difficult to eradicate in FHI (as is the vitreous cell which frequently occurs)

If the inflammation fails to respond to topical steroids, should more aggressive therapies be pursued?
Generally no. In fact, most pts require no anti-inflammatory tx of any sort (including steroids). Instead, the pt should be monitored for the development of glaucoma (first-line treatment: class of glaucoma med) and cataract, which should be removed when visually or medically significant. In rare cases, vitrectomy is required to clear significant vitreous opacities.

Who is the typical pt?
A middle-aged adult

Is there a gender predilection?
No
Unilateral ↑ IOP associated with:

--Intraocular inflammation
---Anterior uveitis
---Trabeculitis
---Fuchs heterochromic iridocyclitis
---Posner-Schlossman

--Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How well does FHI respond to steroid therapy?
Rather poorly--AC cell is notoriously difficult to eradicate in FHI (as is the vitreous cell which frequently occurs)

If the inflammation fails to respond to topical steroids, should more aggressive therapies be pursued?
Generally no. In fact, most pts require no anti-inflammatory tx of any sort (including steroids). Instead, the pt should be monitored for the development of glaucoma (first-line treatment: aqueous suppressants) and cataract, which should be removed when visually or medically significant. In rare cases, vitrectomy is required to clear significant vitreous opacities.

A middle-aged adult

Is there a gender predilection?
No
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --**Fuchs heterochromic iridocyclitis**
  --Posner-Schlossman

--Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP

How well does FHI respond to steroid therapy?
Rather poorly--AC cell is notoriously difficult to eradicate in FHI (as is the vitreous cell which frequently occurs)

If the inflammation fails to respond to topical steroids, should more aggressive therapies be pursued?
Generally no. In fact, most pts require no anti-inflammatory tx of any sort (including steroids). Instead, the pt should be monitored for the development of glaucoma (first-line treatment: aqueous suppressants) and **cataract, which should be removed when visually or medically significant**. In rare cases, vitrectomy is required to clear significant vitreous opacities.

Is there a gender predilection?
No

Is cataract surgery in FHI associated with an increased risk of intraoperative complications?
Unilateral ↑ IOP associated with:

<table>
<thead>
<tr>
<th>Intraocular inflammation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anterior uveitis</td>
</tr>
<tr>
<td>Trabeculitis</td>
</tr>
<tr>
<td><strong>Fuchs heterochronic iridocyclitis</strong></td>
</tr>
<tr>
<td>Posner-Schlossman</td>
</tr>
</tbody>
</table>

Corneal endothelial abnormality

What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)

- Heterochromia iridis
- Cataract
- Stellate KP

How well does FHI respond to steroid therapy?

Rather poorly—AC cell is notoriously difficult to eradicate in FHI (as is the vitreous cell which frequently occurs).

If the inflammation fails to respond to topical steroids, should more aggressive therapies be pursued?

Generally no. In fact, most pts require no anti-inflammatory tx of any sort (including steroids). Instead, the pt should be monitored for the development of glaucoma (first-line treatment: aqueous suppressants) and cataract, which should be removed when visually or medically significant. In rare cases, vitrectomy is required to clear significant vitreous opacities.

Is cataract surgery in FHI associated with an increased risk of intraoperative complications?

No
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - **Posner-Schlossman**

- Corneal endothelial abnormality

- Increased episcleral venous pressure

- CCF

- SVC syndrome

- Sturge-Weber

- Orbital inflammation
  - Cellulitis
  - Pseudotumor
  - TED

- Secondary glaucoma
  - Lens-related: Phacolytic, phacomorphic

**What is the noneponymous name for Posner-Schlossman?**
Unilateral ↑ IOP associated with:

--- Intraocular inflammation
--- Anterior uveitis
--- Trabeculitis
--- Fuchs heterochromic iridocyclitis
--- Posner-Schlossman

--- Corneal endothelial abnormality

--- Increased episcleral venous pressure
--- CCF
--- SVC syndrome
--- Sturge-Weber
--- Orbital inflammation
--- Cellulitis
--- Pseudotumor
--- TED

--- Secondary glaucoma

--- What is the noneponymous name for Posner-Schlossman?
- Glaucomatocyclitic crisis
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- Corneal endothelial abnormality

- Increased episcleral venous pressure
  - CCF
  - SVC syndrome
  - Sturge-Weber
  - Orbital inflammation
cellulitis
pseudotumor
TED

- Secondary glaucoma

---

**What is the noneponymous name for Posner-Schlossman?**
Glaucomatocyclitic crisis

**Who is the typical pt?**
Unilateral ↑ IOP associated with:

---Intraocular inflammation
  ---Anterior uveitis
  ---Trabeculitis
  ---Fuchs heterochromic iridocyclitis
  ---**Posner-Schlossman**

---Corneal endothelial abnormality

---Increased episcleral venous pressure
  ---CCF
  ---SVC syndrome
  ---Sturge-Weber
  ---Orbital inflammation
cellulitis, pseudotumor, TED

---Secondary glaucoma

---Intraocular inflammation
  ---Anterior uveitis
  ---Trabeculitis
  ---Fuchs heterochromic iridocyclitis

**What is the noneponymous name for Posner-Schlossman?**
Glaucmatocyclitic crisis

**Who is the typical pt?**
An adult age 20-50
Unilateral ↑ IOP associated with:

--- Intraocular inflammation
  --- Anterior uveitis
  --- Trabeculitis
  --- Fuchs heterochromic iridocyclitis
  --- **Posner-Schlossman**

--- Corneal endothelial abnormality

--- Increased episcleral venous pressure
  --- CCF
  --- SVC syndrome
  --- Sturge-Weber
  --- Orbital inflammation
    --- Cellulitis
    --- Pseudotumor
    --- TED

--- Secondary glaucoma

--- Intraocular inflammation
  --- Anterior uveitis
  --- Trabeculitis
  --- Fuchs heterochromic iridocyclitis
  --- **Posner-Schlossman**

**What is the nonpronymous name for Posner-Schlossman?**
Glaucotocyclitic crisis

**Who is the typical pt?**
An adult age 20-50

**Does the inflammatory component tend to be mild, or severe?**
Unilateral ↑ IOP associated with:

**Intraocular inflammation**
- Anterior uveitis
- Trabeculitis
- Fuchs heterochromic iridocyclitis
- **Posner-Schlossman**

**Corneal endothelial abnormality**

**Increased episcleral venous pressure**

**Secondary glaucoma**

---

*What is the noneponymous name for Posner-Schlossman?*
Glaucomatocyclitic crisis

*Who is the typical pt?*
An adult age 20-50

*Does the inflammatory component tend to be mild, or severe?*
Mild
Unilateral ↑ IOP associated with:

--- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - **Posner-Schlossman**

--- Corneal endothelial abnormality

--- Increased episcleral venous pressure
  - CCF
  - SVC syndrome
  - Sturge-Weber
  - Orbital inflammation
    - Cellulitis
    - Pseudotumor
    - TED

--- Secondary glaucoma

--- What is the noneponymous name for Posner-Schlossman?
  - Glaucomatocyclitic crisis

--- Who is the typical pt?
  - An adult age 20-50

--- Does the inflammatory component tend to be mild, or severe?
  - Mild

--- Does the IOP elevation tend to be mild, or severe?
  - Severe
Unilateral ↑ IOP associated with:

---Intraocular inflammation
  ---Anterior uveitis
  ---Trabeculitis
  ---Fuchs heterochromic iridocyclitis
  ---**Posner-Schlossman**

---Corneal endothelial abnormality

---Increased episcleral venous pressure
  ---CCF
  ---SVC syndrome
  ---Sturge-Weber
  ---Orbital inflammation
  ---cellulitis
  ---pseudotumor
  ---TED

---Secondary glaucoma

---Intraocular inflammation
  ---Anterior uveitis
  ---Trabeculitis
  ---Fuchs heterochromic iridocyclitis

---Unilateral ↑ IOP associated with:

-What is the non-eponymus name for Posner-Schlossman?
  Glaucomatocyclitic crisis

Who is the typical pt?
  An adult age 20-50

Does the inflammatory component tend to be mild, or severe?
  Mild

Does the IOP elevation tend to be mild, or severe?
  Severe
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --**Posner-Schlossman**

---Corneal endothelial abnormality

---Increased episcleral venous pressure
  --PPMD
  --ICE

---Secondary glaucoma

---What is the noneponymous name for Posner-Schlossman?
  Glaucomatocyclitic crisis

---Who is the typical pt?
  An adult age 20-50

---Does the inflammatory component tend to be mild, or severe?
  Mild

---Does the IOP elevation tend to be mild, or severe?
  Severe

  How severe?
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --**Posner-Schlossman**

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--CCF
--SVC syndrome
--Sturge-Weber
--Orbital inflammation

-Cellulitis
--Pseudotumor
--**TED**

--Secondary glaucoma

What is the noneponymous name for Posner-Schlossman?
Glaucoumatocyclitic crisis

Who is the typical pt?
An adult age 20-50

Does the inflammatory component tend to be mild, or severe?
Mild

Does the IOP elevation tend to be mild, or severe?
Severe

How severe?
IOP in the 40-60 range is typical
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --**Posner-Schlossman**

--Corneal endothelial abnormality

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
cellulitis, pseudotumor, TED

--Secondary glaucoma

*What is the noneponymous name for Posner-Schlossman?*
Glaucomatocyclitic crisis

*Who is the typical pt?*
An adult age 20-50

*Does the inflammatory component tend to be mild, or severe?*
Mild

*Does the IOP elevation tend to be mild, or severe?*
Severe

*Is the angle open, or closed?*
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --**Posner-Schlossman**

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--CCF--SVC syndrome--Sturge-Weber--Orbital inflammation--cellulitis--pseudotumor--TED

--Secondary glaucoma

**What is the noneponymous name for Posner-Schlossman?**
Glaucomatocyclitic crisis

**Who is the typical pt?**
An adult age 20-50

**Does the inflammatory component tend to be mild, or severe?**
Mild

**Does the IOP elevation tend to be mild, or severe?**
Severe

**Is the angle open, or closed?**
Open
Unilateral ↑ IOP associated with:

**--Intraocular inflammation**

- Anterior uveitis
- Trabeculitis
- Fuchs heterochromic iridocyclitis

- **Posner-Schlossman**

**Corneal endothelial abnormality**

- ICE

**Increased episcleral venous pressure**

- CCf
- SVC syndrome

**Sturge-Weber**

**Orbital inflammation**

- Cellulitis
- Pseudotumor

**TED**

**Secondary glaucoma**

---

- What is the noneponymous name for Posner-Schlossman?
  - Glaucomatocyclitic crisis

- Who is the typical pt?
  - An adult age 20-50

- Does the inflammatory component tend to be mild, or severe?
  - Mild

- Does the IOP elevation tend to be mild, or severe?
  - Severe

- Is the angle open, or closed?
  - Open

- How long do the crises last?
  - Hours to weeks

- Do they recur?
  - Yes
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --**Posner-Schlossman**

--Corneal endothelial abnormality

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
  --cellulitis
  --pseudotumor
  --TED

--Secondary glaucoma

---What is the noneponymous name for Posner-Schlossman?
  Glaucomatocyclitic crisis

---Who is the typical pt?
  An adult age 20-50

---Does the inflammatory component tend to be mild, or severe?
  Mild

---Does the IOP elevation tend to be mild, or severe?
  Severe

---Is the angle open, or closed?
  Open

---How long do the crises last?
  Hours to weeks
Unilateral ↑ IOP associated with:

---Intraocular inflammation
  ---Anterior uveitis
  ---Trabeculitis
  ---Fuchs heterochromic iridocyclitis
  ---**Posner-Schlossman**

---Corneal endothelial abnormality

---Increased episcleral venous pressure
  ---CCF
  ---SVC syndrome
  ---Sturge-Weber
  ---Orbital inflammation
    ---cellulitis
    ---pseudotumor
    ---TED

---Secondary glaucoma

---What is the noneponymous name for Posner-Schlossman?
  - Glaucomatocyclitic crisis

---Who is the typical pt?
  - An adult age 20-50

---Does the inflammatory component tend to be mild, or severe?
  - Mild

---Does the IOP elevation tend to be mild, or severe?
  - Severe

---Is the angle open, or closed?
  - Open

---How long do the crises last?
  - Hours to weeks

---Do they recur?
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --**Posner-Schlossman**

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--PPMD--ICE

--CCF--SVC syndrome--Sturge-Weber--Orbital inflammation--cellulitis--pseudotumor--TED

--Secondary glaucoma

---What is the noneponymous name for Posner-Schlossman?
  Glaucomatocyclitic crisis

---Who is the typical pt?
  An adult age 20-50

---Does the inflammatory component tend to be mild, or severe?
  Mild

---Does the IOP elevation tend to be mild, or severe?
  Severe

---Is the angle open, or closed?
  Open

---How long do the crises last?
  Hours to weeks

---Do they recur?
  Yes
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation

--Secondary glaucoma

What is the noneponymous name for Posner-Schlossman?
Glaucomatocyclitic crisis

Who is the typical pt?
An adult age 20-50

Does the inflammatory component tend to be mild, or severe?
Mild

Does the IOP elevation tend to be mild, or severe?
Severe

Is the angle open, or closed?
Open

How long do the crises last?
Hours to weeks

Do they recur?
Yes
Unilateral ↑ IOP associated with:

--Intraocular inflammation
--Anterior uveitis
--Trabeculitis
--Fuchs heterochromic iridocyclitis
--**Posner-Schlossman**

--Corneal endothelial abnormality

--Increased episcleral venous pressure

--CCF--SVC syndrome--Sturge-Weber--Orbital inflammation

--Cellulitis--Pseudotumor--TED

--Secondary glaucoma

**What is the noneponymous name for Posner-Schlossman?**
Glaucamatocyclitic crisis

**Who is the typical pt?**
An adult age 20-50

**Does the inflammatory component tend to be mild, or severe?**
Mild

**Does the IOP elevation tend to be mild, or severe?**
Severe

**Is the angle open, or closed?**
Open

**How long do the crises last?**
Hours to weeks

**Do they recur?**
Yes

**What is/are the presenting complaint(s)?**
Unilateral pain, blurred vision, haloes around lights
Unilateral ↑ IOP associated with:

--- Intraocular inflammation
  --- Anterior uveitis
  --- Trabeculitis
  --- Fuchs heterochromic iridocyclitis
  --- **Posner-Schlossman**
  --- Corneal endothelial abnormality

--- Increased episcleral venous pressure
  --- CCF
  --- SVC syndrome
  --- Sturge-Weber
  --- Orbital inflammation
  --- cellulitis
  --- pseudotumor
  --- TED

--- Secondary glaucoma

--- What is the nonpseudonym name for Posner-Schlossman?
  - Glaucomatocyclitic crisis

--- Who is the typical pt?
  - An adult age 20-50

--- Does the inflammatory component tend to be mild, or severe?
  - Mild

--- Does the IOP elevation tend to be mild, or severe?
  - Severe

--- Is the angle open, or closed?
  - Open

--- How long do the crises last?
  - Hours to weeks

--- Do they recur?
  - Yes

--- What is/are the presenting complaint(s)?
  - Unilateral pain, **blurred vision, haloes around lights**
Unilateral ↑ IOP associated with:

---Intraocular inflammation

---Anterior uveitis
---Trabeculitis
---Fuchs heterochromic iridocyclitis
---**Posner-Schlossman**

---Corneal endothelial abnormality

---Increased episcleral venous pressure

---CCF--SVC syndrome
---Sturge-Weber
---Orbital inflammation

---Cellulitis
---Pseudotumor
---TED

---Secondary glaucoma

---Intraocular inflammation

---Anterior uveitis
---Trabeculitis
---Fuchs heterochromic iridocyclitis
---**Posner-Schlossman**

---Corneal endothelial abnormality

---Increased episcleral venous pressure

---CCF--SVC syndrome
---Sturge-Weber
---Orbital inflammation

---Cellulitis
---Pseudotumor
---TED

---Secondary glaucoma

---Unilateral ↑ IOP associated with:

-What is the noneponymous name for Posner-Schlossman?
  Glaucomatocyclitic crisis

-Who is the typical pt?
  An adult age 20-50

-Does the inflammatory component tend to be mild, or severe?
  Mild

-Does the IOP elevation tend to be mild, or severe?
  Severe

-Is the angle open, or closed?
  Open

-How long do the crises last?
  Hours to weeks

-Do they recur?
  Yes

What is/are the presenting complaint(s)?
Unilateral pain, **blurred vision, haloes around lights**

What is the cause of the blurred vision/haloes?
Corneal edema secondary to the high IOP
Unilateral ↑ IOP associated with:

---Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --**Posner-Schlossman**

---Corneal endothelial abnormality

---Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber

---Orbital inflammation
  --cellulitis
  --pseudotumor
  --TED

---Secondary glaucoma

---What is the noneponymous name for Posner-Schlossman?
  Glaucomatocyclitic crisis

---Who is the typical pt?
  An adult age 20-50

---Does the inflammatory component tend to be mild, or severe?
  Mild

---Does the IOP elevation tend to be mild, or severe?
  Severe

---Is the angle open, or closed?
  Open

---How long do the crises last?
  Hours to weeks

---Do they recur?
  Yes
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --**Posner-Schlossman**

--Corneal endothelial abnormality

-What is the noneponymous name for Posner-Schlossman?
  Glaucmatacycliclitic crisis

Infection with what entity has been implicated in the pathogenesis of P-S syndrome?
  CMV, a member of the *Herpesvirus* family

Does the inflammatory component tend to be mild, or severe?
  Mild

Does the IOP elevation tend to be mild, or severe?
  Severe

Is the angle open, or closed?
  Open

How long do the crises last?
  Hours to weeks

Do they recur?
  Yes
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --**Posner-Schlossman**

--Corneal endothelial abnormality

--- What is the noneponymous name for Posner-Schlossman?
  Glaucomatocyclitic crisis

--- Infection with what entity has been implicated in the pathogenesis of P-S syndrome?
  CMV, a member of the **Herpesvirus** family

--- What four anterior uveitis etiologies are notorious for elevating IOP?
  1) Herpesvirus (all forms)
  2) Toxoplasmosis
  3) Sarcoidosis
  4) Syphilis

--- Does the inflammatory component tend to be mild, or severe?
  Mild

--- Does the IOP elevation tend to be mild, or severe?
  Severe

--- Is the angle open, or closed?
  Open

--- How long do the crises last?
  Hours to weeks

--- Do they recur?
  Yes
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --a dystrophy
  --several syndromes

- Increased episcleral venous pressure

--Secondary glaucoma
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD (posterior polymorphous dystrophy)
  --ICE (iridocorneal endothelial syndrome)

  Increased episcleral venous pressure

--Secondary glaucoma
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- Corneal endothelial abnormality
  - PPMD

In a nutshell, what is abnormal about the endothelial cells in PPMD?

They 'behave' like epithelial cells and/or fibroblasts. Specifically and problematically, the endothelial cells proliferate, form multiple layers, and migrate – none of which normal endothelial cells do.

PPMD presents with three posterior-K slit-lamp findings. What are they?

- Linear band-shaped opacities
- Vesicular lesions
- Diffuse opacities

Is PPMD painful? Does it affect vision?

In the majority of pts, PPMD is a stable, painless and visually insignificant condition. However, severe cases can be associated with glaucoma, stromal edema, and significant vision loss.
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

*In a nutshell, what is abnormal about the endothelial cells in PPMD?
They ‘behave’ like epithelial cells and/or fibroblasts.*
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman
--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
--CCF
--SVC syndrome
--Sturge-Weber
--Orbital inflammation
cellulitis
pseudotumor
TED

Secondary glaucoma

In a nutshell, what is abnormal about the endothelial cells in PPMD?
They ‘behave’ like epithelial cells and/or fibroblasts. Specifically and problematically, the endothelial cells proliferate, form multiple layers, and migrate—none of which normal endothelial cells do.

Is PPMD painful? Does it affect vision?
In the majority of pts, PPMD is a stable, painless and visually insignificant condition. However, severe cases can be associated with glaucoma, stromal edema, and significant vision loss.
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD

In a nutshell, what is abnormal about the endothelial cells in PPMD? They ‘behave’ like epithelial cells and/or fibroblasts. Specifically and problematically, the endothelial cells proliferate, form multiple layers, and migrate –none of which normal endothelial cells do.
Unilateral ↑ IOP associated with:

**Normal cornea.** Note the single-cell-thick nature of the endothelial cells.
Unilateral ↑ IOP associated with:

Normal cornea. Note the single-cell-thick nature of the endothelial cells.

Posterior polymorphous corneal dystrophy. Instead of being lined by cells with the attributes of corneal endothelium, the posterior cornea is covered by cells with epithelial/fibroblast-like features.
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
  - PPMD
  - ICE
- Increased episcleral venous pressure
  - CCF
  - SVC syndrome
  - Sturge-Weber
  - Orbital inflammation
    - cellulitis
    - pseudotumor
    - TED
- Secondary glaucoma

In a nutshell, what is abnormal about the endothelial cells in PPMD? They ‘behave’ like epithelial cells and/or fibroblasts. Specifically and problematically, the endothelial cells **proliferate**, form **multiple layers**, and **migrate** – none of which normal endothelial cells do.

**PPMD presents with three posterior-K slit-lamp findings. What are they?**

---

---
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD

In a nutshell, what is abnormal about the endothelial cells in PPMD? They ‘behave’ like epithelial cells and/or fibroblasts. Specifically and problematically, the endothelial cells proliferate, form multiple layers, and migrate—none of which normal endothelial cells do.

PPMD presents with three posterior-K slit-lamp findings. What are they?
  --Linear band-shaped opacities
  --Vesicular lesions
  --Diffuse opacities
Unilateral ↑ IOP associated with:

- Diffuse opacities
- Linear band-shaped opacities
- Vesicular lesions
- PPMD
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

In a nutshell, what is abnormal about the endothelial cells in PPMD? They ‘behave’ like epithelial cells and/or fibroblasts. Specifically and problematically, the endothelial cells proliferate, form multiple layers, and migrate – none of which normal endothelial cells do.

PPMD presents with three posterior-K slit-lamp findings. What are they?
  --Linear band-shaped opacities
  --Vesicular lesions
  --Diffuse opacities

Is PPMD painful? Does it affect vision?
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

In a nutshell, what is abnormal about the endothelial cells in PPMD?
They ‘behave’ like epithelial cells and/or fibroblasts. Specifically and
problematically, the endothelial cells \textit{proliferate}, form \textit{multiple layers},
and \textit{migrate} – none of which normal endothelial cells do.

\textit{PPMD presents with three posterior-K slit-lamp findings. What are they?}
  --Linear band-shaped opacities
  --Vesicular lesions
  --Diffuse opacities

Is PPMD painful? Does it affect vision?
In the majority of pts, PPMD is a stable, painless and visually insignificant
condition.
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD

In a nutshell, what is abnormal about the endothelial cells in PPMD?
They ‘behave’ like epithelial cells and/or fibroblasts. Specifically and problematically, the endothelial cells proliferate, form multiple layers, and migrate – none of which normal endothelial cells do.

PPMD presents with three posterior-K slit-lamp findings. What are they?
--Linear band-shaped opacities
--Vesicular lesions
--Diffuse opacities

Is PPMD painful? Does it affect vision?
In the majority of pts, PPMD is a stable, painless and visually insignificant condition. However, severe cases can be associated with glaucoma, stromal edema, and significant vision loss.
In a nutshell, what is abnormal about the endothelial cells in PPMD? They ‘behave’ like epithelial cells and/or fibroblasts. Specifically and problematically, the endothelial cells proliferate, form multiple layers, and migrate—none of which normal endothelial cells do.

Hol up—as a dystrophy, PPMD must by definition be bilateral, yes?
Unilateral ↑ IOP associated with:
--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman
--Corneal endothelial abnormality
  --PPMD
  --ICE

In a nutshell, what is abnormal about the endothelial cells in PPMD?
They ‘behave’ like epithelial cells and/or fibroblasts. Specifically and problematically, the endothelial cells proliferate, form multiple layers, and migrate—none of which normal endothelial cells do.

Hol up—as a dystrophy, PPMD must by definition be bilateral, yes?
Tru dat

Is PPMD painful? Does it affect vision?
In the majority of pts, PPMD is a stable, painless and visually insignificant condition. However, severe cases can be associated with glaucoma, stromal edema, and significant vision loss.

Increased episcleral venous pressure
--CCF
--SVC syndrome
--Sturge-Weber
--Orbital inflammation
--cellulitis
--pseudotumor
--TED

Secondary glaucoma
**Unilateral** \( \rightarrow \) IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
--CCF
--SVC syndrome
--Sturge-Weber
--Orbital inflammation
--cellulitis
--pseudotumor
--TED

Secondary glaucoma

---

**In a nutshell, what is abnormal about the endothelial cells in PPMD?**

They ‘behave’ like epithelial cells and/or fibroblasts. Specifically and problematically, the endothelial cells **proliferate**, form **multiple layers**, and **migrate** — none of which normal endothelial cells do.

*Hol up—as a dystrophy, PPMD must by definition be bilateral, yes?*

*Tru dat*

*Given this, how can it cause **unilateral IOP elevation**?*

---

**Is PPMD painful? Does it affect vision?**

In the majority of pts, PPMD is a stable, painless and visually insignificant condition. However, severe cases can be associated with glaucoma, stromal edema, and significant vision loss.

---

**Hol up—as a dystrophy, PPMD must by definition be bilateral, yes?**
Intraocular inflammation:
- Anterior uveitis
- Trabeculitis
- Fuchs heterochromic iridocyclitis
- Posner-Schlossman

Corneal endothelial abnormality:
- PPMD
- ICE

Increased episcleral venous pressure:
- CCF
- SVC syndrome
- Sturge-Weber

Orbital inflammation:
- cellulitis
- pseudotumor
- TED

Secondary glaucoma

Unilateral ↑ IOP associated with:

In a nutshell, what is abnormal about the endothelial cells in PPMD?
They ‘behave’ like epithelial cells and/or fibroblasts. Specifically and problematically, the endothelial cells proliferate, form multiple layers, and migrate—none of which normal endothelial cells do.

Hol up—as a dystrophy, PPMD must by definition be bilateral, yes? Tru dat

Given this, how can it cause unilateral IOP elevation?
Because it can be highly asymmetric

Is PPMD painful? Does it affect vision?
In the majority of pts, PPMD is a stable, painless and visually insignificant condition. However, severe cases can be associated with glaucoma, stromal edema, and significant vision loss.
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- Corneal endothelial abnormality
  - PPMD
  - ICE

Increased episcleral venous pressure

Briefly, what is ICE?

Demographically speaking, who is the typical patient?
A young-to-middle-aged adult female

How will a pt with ICE present?
- The pt will c/o changes in the eye’s appearance, and/or pain, and/or decreased VA
- The affected eye will have elevated IOP +/- glaucomatous ONH damage
- The cornea of the affected eye will have abnormal endothelium, and may be edematous
- The fellow eye will be essentially normal
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

Increased episcleral venous pressure

Briefly, what is ICE?
A progressive, nonfamilial, unilateral disorder characterized by abnormalities of the corneal endothelium and iris, as well as PAS formation
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

Increased episcleral venous pressure

Briefly, what is ICE?
A progressive, nonfamilial, unilateral disorder characterized by abnormalities of the corneal endothelium and iris, as well as PAS formation

Demographically speaking, who is the typical patient?
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

Increased episcleral venous pressure

_Briefly, what is ICE?_
A progressive, nonfamilial, unilateral disorder characterized by abnormalities of the corneal endothelium and iris, as well as PAS formation

_Demographically speaking, who is the typical patient?_
A young-to-middle-aged adult female
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

Increased episcleral venous pressure

Briefly, what is ICE?
A progressive, nonfamilial, unilateral disorder characterized by abnormalities of the corneal endothelium and iris, as well as PAS formation

Demographically speaking, who is the typical patient?
A young-to-middle-aged adult female

How will a pt with ICE present?
--
--
--
--
Unilateral ↑ IOP associated with:

--- Intraocular inflammation
- Anterior uveitis
- Trabeculitis
- Fuchs heterochromic iridocyclitis
- Posner-Schlossman

--- Corneal endothelial abnormality
- PPMD
- ICE

*Increased episcleral venous pressure*

**Briefly, what is ICE?**
A progressive, nonfamilial, unilateral disorder characterized by abnormalities of the corneal endothelium and iris, as well as PAS formation

**Demographically speaking, who is the typical patient?**
A young-to-middle-aged adult female

**How will a pt with ICE present?**
-- The pt will c/o changes in the eye’s appearance, and/or pain, and/or decreased VA
-- The affected eye will have elevated IOP +/- glaucomatous ONH damage
-- The cornea of the affected eye will have abnormal endothelium, and may be edematous
-- The fellow eye will be essentially normal
Unilateral ↑ IOP associated with:

--- Intraocular inflammation
  --- Anterior uveitis
  --- Trabeculitis
  --- Fuchs heterochromic iridocyclitis
  --- Posner-Schlossman

--- Corneal endothelial abnormality
  --- PPMD
  --- ICE

--- Increased episcleral venous pressure

The BCSC books recognize three ICE variants—what are they?  
--- Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP; iris nevi/nodules
--- Chandler syndrome: Abnormal endothelium; PAS; elevated IOP; corneal edema
--- Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP; iris atrophy/holes

Demographically speaking, who is the typical patient?
A young-to-middle-aged adult female

How will a pt with ICE present?
--- The pt will c/o changes in the eye’s appearance, and/or pain, and/or decreased VA
--- The affected eye will have elevated IOP +/- glaucomatous ONH damage
--- The cornea of the affected eye will have abnormal endothelium, and may be edematous
--- The fellow eye will be essentially normal
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

Increased episcleral venous pressure

*The BCSC books recognize three ICE variants—what are they?*

--Iris nevus syndrome
--Chandler syndrome
--Essential iris atrophy

A young-to-middle-aged adult female

*How will a pt with ICE present?*
--The pt will c/o changes in the eye’s appearance, and/or pain, and/or decreased VA
--The affected eye will have elevated IOP +/- glaucomatous ONH damage
--The cornea of the affected eye will have abnormal endothelium, and may be edematous
--The fellow eye will be essentially normal
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

Increased episcleral venous pressure

The BCSC books recognize three ICE variants—what are they? All have three findings in common—what are they?
--Iris nevus syndrome: ?
--Chandler syndrome: ?
--Essential iris atrophy: ?

A young-to-middle-aged adult female

How will a pt with ICE present?
--The pt will c/o changes in the eye’s appearance, and/or pain, and/or decreased VA
--The affected eye will have elevated IOP +/- glaucomatous ONH damage
--The cornea of the affected eye will have abnormal endothelium, and may be edematous
--The fellow eye will be essentially normal
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

Increased episcleral venous pressure

The BCSC books recognize three ICE variants—what are they? All have three findings in common—what are they?

--Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP
--Chandler syndrome: Abnormal endothelium; PAS; elevated IOP
--Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP

Demographically speaking, who is the typical patient?
A young-to-middle-aged adult female

How will a pt with ICE present?
--The pt will c/o changes in the eye’s appearance, and/or pain, and/or decreased VA
--The affected eye will have elevated IOP +/- glaucomatous ONH damage
--The cornea of the affected eye will have abnormal endothelium, and may be edematous
--The fellow eye will be essentially normal
Unilateral ↑ IOP associated with:

ICE: PAS
**Unilateral ↑ IOP associated with:**

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--- Increased episcleral venous pressure
--- CCF
--- SVC syndrome
--- Sturge-Weber
--- Orbital inflammation
cellulitis
pseudotumor
TED
--- Secondary glaucoma
--- Lens-related: Phacolytic, phacomorphic
--- PXS
--- PDS

---Intraocular inflammation
---Anterior uveitis
---Trabeculitis
---Fuchs heterochromic iridocyclitis
---Posner-Schlossman

---Corneal endothelial abnormality
---PPMD
---ICE

Unilateral ↑ IOP associated with:

**The BCSC books recognize three ICE variants—what are they? All have three findings in common—what are they?**

--Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP
--Chandler syndrome: Abnormal endothelium; PAS; elevated IOP
--Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP

A young-to-middle-aged, female

**What is the classic metal-related description of the endothelium?**

---The pt will c/o changes in the eye's appearance, and/or pain, and/or decreased VA
---The affected eye will have elevated IOP +/- glaucomatous ONH damage
---The cornea of the affected eye will have abnormal endothelium, and may be edematous
---The fellow eye will be essentially normal
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

The BCSC books recognize three ICE variants—what are they? All have three findings in common—what are they?

--Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP
--Chandler syndrome: Abnormal endothelium; PAS; elevated IOP
--Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP

A young-to-middle-aged adult female

What is the classic metal-related description of the endothelium? ‘Hammered silver’

The pt will c/o changes in the eye’s appearance, and/or pain, and/or decreased VA
--The affected eye will have elevated IOP +/- glaucomatous ONH damage
--The cornea of the affected eye will have abnormal endothelium, and may be edematous
--The fellow eye will be essentially normal
Unilateral ↑ IOP associated with:

ICE: ‘Hammered silver’ corneal endothelium
Unilateral ↑ IOP associated with:

--Intraocular inflammation
    --Anterior uveitis
    --Trabeculitis
    --Fuchs heterochromic iridocyclitis
    --Posner-Schlossman

--Corneal endothelial abnormality
    --PPMD
    --ICE

--Increased episcleral venous pressure

The BCSC books recognize three ICE variants—what are they? All have three findings in common—what are they?

--Iris nevus syndrome: Abnormal endothelium; PAS; *elevated IOP (not so much)*?
--Chandler syndrome: Abnormal endothelium; PAS; *elevated IOP (not so much)*?
--Essential iris atrophy: Abnormal endothelium; PAS; *elevated IOP (not so much)*?

One variant is less likely than the others to manifest elevated IOP—which one?

How will a pt with ICE present?

--The pt will c/o changes in the eye’s appearance, and/or pain, and/or decreased VA
--The affected eye will have elevated IOP +/- glaucomatous ONH damage
--The cornea of the affected eye will have abnormal endothelium, and may be edematous
--The fellow eye will be essentially normal
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

The BCSC books recognize three ICE variants—what are they? All have three findings in common—what are they?
--Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP
--Chandler syndrome: Abnormal endothelium; PAS; elevated IOP (not so much)
--Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP

A young-to-middle-aged adult female

How will a pt with ICE present?
--The pt will c/o changes in the eye’s appearance, and/or pain, and/or decreased VA
--The affected eye will have elevated IOP +/- glaucomatous ONH damage
--The cornea of the affected eye will have abnormal endothelium, and may be edematous
--The fellow eye will be essentially normal

One variant is less likely than the others to manifest elevated IOP—which one?

Chandler syndrome
Unilateral ↑ IOP associated with:

--Intraocular inflammation
- Anterior uveitis
- Trabeculitis
- Fuchs heterochromic iridocyclitis
- Posner-Schlossman

--Corneal endothelial abnormality
- PPMD
- ICE

Unilateral ↑ IOP associated with:

- Increased episcleral venous pressure
- CCF
- SVC syndrome
- Sturge-Weber
- Orbital inflammation
- Cellulitis
- Pseudotumor
- TED

The BCSC books recognize three ICE variants—what are they? All have three findings in common—what are they? Each has a predominant finding—what is it?

- Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP; ?
- Chandler syndrome: Abnormal endothelium; PAS; elevated IOP
- Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP

A young-to-middle-aged adult female

How will a pt with ICE present?
- The pt will c/o changes in the eye’s appearance, and/or pain, and/or decreased VA
- The affected eye will have elevated IOP +/- glaucomatous ONH damage
- The cornea of the affected eye will have abnormal endothelium, and may be edematous
- The fellow eye will be essentially normal
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- Corneal endothelial abnormality
  - PPMD
  - ICE

- Increased episcleral venous pressure

**The BCSC books recognize three ICE variants—what are they? All have three findings in common—what are they? Each has a predominant finding—what is it?**

- Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP; iris nevi/nodules
- Chandler syndrome: Abnormal endothelium; PAS; elevated IOP
- Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP

A young-to-middle-aged adult female

**How will a pt with ICE present?**

- The pt will c/o changes in the eye’s appearance, and/or pain, and/or decreased VA
- The affected eye will have elevated IOP +/- glaucomatous ONH damage
- The cornea of the affected eye will have abnormal endothelium, and may be edematous
- The fellow eye will be essentially normal
Unilateral ↑ IOP associated with:

Iris nevus syndrome
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman
--Corneal endothelial abnormality
  --PPMD
  --ICE

Increased episcleral venous pressure

The BCSC books recognize three ICE variants—what are they? All have three findings in common—what are they? Each has a predominant finding—what is it?

--Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP; iris nevi/nodules
--Chandler syndrome: Abnormal endothelium; PAS; elevated IOP; ?
--Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP

Demographically speaking, who is the typical patient?
A young-to-middle-aged adult female

How will a pt with ICE present?
--The pt will c/o changes in the eye’s appearance, and/or pain, and/or decreased VA
--The affected eye will have elevated IOP +/- glaucomatous ONH damage
--The cornea of the affected eye will have abnormal endothelium, and may be edematous
--The fellow eye will be essentially normal
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

Increased episcleral venous pressure

The BCSC books recognize three ICE variants—what are they? All have three findings in common—what are they? Each has a predominant finding—what is it?

--Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP; iris nevi/nodules
--Chandler syndrome: Abnormal endothelium; PAS; elevated IOP; corneal edema
--Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP

A young-to-middle-aged adult female

How will a pt with ICE present?
--The pt will c/o changes in the eye’s appearance, and/or pain, and/or decreased VA
--The affected eye will have elevated IOP +/- glaucomatous ONH damage
--The cornea of the affected eye will have abnormal endothelium, and may be edematous
--The fellow eye will be essentially normal
Unilateral ↑ IOP associated with:

Chandler syndrome
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

Increased episcleral venous pressure

The BCSC books recognize three ICE variants—what are they? All have three findings in common—what are they? Each has a predominant finding—what is it?

--Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP; iris nevi/nodules
--Chandler syndrome: Abnormal endothelium; PAS; elevated IOP; corneal edema
--Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP; ?

Demographically speaking, who is the typical patient?

A young-to-middle-aged adult female

How will a pt with ICE present?

--The pt will c/o changes in the eye’s appearance, and/or pain, and/or decreased VA
--The affected eye will have elevated IOP +/- glaucomatous ONH damage
--The cornea of the affected eye will have abnormal endothelium, and may be edematous
--The fellow eye will be essentially normal
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

Increased episcleral venous pressure

The BCSC books recognize three ICE variants—what are they? All have three findings in common—what are they? Each has a predominant finding—what is it?

--Iris nevus syndrome: Abnormal endothelium; PAS; elevated IOP; iris nevi/nodules
--Chandler syndrome: Abnormal endothelium; PAS; elevated IOP; corneal edema
--Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP; iris atrophy/holes

A young-to-middle-aged adult female

How will a pt with ICE present?
  --The pt will c/o changes in the eye’s appearance, and/or pain, and/or decreased VA
  --The affected eye will have elevated IOP +/- glaucomatous ONH damage
  --The cornea of the affected eye will have abnormal endothelium, and may be edematous
  --The fellow eye will be essentially normal
Unilateral $\uparrow$ IOP associated with:

Essential iris atrophy
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure

How common is glaucoma in each?

--ICE:
  - Very common—80 to 100% of cases

--PPMD:
  - Not that common—25% of cases

What is the mechanism for increased IOP?

--ICE:
  - Angle closure via PAS and/or a membrane

--PPMD:
  - Unclear; angle can be closed a la ICE, but some have an open angle

--Secondary glaucoma
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure

How common is glaucoma in each?
--ICE: Very common—80 to 100% of cases
--PPMD: Not that common—25% of cases

--Secondary glaucoma
**Unilateral ↑ IOP associated with:**

- **Intraocular inflammation**
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- **Corneal endothelial abnormality**
  - PPMD
  - ICE

- Increased episcleral venous pressure

- Secondary glaucoma

---

**How common is glaucoma in each?**

- **ICE:** Very common—80 to 100% of cases
- **PPMD:** Not that common—25% of cases

**What is the mechanism for increased IOP?**

- **ICE:**
- **PPMD:**
Unilateral ↑ IOP associated with:

--- Intraocular inflammation
  --- Anterior uveitis
  --- Trabeculitis
  --- Fuchs heterochromic iridocyclitis
  --- Posner-Schlossman

--- Corneal endothelial abnormality
  --- PPMD
  --- ICE

--- Increased episcleral venous pressure

--- Secondary glaucoma

--- How common is glaucoma in each?
  --- ICE: Very common—80 to 100% of cases
  --- PPMD: Not that common—25% of cases

--- What is the mechanism for increased IOP?
  --- ICE: Angle closure via PAS and/or a membrane
  --- PPMD: Unclear; angle can be closed a la ICE, but some have an open angle
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure

--Secondary glaucoma

How common is glaucoma in each?

--ICE: Very common—80 to 100% of cases

--PPMD: Not that common—25% of cases

What is the mechanism for increased IOP?

--ICE: Angle closure via PAS and/or a membrane

--PPMD: Unclear; angle can be closed a la ICE, but some have an open angle

What is the take-home message regarding PPMD/ICE and unilateral elevated IOP?

It is incumbent upon you to do a thorough slit-lamp examination of the posterior corneal surface in all pts with unilateral elevated IOP!
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure

--Secondary glaucoma

---How common is glaucoma in each?
  --PPMD: Very common—80 to 100% of cases
  --ICE: Not that common—25% of cases

---What is the take-home message regarding PPMD/ICE and unilateral elevated IOP?
  It is incumbent upon you to do a thorough slit-lamp examination of the posterior corneal surface in all pts with unilateral elevated IOP!

---PPMD: Unclear; angle can be closed a la ICE, but some have an open angle
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- Corneal endothelial abnormality
  - PPMD
  - ICE

- Increased episcleral venous pressure

- Secondary glaucoma

How common is glaucoma in each?

- PPMD: Very common—80 to 100% of cases
- ICE: Not that common—25% of cases

What is the mechanism for increased IOP?

- PPMD: Unclear; angle can be closed a la ICE, but some have an open angle
- ICE: Angle closure via PAS and/or a membrane

What is the take-home message regarding PPMD/ICE and unilateral elevated IOP?
It is incumbent upon you to do a thorough slit-lamp examination of the posterior corneal surface in all pts with unilateral elevated IOP!

For more on PPMD, see slide-set K45

For more ICE, see slide-set K26
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman
--Corneal endothelial abnormality
  --PPMD
  --ICE
--Increased episcleral venous pressure

--Secondary glaucoma
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure

Recall that the Goldmann equation for IOP contains two terms:
--The first term…

--Secondary glaucoma

\[ IOP = \text{First term} + \text{EVP} \]
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure

Recall that the Goldmann equation for IOP contains two terms:
--The first term...quantifies the balance between aqueous formation and aqueous egress

--Secondary glaucoma

\[
IOP = \frac{\text{Aqueous Formation Rate (\(\mu\text{L/min}\))}}{\text{Outflow Facility (\(\mu\text{L/min/mmHg}\))}} + \text{Episcleral Venous Pressure (mmHg)}
\]
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure

Recall that the Goldmann equation for IOP contains two terms:
--The first term...quantifies the balance between aqueous formation and aqueous egress
--The second term in the equation is...

--Secondary glaucoma

\[
IOP = \frac{\text{Aqueous Formation Rate (\(\mu\text{L/min}\))}}{\text{Outflow Facility (\(\mu\text{L/min/mmHg}\))}} + \text{Second term}
\]
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure

Recall that the Goldmann equation for IOP contains two terms:
--The first term…quantifies the balance between aqueous formation and aqueous egress
--The second term in the equation is…EVP.

--Secondary glaucoma

\[
IOP = \frac{\text{Aqueous Formation Rate (} \mu \text{L/min)}}{\text{Outflow Facility (} \mu \text{L/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)}
\]
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure

Recall that the Goldmann equation for IOP contains two terms:
--The first term…quantifies the balance between aqueous formation and aqueous egress
--The second term in the equation is…EVP.

Thus, any ocular and/or systemic conditions that lead to a persistent increase in EVP can produce a persistent elevation in IOP and thus glaucoma. And if this condition affects only one eye, it can produce unilateral elevated IOP.

--Secondary glaucoma

\[
IOP = \frac{\text{Aqueous Formation Rate} \ (\mu L/min)}{\text{Outflow Facility} \ (\mu L/min/mmHg)} + \text{Episcleral Venous Pressure} \ (\text{mmHg})
\]
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --a specific intracranial problem
  --a general intrathoracic problem
  --a phakomatosis
  --a very general ophthalmic problem

--Secondary glaucoma

\[ IOP = \frac{\text{Aqueous Formation Rate (}\mu\text{L/min})}{\text{Outflow Facility (}\mu\text{L/min/mmHg})} + \text{Episcleral Venous Pressure (mmHg)} \]
Unilateral $\uparrow$ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF (carotid-cavernous sinus fistula)
  --SVC syndrome (SVC = superior vena cava)
  --Sturge-Weber
  --Orbital inflammation

--Secondary glaucoma

$$IOP = \frac{\text{Aqueous Formation Rate (}\mu\text{L/min})}{\text{Outflow Facility (}\mu\text{L/min/mmHg})} + \text{Episcleral Venous Pressure (mmHg)}$$
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
  - PPMD
  - ICE
- Increased episcleral venous pressure
  - CCF
  - SVC syndrome
  - Sturge-Weber
  - Orbital inflammation
- Cellulitis
- Pseudotumor
- TED
- Secondary glaucoma
- Lens-related

What is the fundamental anatomical abnormality in CCF?

A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

What important effect does this have on the CS?

The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure (MAP).

How does a CCF lead to unilateral increased IOP?

When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

What two classes of mechanism account for the formation of CCF?

They can be traumatic or spontaneous.

\[
IOP = \frac{\text{Aqueous Formation Rate (} \mu \text{L/min)} + \text{Episcleral Venous Pressure (} \text{mmHg)}}{\text{Outflow Facility (} \mu \text{L/min/mmHg)}}
\]
Unilateral ↑ IOP associated with:

- Intraocular inflammation
- Anterior uveitis
- Trabeculitis
- Fuchs heterochromic iridocyclitis
- Posner-Schlossman
- Corneal endothelial abnormality
- PPMD
- ICE
- Increased episcleral venous pressure
- CCF
- SVC syndrome
- Sturge-Weber
- Orbital inflammation
- Cellulitis
- Pseudotumor
- TED
- Secondary glaucoma

---

**What is the fundamental anatomical abnormality in CCF?**

A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

---

**What important effect does this have on the CS?**

The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure (MAP).

---

**How does a CCF lead to unilateral increased IOP?**

When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

---

**What two classes of mechanism account for the formation of CCF?**

They can be traumatic or spontaneous.

---

\[
IOP = \frac{\text{Aqueous Formation Rate (}\mu\text{L/min})}{\text{Outflow Facility (}\mu\text{L/min/mmHg})} + \text{Episcleral Venous Pressure (mmHg)}
\]
Unilateral $\uparrow$ IOP associated with:

- Intraocular inflammation
- Anterior uveitis
- Trabeculitis
- Fuchs heterochromic iridocyclitis
- Posner-Schlossman
- Corneal endothelial abnormality
- PPMD
- ICE
- Increased episcleral venous pressure
- CCF
- SVC syndrome
- Sturge-Weber
- Orbital inflammation
- Cellulitis
- Pseudotumor
- TED
- Secondary glaucoma
- Lens-related: Phacolytic, phacomorphic
- PXS

What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

What important effect does this have on the CS?
The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure (MAP).

How does a CCF lead to unilateral increased IOP?
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

What two classes of mechanism account for the formation of CCF?
They can be traumatic or spontaneous.

$IOP = \frac{\text{Aqueous Formation Rate (}\mu\text{L/min})}{\text{Outflow Facility (}\mu\text{L/min/mmHg})} + \text{Episcleral Venous Pressure (mmHg)}$
Unilateral $\uparrow$ IOP associated with:

--- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
  - Corneal endothelial abnormality

--- PPMD
--- ICE
--- Increased episcleral venous pressure

--- CCF
--- SVC syndrome
--- Sturge-Weber
--- Orbital inflammation

--- Secondary glaucoma

What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

What important effect does this have on the CS?
The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure (MAP).

How does a CCF lead to unilateral increased IOP?
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

What two classes of mechanism account for the formation of CCF?
They can be traumatic or spontaneous.

$IOP = \frac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)}$
Unilateral ↑ IOP associated with:

--Intraocular inflammation
   --Anterior uveitis
   --Trabeculitis
   --Fuchs heterochromic iridocyclitis
   --Posner-Schlossman

--Corneal endothelial abnormality
   --PPMD
   --ICE

--Increased episcleral venous pressure
   --CCF
   --SVC syndrome
   --Sturge-Weber
   --Orbital inflammation

--Secondary glaucoma

--- What is the fundamental anatomical abnormality in CCF? 
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

--- What important effect does this have on the CS? 
The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure (MAP).

--- How does a CCF lead to unilateral increased IOP? 
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

--- What two classes of mechanism account for the formation of CCF? 
They can be traumatic or spontaneous.

--- IOP = \[ \frac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} \] + \text{Episcleral Venous Pressure (mmHg)}
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
  - PPMD
  - ICE
- Increased episcleral venous pressure
  - CCF
  - SVC syndrome
  - Sturge-Weber
  - Orbital inflammation
- Secondary glaucoma

What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

What important effect does this have on the CS?
The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure (MAP).

How does a CCF lead to unilateral increased IOP?
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

\[
IOP = \frac{\text{Aqueous Formation Rate (}\mu\text{L/min})}{\text{Outflow Facility (}\mu\text{L/min/mmHg})} + \text{Episcleral Venous Pressure (mmHg)}
\]
What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

What important effect does this have on the CS?
The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure (MAP).

How does a CCF lead to unilateral increased IOP?
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

What two classes of mechanism account for the formation of CCF?
They can be traumatic or spontaneous.
What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

What important effect does this have on the CS?
The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure (MAP).

How does a CCF lead to unilateral increased IOP?
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

What two classes of mechanism account for the formation of CCF?
They can be traumatic or spontaneous.

\[ IOP = \frac{\text{Aqueous Formation Rate (\(\mu\text{L/min}\))}}{\text{Outflow Facility (\(\mu\text{L/min/mmHg}\))}} + \text{Episcleral Venous Pressure (mmHg)} \]
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
  - PPMD
  - ICE
- Increased episcleral venous pressure
  - CCF
  - SVC syndrome
  - Sturge-Weber
  - Orbital inflammation
- Cellulitis
- Pseudotumor
- TED
- Secondary glaucoma

What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS)

What important effect does this have on the CS?
The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure (MAP).

How does a CCF lead to unilateral increased IOP?
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

Demographically speaking, who gets:
- Traumatic CCF?
- Spontaneous CCF?

What two classes of mechanism account for the formation of CCF?
They can be traumatic or spontaneous

\[ IOP = \frac{\text{Aqueous Formation Rate (}\mu\text{L/min})}{\text{Outflow Facility (}\mu\text{L/min/mmHg})} + \text{Episcleral Venous Pressure (mmHg)} \]
Unilateral ↑ IOP associated with:

--- Intraocular inflammation
  -- Anterior uveitis
  -- Trabeculitis
  -- Fuchs heterochromic iridocyclitis
  -- Posner-Schlossman
--- Corneal endothelial abnormality
  -- PPMD
  -- ICE
--- Increased episcleral venous pressure
  -- CCF
  -- SVC syndrome
  -- Sturge-Weber
  -- Orbital inflammation
--- Secondary glaucoma

**What is the fundamental anatomical abnormality in CCF?**
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

**What important effect does this have on the CS?**
The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure (MAP).

**How does a CCF lead to unilateral increased IOP?**
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is ultimately results in increased EVP, which results in turn in elevated IOP.

**Demographically speaking, who gets:**
--- Traumatic CCF? Young persons
--- Spontaneous CCF? Middle-aged to elderly females

**What two classes of mechanism account for the formation of CCF?**
They can be traumatic or spontaneous

**IOP** = \[rac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)}\]
What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

What important effect does this have on the CS?
The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure.

What is the classic sign of CCF on imaging?
A dilated and tortuous superior ophthalmic vein

How does a CCF lead to unilateral increased IOP?
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

What two classes of mechanism account for the formation of CCF?
They can be traumatic or spontaneous

\[ IOP = \frac{\text{Aqueous Formation Rate (}\mu\text{L/min})}{\text{Outflow Facility (}\mu\text{L/min/mmHg})} + \text{Episcleral Venous Pressure (mmHg)} \]
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
  - Corneal endothelial abnormality
  - PPMD
  - ICE
- Increased episcleral venous pressure
- CCF
- SVC syndrome
- Sturge-Weber
- Orbital inflammation

What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS)

What important effect does this have on the CS?
The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure.

What classic sign of CCF on imaging?
A dilated and tortuous superior ophthalmic vein

What is the equation for IOP?

\[ IOP = \frac{\text{Aqueous Formation Rate} \, (\mu L/min)}{\text{Outflow Facility} \, (\mu L/min/mmHg)} + \text{Episcleral Venous Pressure} \, (mmHg) \]

What two classes of mechanism account for the formation of CCF?
They can be traumatic or spontaneous

What is the effect of this on IOP?
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.
Unilateral ↑ IOP associated with:

Carotid cavernous fistula in a 20-year-old man s/p motor vehicle collision. CT angiography shows asymmetric enlargement of the left cavernous sinus (arrowheads) and left superior ophthalmic vein (arrow).
**Unilateral ↑ IOP associated with:**

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
  - PPMD
  - ICE
- Increased episcleral venous pressure
  - CCF
  - SVC syndrome
  - Sturge-Weber
  - Orbital inflammation

---

**What is the fundamental anatomical abnormality in CCF?**
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

**In addition to unilateral increased IOP, what other signs/symptoms are commonly associated with a CCF?**

---

**How does a CCF lead to unilateral increased IOP?**
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

**What two classes of mechanism account for the formation of CCF?**
They can be traumatic or spontaneous.

---

**IOP =** \[ \frac{\text{Aqueous Formation Rate (}\mu\text{L/min)}}{\text{Outflow Facility (}\mu\text{L/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)} \]
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
- PPMD
- ICE
- Increased episcleral venous pressure
- CCF
- SVC syndrome
- Sturge-Weber
- Orbital inflammation

What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

In addition to unilateral increased IOP, what other signs/symptoms are commonly associated with a CCF?
- A chronically red eye (beware of the little old lady with ‘chronic conjunctivitis’ that never seems to get any better!)
  - Chemosis
  - Proptosis (in high-flow CCF)
  - Tinnitus

How does a CCF lead to unilateral increased IOP?
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

What two classes of mechanism account for the formation of CCF?
They can be traumatic or spontaneous

\[ IOP = \frac{\text{Aqueous Formation Rate (} \mu\text{L/min)} }{\text{Outflow Facility (} \mu\text{L/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)} \]
A 55 year old woman with a history of HTN presented with a 1-day history of periorbital discomfort, inferior chemosis, and conjunctival injection of the left eye (Panel A). IOP OS was 48. Exam OD was unremarkable. She reported a 2-year history of episodic headache and pulsatile tinnitus in the left ear. Contrast-enhanced computed tomography of the orbit showed proptosis and a dilated left superior ophthalmic vein (Panel B, arrow), suggesting the presence of a carotid–cavernous sinus fistula.
Unilateral ↑ IOP associated with:

What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

In addition to unilateral increased IOP, what other signs/symptoms are commonly associated with a CCF?

- A chronically red eye (beware of the little old lady with ‘chronic conjunctivitis’ that never seems to get any better!)

What slit-lamp finding concerning the appearance of the conj vessels is a tipoff that you’re dealing with a CCF?
The vessels have a kinked or ‘corkscrew’ appearance

What causes this corkscrewing?
Arterialization of blood flow in the vessels (think about how a garden hose will twist and kink when the water is turned on but is held in by a closed nozzle on the end of the hose)

What two classes of mechanism account for the formation of CCF?
They can be traumatic or spontaneous

Unilateral ↑ IOP associated with:

What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

In addition to unilateral increased IOP, what other signs/symptoms are commonly associated with a CCF?

- A chronically red eye (beware of the little old lady with ‘chronic conjunctivitis’ that never seems to get any better!)

What slit-lamp finding concerning the appearance of the conj vessels is a tipoff that you’re dealing with a CCF?
The vessels have a kinked or ‘corkscrew’ appearance

What causes this corkscrewing?
Arterialization of blood flow in the vessels (think about how a garden hose will twist and kink when the water is turned on but is held in by a closed nozzle on the end of the hose)

What two classes of mechanism account for the formation of CCF?
They can be traumatic or spontaneous

\[ IOP = \frac{\text{Aqueous Formation Rate (}\mu\text{L/min})}{\text{Outflow Facility (}\mu\text{L/min/mmHg})} + \text{Episcleral Venous Pressure (mmHg)} \]
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
- PPMD
- ICE
- Increased episcleral venous pressure
- CCF
- SVC syndrome
- Sturge-Weber
- Orbital inflammation

Unilateral ↑ IOP associated with:

\[ IOP = \frac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)} \]

What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

In addition to unilateral increased IOP, what other signs/symptoms are commonly associated with a CCF?
- A chronically red eye (be aware of the little old lady with ‘chronic conjunctivitis’ that never seems to get any better!)

What slit-lamp finding concerning the appearance of the conj vessels is a tipoff that you’re dealing with a CCF?
The vessels have a kinked or ‘corkscrew’ appearance

What causes this corkscrewing?
Arterialization of blood flow in the vessels (think about how a garden hose will twist and kink when the water is turned on but is held in by a closed nozzle on the end of the hose)

What two classes of mechanism account for the formation of CCF?
They can be traumatic or spontaneous.

In addition to unilateral increased IOP, what other signs/symptoms are commonly associated with a CCF?
- A chronically red eye (be aware of the little old lady with ‘chronic conjunctivitis’ that never seems to get any better!)
- Chemosis
- Proptosis (in high-flow CCF)
- Tinnitus

What slit-lamp finding concerning the appearance of the conj vessels is a tipoff that you’re dealing with a CCF?
The vessels have a kinked or ‘corkscrew’ appearance

What causes this corkscrewing?
Arterialization of blood flow in the vessels (think about how a garden hose will twist and kink when the water is turned on but is held in by a closed nozzle on the end of the hose)
Unilateral ↑ IOP associated with:

Corkscrewing of conj vessels 2ndry to CCF
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
- PPMD
- ICE
- Increased episcleral venous pressure
- CCF
- SVC syndrome
- Sturge-Weber
- Orbital inflammation

What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

In addition to unilateral increased IOP, what other signs/symptoms are commonly associated with a CCF?
-A chronically red eye (beware of the little old lady with ‘chronic conjunctivitis’ that never seems to get any better!)

What slit-lamp finding concerning the appearance of the conj vessels is a tipoff that you’re dealing with a CCF?
The vessels have a kinked or ‘corkscrew’ appearance

What causes this corkscrewing?

What two classes of mechanism account for the formation of CCF?
They can be traumatic or spontaneous

IOP = \[
\frac{\text{Aqueous Formation Rate (\(\mu\text{L/min}\))}}{\text{Outflow Facility (\(\mu\text{L/min/mmHg}\))}} + \text{Episcleral Venous Pressure (mmHg)}
\]
What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

What important effect does this have on the CS?
The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure (MAP).

How does a CCF lead to unilateral increased IOP?
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

What two classes of mechanism account for the formation of CCF?
They can be traumatic or spontaneous.

Unilateral ↑ IOP associated with:

- Intraocular inflammation
- Anterior uveitis
- Trabeculitis
- Fuchs heterochromic iridocyclitis
- Posner-Schlossman
- Corneal endothelial abnormality
- PPMD
- ICE
- Increased episcleral venous pressure
- CCF
- SVC syndrome
- Sturge-Weber
- Orbital inflammation

If you're dealing with a CCF:

- A chronically red eye (beware of the little old lady with ‘chronic conjunctivitis’ that never seems to get any better!)
- A chronically red eye
- Chemosis
- Proptosis (in high-flow CCF)
- Tinnitus

What slit-lamp finding concerning the appearance of the conj vessels is a tipoff that you’re dealing with a CCF?
The vessels have a kinked or ‘corkscrew’ appearance.

What causes this corkscrewing?
Arterialization of blood flow in the vessels (think about how a garden hose will twist and kink when the water is turned on but is held in by a closed nozzle on the end of the hose)

IOP = Aqueous Formation Rate (µL/min) + Episcleral Venous Pressure (mmHg)

Outflow Facility (µL/min/mmHg)
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
- PPMD
- ICE
- Increased episcleral venous pressure
- CCF
- SVC syndrome
- Sturge-Weber
- Orbital inflammation

What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

What important effect does this have on the CS?
The normal pressure within the CS is about the same as central venous pressure (CVP; ~9-12 mmHg). When a CCF forms, arterial-pressure blood enters the CS. Thus, a CCF can raise pressure within the CS to as high as mean arterial pressure (MAP).

How does a CCF lead to unilateral increased IOP?
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

What two classes of mechanism account for the formation of CCF?
They can be traumatic or spontaneous.

What sign may be present in a pt who reports the symptom of tinnitus, and should be checked for?
A bruit

How should one check for a bruit in cases of suspected CCF?
By auscultating the globe (through closed lids of course), and at the temporal region.

What is the formula for calculating IOP?
\[
IOP = \frac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)}
\]
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- Corneal endothelial abnormality
  - PPMD
  - ICE

- Increased episcleral venous pressure
  - CCF
  - SVC syndrome
  - Sturge-Weber
  - Orbital inflammation

**What is the fundamental anatomical abnormality in CCF?**
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

**In addition to unilateral increased IOP, what other signs/symptoms are commonly associated with a CCF?**
- A chronically red eye (beware of the little old lady with ‘chronic conjunctivitis’ that never seems to get any better!)
- Chemosis
- Proptosis (in high-flow CCF)

**What sign may be present in a pt who reports the symptom of tinnitus, and should be checked for?**
A bruit

**What two classes of mechanism account for the formation of CCF?**
They can be traumatic or spontaneous

**How does a CCF lead to unilateral increased IOP?**
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

**What is the normal IOP formula?**

\[
IOP = \frac{\text{Aqueous Formation Rate (}\mu\text{L/min})}{\text{Outflow Facility (}\mu\text{L/min/mmHg})} + \text{Episcleral Venous Pressure (mmHg)}
\]
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
  - PPMD
  - ICE
- Increased episcleral venous pressure
  - CCF
  - SVC syndrome
  - Sturge-Weber
  - Orbital inflammation
  - Cellulitis
  - Pseudotumor
  - TED
- Secondary glaucoma

**What is the fundamental anatomical abnormality in CCF?**
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

**In addition to unilateral increased IOP, what other signs/symptoms are commonly associated with a CCF?**
- A chronically red eye (beware of the little old lady with ‘chronic conjunctivitis’ that never seems to get any better!)
- Chemosis
- Proptosis (in high-flow CCF)

**What sign may be present in a pt who reports the symptom of tinnitus, and should be checked for?**
A bruit

**How does a CCF lead to unilateral increased IOP?**
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

**What two classes of mechanism account for the formation of CCF?**
They can be traumatic or spontaneous.

**What is the IOP?**

\[
IOP = \frac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)}
\]
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
  - Corneal endothelial abnormality
  - PPMD
  - ICE
- Increased episcleral venous pressure
  - CCF
  - SVC syndrome
  - Sturge-Weber
  - Orbital inflammation

Unilateral ↑ IOP associated with:

- Aqueous Formation Rate (µL/min)
- Outflow Facility (µL/min/mmHg)
- Episcleral Venous Pressure (mmHg)

What is the fundamental anatomical abnormality in CCF?
A channel forms that allows direct communication between the carotid artery (or one of its dural branches) and the cavernous sinus (CS).

In addition to unilateral increased IOP, what other signs/symptoms are commonly associated with a CCF?
- A chronically red eye (beware of the little old lady with ‘chronic conjunctivitis’ that never seems to get any better!)
- Chemosis
- Proptosis (in high-flow CCF)

What sign may be present in a pt who reports the symptom of tinnitus, and should be checked for?
A bruit

How should one check for a bruit in cases of suspected CCF?
By auscultating the globe (through closed lids of course), and at the temporal region.

How does a CCF lead to unilateral increased IOP?
When the blood pressure within the CS exceeds that of the venous structures that drain into it (including the superior ophthalmic vein), venous drainage is impeded. Retrograde transmission of this impedance ultimately results in increased EVP, which results in turn in elevated IOP.

What two classes of mechanism account for the formation of CCF?
They can be traumatic or spontaneous.
Unilateral ↑ IOP associated with:

--- Intraocular inflammation
  --- Anterior uveitis
  --- Trabeculitis
  --- Fuchs heterochromic iridocyclitis
  --- Posner-Schlossman

--- Corneal endothelial abnormality
  --- PPMD
  --- ICE

--- Increased episcleral venous pressure
  --- CCF
  --- SVC syndrome
  --- Sturge-Weber
  --- Orbital inflammation

--- Secondary glaucoma

\[ IOP = \frac{\text{Aqueous Formation Rate (μL/min)}}{\text{Outflow Facility (μL/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)} \]
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED (Thyroid eye disease)

--Secondary glaucoma

\[
IOP = \frac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)}
\]
Unilateral $\uparrow$ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- Corneal endothelial abnormality
  - PPMD
  - ICE

- Increased episcleral venous pressure
  - CCF
  - SVC syndrome
  - Sturge-Weber
  - Orbital inflammation
    - cellulitis
    - pseudotumor
    - TED

- Secondary glaucoma

$\text{IOP} = \frac{\text{Aqueous Formation Rate (}\mu\text{L/min})}{\text{Outflow Facility (}\mu\text{L/min/mmHg}) + \text{Episcleral Venous Pressure (mmHg)}}$

By what two mechanisms do these raise EVP?

1) Inflammation near the vortex veins causes them to swell, decreasing lumen diameter and thereby impeding drainage.

2) Orbital congestion mechanically compresses the vortex veins.

Unilateral $\uparrow$ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- Corneal endothelial abnormality
  - PPMD
  - ICE

- Increased episcleral venous pressure
  - CCF
  - SVC syndrome
  - Sturge-Weber
  - Orbital inflammation
    - cellulitis
    - pseudotumor
    - TED

- Secondary glaucoma

$\text{IOP} = \frac{\text{Aqueous Formation Rate (}\mu\text{L/min})}{\text{Outflow Facility (}\mu\text{L/min/mmHg}) + \text{Episcleral Venous Pressure (mmHg)}}$
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--Secondary glaucoma

By what two mechanisms do these raise EVP?
1) Inflammation near the vortex veins causes them to swell, decreasing lumen diameter and thereby impeding drainage and/or
2)

\[
IOP = \frac{\text{Aqueous Formation Rate (} \mu\text{L/min)} + \text{Episcleral Venous Pressure (mmHg)}}{\text{Outflow Facility (} \mu\text{L/min/mmHg)}}
\]
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--Secondary glaucoma

By what two mechanisms do these raise EVP?
1) Inflammation near the vortex veins causes them to swell, decreasing lumen diameter and thereby impeding drainage and/or
2) Orbital congestion mechanically compresses the vortex veins

\[
IOP = \frac{\text{Aqueous Formation Rate (\(\mu\text{L/min}\))}}{\text{Outflow Facility (\(\mu\text{L/min/mmHg}\))}} + \text{Episcleral Venous Pressure (mmHg)}
\]
Unilateral ↑ IOP associated with:

--**Intraocular inflammation**
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--**Corneal endothelial abnormality**
  --PPMD
  --ICE

--**Increased episcleral venous pressure**
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--**Secondary glaucoma**

*Before we proceed, a note…It is misleading to have a section labeled ‘secondary glaucoma.’*

*No question—proceed when ready*
Unilateral ↑ IOP associated with:

---Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

---Corneal endothelial abnormality
  --PPMD
  --ICE

---Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

---Secondary glaucoma

Before we proceed, a note…It is misleading to have a section labeled ‘secondary glaucoma.’ Why? Because every etiology above also qualifies as a secondary glaucoma (assuming the elevated IOP results in glaucomatous damage, that is). The term here is used as a catch-all for conditions that didn’t fit easily into other categories.

No question—proceed when ready
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  a general sort of condition
  a specific condition
  another specific condition
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--Secondary glaucoma

These are bilateral, but can be so asymmetric as to seem unilateral

--Lens-related: two specific conditions
  --PXS pseudoexfoliation syndrome
  --PDS pigment dispersion syndrome
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  --Lens-related: Phacolytic, phacomorphic
    --PXS
    --PDS
Unilateral ↑ IOP associated with:

---Intraocular inflammation
   --Anterior uveitis
   --Trabeculitis
   --Fuchs heterochromic iridocyclitis
   --Posner-Schlossman

---Corneal endothelial abnormality
   --PPMD
   --ICE

---Increased episcleral venous pressure
   --CCF
   --SVC syndrome
   --Sturge-Weber
   --Orbital inflammation
     --cellulitis
     --pseudotumor
     --TED

---Secondary glaucoma
   These are bilateral, but can be so asymmetric as to seem unilateral
   --Lens-related: Phacolytic, pPXs
     --PXS
     --PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
--Elevated IOP (duh)
Unilateral ↑ IOP associated with:

-- Intraocular inflammation
  -- Anterior uveitis
  -- Trabeculitis
  -- Fuchs heterochromic iridocyclitis
  -- Posner-Schlossman

-- Corneal endothelial abnormality
  -- PPMD
  -- ICE

-- Increased episcleral venous pressure
  -- CCF
  -- SVC syndrome
  -- Sturge-Weber
  -- Orbital inflammation
    -- cellulitis
    -- pseudotumor
    -- TED

-- Secondary glaucoma
  \[\text{These are bilateral, but can be so asymmetric as to seem unilateral}\]
  \[\text{Lens-related: Phacolytic, pseudophakic, PXS, PDS}\]

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
  -- Elevated IOP (duh)
  -- A mature or hypermature cataract

The pt will complain of the acute onset of pain in an eye that has had poor vision for an extended period of time.

Is the angle open or closed?
  Open

Is an anterior-chamber inflammatory reaction present?
  Yes

What is the underlying pathophysiology?
  Liquefaction of the cataract leads to lens proteins leaching across the capsule into the AC, where they prompt an inflammatory reaction; the protein + inflammatory cells clog the trabecular meshwork, leading to the IOP rise.
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --CE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  --Lens-related: Phacolytic, p--PXS
    --PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
--Elevated IOP (duh)
--A mature or hypermature cataract

What is a mature cataract?
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  --Lens-related: Phacolytic, phacomorphic
    --PXS
    --PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
  --Elevated IOP (duh)
  --A mature or hypermature cataract

What is a mature cataract?
A cortical cataract that has advanced to the point that the entire lens is opaque
Unilateral ↑ IOP associated with:

Mature cataract
Unilateral ↑ IOP associated with:

---Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

---Corneal endothelial abnormality
  --PPMD
  --ICE

---Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

---Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  --Lens-related: **Phacolytic**, p. 206
    --PXS
    --PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
  --Elevated IOP (duh)
  --A **mature or hypermature cataract**

*What is a mature cataract?*
A cortical cataract that has advanced to the point that the entire lens is opaque

*What is a hypermature cataract?*
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber

--Orbital inflammation
  --cellulitis
  --pseudotumor
  --TED

--Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  --Lens-related: Phacolytic, p...
    --PXS
    --PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
  --Elevated IOP (duh)
  --A mature or hypermature cataract

What is a mature cataract?
A cortical cataract that has advanced to the point that the entire lens is opaque

What is a hypermature cataract?
A mature cataract in which the cortical material has partially liquefied
Unilateral ↑ IOP associated with:

Hypermature cataract
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber

--Orbital inflammation
  --cellulitis
  --pseudotumor
  --TED

--Secondary glaucoma
  --Lens-related: Phacolytic, PXS, PDS

These are bilateral, but can be so asymmetric as to seem unilateral

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
--Elevated IOP (duh)
--A mature or hypermature cataract

What is a mature cataract?
A cortical cataract that has advanced to the point that the entire lens is opaque

What is a hypermature cataract?
A mature cataract in which the cortical material has partially liquefied

Once the cortical component has liquefied enough to allow the nuclear component to move freely, what is the cataract called?
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber

--Secondary glaucoma
  --Lens-related: Phacolytic, phacomorphic
  --PXS
  --PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
  --Elevated IOP (duh)
  --A mature or hypermature cataract

What is a mature cataract?
A cortical cataract that has advanced to the point that the entire lens is opaque

What is a hypermature cataract?
A mature cataract in which the cortical material has partially liquefied

Once the cortical component has liquefied enough to allow the nuclear component to move freely, what is the cataract called?
A morgagnian cataract
Unilateral ↑ IOP associated with:

Morgagnian cataract
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  --Lens-related: **Phacolytic**, pseudoxiphophthalmia (PXS), pseudotrabeculitis (PDS)

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
--Elevated IOP (duh)
--A mature or hypermature cataract

What is the classic presentation and history?

The patient will complain of the acute onset of pain in an eye that has had poor vision for an extended period of time. Is the angle open or closed? Open
Is an anterior-chamber inflammatory reaction present? Yes
What is the underlying pathophysiology?
Liquefaction of the cataract leads to lens proteins leaching across the capsule into the AC, where they prompt an inflammatory reaction; the protein + inflammatory cells clog the trabecular meshwork, leading to the IOP rise.
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  --Lens-related: Phacolytic, p
  --PXS
  --PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
--Elevated IOP (duh)
--A mature or hypermature cataract

What is the classic presentation and history?
The pt will complain of the acute onset of pain in an eye that has had a different complaint

These are bilateral, but can be so asymmetric as to seem unilateral
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  --Lens-related: Phacolytic, phacomorphic
    --PXS
    --PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
--Elevated IOP (duh)
--A mature or hypermature cataract

What is the classic presentation and history?
The pt will complain of the acute onset of pain in an eye that has had poor vision for an extended period of time.
Unilateral ↑ IOP associated with:

--- Intraocular inflammation
  --- Anterior uveitis
  --- Trabeculitis
  --- Fuchs heterochromic iridocyclitis
  --- Posner-Schlossman

--- Corneal endothelial abnormality
  --- PPMD
  --- ICE

--- Increased episcleral venous pressure
  --- CCF
  --- SVC syndrome
  --- Sturge-Weber
  --- Orbital inflammation
    --- cellulitis
    --- pseudotumor
    --- TED

--- Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  --- Lens-related: Phacolytic, phacomorphic
    --- PXS
    --- PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
--- Elevated IOP (duh)
--- A mature or hypermature cataract

What is the classic presentation and history?
The pt will complain of the acute onset of pain in an eye that has had poor vision for an extended period of time

Is the angle open or closed?

--- Open

Is an anterior-chamber inflammatory reaction present?
--- Yes

What is the underlying pathophysiology?
Liquefaction of the cataract leads to lens proteins leaching across the capsule into the AC, where they prompt an inflammatory reaction; the protein + inflammatory cells clog the trabecular meshwork, leading to the IOP rise
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber

--Orbital inflammation
  --cellulitis
  --pseudotumor
  --TED

--Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  --Lens-related: Phacolytic, PXS, PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
--Elevated IOP (duh)
--A mature or hypermature cataract

What is the classic presentation and history?
The pt will complain of the acute onset of pain in an eye that has had poor vision for an extended period of time

Is the angle open or closed?
Open

These are bilateral, but can be so asymmetric as to seem unilateral

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
--Elevated IOP (duh)
--A mature or hypermature cataract

What is the classic presentation and history?
The pt will complain of the acute onset of pain in an eye that has had poor vision for an extended period of time

Is the angle open or closed?
Open
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  --Lens-related: Phacolytic, PXS, PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
--Elevated IOP (duh)
--A mature or hypermature cataract

What is the classic presentation and history?
The pt will complain of the acute onset of pain in an eye that has had poor vision for an extended period of time

Is the angle open or closed?
Open

Is an anterior-chamber inflammatory reaction present?
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  --Lens-related: Phacolytic, p---
    --PXS
    --PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
--Elevated IOP (duh)
--A mature or hypermature cataract

What is the classic presentation and history?
The pt will complain of the acute onset of pain in an eye that has had poor vision for an extended period of time

Is the angle open or closed?
Open

Is an anterior-chamber inflammatory reaction present?
Yes
Unilateral ↑ IOP associated with:

--Intraocular inflammation
   --Anterior uveitis
   --Trabeculitis
   --Fuchs heterochromic iridocyclitis
   --Posner-Schlossman

--Corneal endothelial abnormality
   --PPMD
   --ICE

--Increased episcleral venous pressure
   --CCF
   --SVC syndrome
   --Sturge-Weber
   --Orbital inflammation
      --cellulitis
      --pseudotumor
      --TED

--Secondary glaucoma
   These are bilateral, but can be so asymmetric as to seem unilateral
   --Lens-related: Phacolytic, phacomorphic
      --PXS
      --PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
--Elevated IOP (duh)
--A mature or hypermature cataract

What is the classic presentation and history?
The pt will complain of the acute onset of pain in an eye that has had poor vision for an extended period of time

Is the angle open or closed?
Open

Is an anterior-chamber inflammatory reaction present?
Yes

What is the underlying pathophysiology?
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- Corneal endothelial abnormality
  - PPMD
  - ICE

- Increased episcleral venous pressure
  - CCF
  - SVC syndrome
  - Sturge-Weber
  - Orbital inflammation
    - cellulitis
    - pseudotumor
    - TED

- Secondary glaucoma
  - Lens-related: Phacolytic, p2
    - PXS
    - PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
- Elevated IOP (duh)
- A mature or hypermature cataract

What is the classic presentation and history?
The pt will complain of the acute onset of pain in an eye that has had poor vision for an extended period of time

Is the angle open or closed?
Open

Is an anterior-chamber inflammatory reaction present?
Yes

What is the underlying pathophysiology?
Liquefaction of the cataract leads to lens proteins leaching across the capsule into the AC, where they prompt an inflammatory reaction; the protein + inflammatory cells clog the trabecular meshwork, leading to the IOP rise

These are bilateral, but can be so asymmetric as to seem unilateral
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman
--Corneal endothelial abnormality
  --PPMD
  --ICE
--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED
--Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  --Lens-related: Phacolytic, phacomorphic
    --PXS
    --PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
  --Elevated IOP (duh)
  --A mature or hypermature cataract

What is the classic presentation and history?
The pt will complain of the acute onset of pain in an eye that has had poor vision for an extended period of time

Is the angle open or closed?
Open

Is an anterior-chamber inflammatory reaction present?
Yes

What is the underlying pathophysiology?
Liquefaction of the cataract leads to lens proteins leaching across the capsule into the AC, where they prompt an inflammatory reaction; the protein + inflammatory cells clog the trabecular meshwork, leading to the IOP rise

What will cytologic evaluation of an aqueous aspirate reveal?
(This is another classic feature of phacolytic glaucoma.)

Big fat macrophages loaded down with phagocytized lens proteins
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  --Lens-related: Phacolytic, p
    --PXS
    --PDS

To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
  --Elevated IOP (duh)
  --A mature or hypermature cataract

What is the classic presentation and history?
The pt will complain of the acute onset of pain in an eye that has had poor vision for an extended period of time

Is the angle open or closed?
Open

Is an anterior-chamber inflammatory reaction present?
Yes

What is the underlying pathophysiology?
Liquefaction of the cataract leads to lens proteins leaching across the capsule into the AC, where they prompt an inflammatory reaction; the protein + inflammatory cells clog the trabecular meshwork, leading to the IOP rise

What will cytologic evaluation of an aqueous aspirate reveal?
(This is another classic feature of phacolytic glaucoma.)
Big fat macrophages loaded down with phagocytized lens proteins
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman
--Corneal endothelial abnormality
--PPMD
--ICE
--Increased episcleral venous pressure
--CCF
--SVC syndrome
--Sturge-Weber
--Orbital inflammation
--cellulitis
--pseudotumor
--TED

--Secondary glaucoma
These are bilateral, but can be so asymmetric as to seem unilateral
  --Lens-related: Phacolytic, phacomorphic
    --PXS
    --PDS

Is phacomorphic glaucoma a form of open-angle, or angle-closure glaucoma?

In phacomorphic glaucoma, cataractous lens proteins in the AC are the culprit. What is the culprit in phacomorphic glaucoma?

Cataractous increase in lens size

How does cataractous increase in lens size lead to elected IOP?

1) It alters the anatomic relationship between the anterior lens surface and the pupil margin in a manner that leads to pupillary block and subsequent angle closure; and
2) it pushes the peripheral iris forward, narrowing the angle, thereby reducing the magnitude of the PC-AC pressure gradient needed to induce angle closure
Is phacomorphic glaucoma a form of open-angle, or angle-closure glaucoma?

Angle closure

Unilateral ↑ IOP associated with:

---Intraocular inflammation
  ---Anterior uveitis
  ---Trabeculitis
  ---Fuchs heterochromic iridocyclitis
  ---Posner-Schlossman

---Corneal endothelial abnormality
  ---PPMD
  ---ICE

---Increased episcleral venous pressure
  ---CCF
  ---SVC syndrome
  ---Sturge-Weber
  ---Orbital inflammation
  ---cellulitis
  ---pseudotumor
  ---TED

---Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  ---Lens-related: Phacolytic, phacomorphic
    ---PXS
    ---PDS
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--Secondary glaucoma
  These are bilateral, but can be so asymmetric as to seem unilateral
  --Lens-related: Phacolytic, phacomorphic
    --PXS
    --PDS

Is phacomorphic glaucoma a form of open-angle, or angle-closure glaucoma?
Angle closure

In phacolytic glaucoma, cataractous lens proteins in the AC are the culprit. What is the culprit in phacomorphic glaucoma?
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge
  --Orbital inflammation
  --cellulitis
  --pseudotumor
  --TED

--Secondary glaucoma

These are bilateral, but can be so asymmetric as to seem unilateral

--Lens-related: Phacolytic, **phacomorphic**
  --PXS
  --PDS

Is phacomorphic glaucoma a form of open-angle, or angle-closure glaucoma?

Angle closure

In phacolytic glaucoma, cataractous lens proteins in the AC are the culprit. What is the culprit in phacomorphic glaucoma?

Cataractous increase in lens size

How does cataractous increase in lens size lead to elected IOP?

1) It alters the anatomic relationship between the anterior lens surface and the pupil margin in a manner that leads to pupillary block and subsequent angle closure; and
2) it pushes the peripheral iris forward, narrowing the angle, thereby reducing the magnitude of the PC-AC pressure gradient needed to induce angle closure
Unilateral ↑ IOP associated with:

---Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

---Corneal endothelial abnormality
  --PPMD
  --ICE

---Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital cellulitis
  --pseudotumor
  --TED

---Secondary glaucoma

These are bilateral, but can be so asymmetric as to seem unilateral

---Lens-related: Phacolytic, phacomorphic
  --PXS
  --PDS

Is phacomorphic glaucoma a form of open-angle, or angle-closure glaucoma?
Angle closure

In phacolytic glaucoma, cataractous lens proteins in the AC are the culprit. What is the culprit in phacomorphic glaucoma?
Cataractous increase in lens size

How does cataractous increase in lens size lead to elected IOP?
1) It alters the anatomic relationship between the anterior lens surface and the pupil margin in a manner that leads to pupillary block and subsequent angle closure; and
2) it pushes the peripheral iris forward, narrowing the angle, thereby reducing the magnitude of the PC-AC pressure gradient needed to induce angle closure
Unilateral ↑ IOP associated with:

- Intraocular inflammation
  - Anterior uveitis
  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman

- Corneal endothelial abnormality
  - PPMD
  - ICE

- Increased episcleral venous pressure
  - CCF
  - SVC syndrome
  - Sturge-Weber
  - Orbital cellulitis
  - Pseudotumor
  - TED

- Secondary glaucoma
  - Lens-related: Phacolytic, phacomorphic
    - PXS
    - PDS

Is phacomorphic glaucoma a form of open-angle, or angle-closure glaucoma?
Angle closure

In phacolytic glaucoma, cataractous lens proteins in the AC are the culprit. What is the culprit in phacomorphic glaucoma?
Cataractous increase in lens size

How does cataractous increase in lens size lead to elevated IOP?
1) It alters the anatomic relationship between the anterior lens surface and the pupil margin in a manner that leads to pupillary block and subsequent angle closure; and
2) it pushes the peripheral iris forward, narrowing the angle, thereby reducing the magnitude of the PC-AC pressure gradient needed to induce angle closure
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cellulitis
    --pseudotumor
    --TED

--Secondary glaucoma
  --Lens-related: Phacolytic, phacomorphic
  --PXS
  --PDS

What is the status of the lens capsule in these conditions?
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cell
    --psl
    --TE

--Secondary glaucoma
  --Lens-related: Phacolytic, phacomorphic
    --PXS
    --PDS

What is the status of the lens capsule in these conditions? By definition, intact
Unilateral ↑ IOP associated with:

--Intraocular inflammation
   --Anterior uveitis
   --Trabeculitis
   --Fuchs heterochromic iridocyclitis
   --Posner-Schlossman

--Corneal endothelial abnormality
   --PPMD
   --ICE

--Increased episcleral venous pressure
   --CCF
   --SVC syndrome
   --Sturge-Weber
   --Orbital inflammation
     --cell
     --pseudotumor
     --TED

--Secondary glaucoma
   --Lens-related: Phacolytic, phacomorphic
   --PXS
   --PDS

What lens condition associated with uveitis has, by definition, a capsule that has been traumatically or surgically breached?

What is the status of the lens capsule in these conditions?
By definition, intact
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber
  --Orbital inflammation
    --cell
    --ps
    --TE

--Secondary glaucoma
  --Lens-related: Phacolytic, phacomorphic
    --PXS
    --PDS

What lens condition associated with uveitis has, by definition, a capsule that has been traumatically or surgically breached? Phacoantigenic uveitis

What is the status of the lens capsule in these conditions? By definition, intact
Unilateral ↑ IOP associated with:

--Intraocular inflammation
   --Anterior uveitis
   --Trabeculitis
   --Fuchs heterochromic iridocyclitis
   --Posner-Schlossman

--Corneal endothelial abnormality
   --PPMD
   --ICE

--Increased episcleral venous pressure
   --CCF
   --SVC syndrome
   --Sturge-Weber
   --Orbital inflammation
     --cellulitis
     --pseudotumor
     --TED

--Secondary glaucoma
   --Lens-related: Phacolytic, phacomorphic
   --PXS
   --PDS

*PXS and PDS are compared and contrasted in detail in slide-set G4*
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure
  --CCF
  --SVC syndrome
  --Sturge-Weber

There are many other causes of secondary glaucoma—see both G13 (2ndry open-angle glaucoma) and G16 (2ndry angle-closure glaucoma)

--TED

--Secondary glaucoma
  --Lens-related: Phacolytic, phacomorphic
  --PXS
  --PDS