Unilateral $\uparrow$ 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

--Posner-Schlossman

--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

--SVC syndrome

--Secondary glaucoma
Unilateral ↑ IOP associated with:

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

**Hypotensive**

**Why hypotensive?**

--Corneal endothelial abnormality

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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.

--Increased episcleral venous pressure

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--Secondary glaucoma
Unilateral ↑ IOP associated with:

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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
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Unilateral ↑ IOP associated with:

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

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

--Increased episcleral venous pressure

--Secondary glaucoma
Unilateral ↑ IOP associated with:

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

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.

--Increased episcleral venous pressure

--Secondary glaucoma
Unilateral $\uparrow$ IOP associated with:

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

--Increased episcleral venous pressure

Both of these present with unilateral AC cell and $\uparrow$ 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 $\uparrow$ IOP, the inflammatory reaction must be quite severe. In contrast, the cell associated with trabeculitis can be quite mild.

--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 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
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--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
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    --Fuchs heterochromic iridocyclitis
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--Corneal endothelial abnormality

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What four anterior uveitis etiologies are notorious for elevating IOP?
1) this one is a family of infectious agents
2)
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What four anterior uveitis etiologies are notorious for elevating IOP?

1) Herpesvirus (all forms)
2) this one is a specific bug
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Unilateral ↑ IOP associated with:

--Intraocular inflammation
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  - 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
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1) Herpesvirus (all forms)
2) Toxoplasmosis
3) Sarcoidosis
4) This one is another specific bug

---

Unilateral ↑ IOP associated with:

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Which of these can present with granulomatous-appearing KP?

All of them
Unilateral ↑ IOP associated with:

--Intraocular inflammation
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    --Trabeculitis
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What four anterior uveitis etiologies are notorious for elevating IOP?
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Which can present with stellate KP?

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

--Intraocular inflammation
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1) Herpesvirus (all forms)
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Which can present with stellate KP?
All but sarcoid (and don’t forget about FHI)

FHI causes stellate KP too
**Unilateral ↑ IOP associated with:**

<table>
<thead>
<tr>
<th>Intraocular inflammation</th>
<th>What is the usual treatment regimen for anterior uveitis accompanied by increased IOP?</th>
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<tbody>
<tr>
<td>- Anterior uveitis</td>
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- **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
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**What two classes of topical hypotensives should probably be avoided?**

--Miotics
--Prostaglandin analogues/prostamides

Both are potentially inflammogenic
Unilateral ↑ IOP associated with:

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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 an 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).
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So how do you know whether steroids are the solution to, or the cause of, ocular hypertension in uveitis?
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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.
Intraocular inflammation
- Anterior uveitis
- Trabeculitis
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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.*)*
What exam findings comprise the ‘classic triad’ of FHI? (Hint: Elevated IOP is not one of them.)
Heterochromia iridis, cataract, and stellate KP
Unilateral ↑ IOP associated with:

FHI: Heterochromia
Unilateral $\uparrow$ IOP associated with:

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

Heterochromia iridis, cataract, and stellate KP

In addition to heterochromia, the atrophic changes give the iris an appearance that has been likened to damage caused by an insect. What is the two-word description for this appearance?

'Moth eaten'

Is it the darker eye or the lighter eye?

The lighter (with one exception)

In individuals with light-blue eyes…

the iris atrophy stemming from the FHI process

What is the exception; ie, under what circumstances is the darker eye the one with FHI?

In individuals with light-blue eyes, the atrophic changes will make visible the darkly-pigmented epithelium of the posterior iris, thus making the eye appear darker.
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*Is it true? 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.*

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---

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

---

*What is the etiology?* Unknown. Some experts think it’s infectious (various viruses as well as toxoplasmosis have been proposed as the inciting agent), but this has yet to be proven.

---

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

---

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

FHI: ‘Moth eaten’ iris. Note the smooth stromal architecture and loss of iris crypts
Unilateral ↑ IOP associated with:

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

--Intraocular inflammation
  --Anterior uveitis
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**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**
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*How common is glaucoma in FHI?*
It develops in about 25-50% of cases
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How common is **glaucoma in FHI**?
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Is the angle in FHI glaucoma open, or is it closed?
Unilateral ↑ IOP associated with:

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**How common is glaucoma in FHI?**
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*Speaking of the angle in FHI… It has two characteristics that are unusual, and may aid in making the diagnosis:*

--

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Unilateral ↑ IOP associated with:

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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, **three words (and an abb.)** never develop

--The angle in FHI is glaucoma open, or is it closed? **Open**
Unilateral ↑ IOP associated with:

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Unilateral ↑ IOP associated with:

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

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-- 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
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**The NVA vessels in FHI rupture easily, and it not uncommon for FHI pts to develop a small hyphema after the paracentesis wound is made at the start of cataract surgery.**

**What is the eponymous name for this classic finding?**

---
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What is the eponymous name for this classic finding?
**Amsler’s sign**
Unilateral ↑ IOP associated with:

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What is the eponymous name for this classic finding?
Amsler’s sign

Is Amsler’s sign pathognomonic for FHI?
Unilateral \( \uparrow \) IOP associated with:

--Intraocular inflammation
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Open

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Can other clinical maneuvers cause these vessels to bleed?
Yes—hyphema in FHI can occur subsequent to gonioscopy, and even after applanation tonometry

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

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

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

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

--- Intraocular inflammation
  --- Anterior uveitis
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Who is the typical pt?
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Is there a gender predilection?
Unilateral ↑ IOP associated with:

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

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

**Intraocular inflammation**

- Anterior uveitis
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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.

Who is the typical pt?

- A middle-aged adult

Is there a gender predilection?

- No
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**Is cataract surgery in FHI associated with an increased risk of intraoperative complications?**

No

Is there a gender predilection?

No
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  --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
- 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?**

- 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

--PXS

--PDS

---What is the non-eponymous name for Posner-Schlossman?

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?
  Glaucmatocyclitic crisis

---

--Increased episcleral venous pressure

--CCF

--SVC syndrome

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Glaucmatocyclitic crisis

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  Glaucomatocyclitic crisis

--Increased episcleral venous pressure

--Secondary glaucoma

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

*Yes*
Unilateral ↑ IOP associated with:

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  --Anterior uveitis
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Mild

--Secondary glaucoma
Unilateral ↑ IOP associated with:

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

--Increased episcleral venous pressure
  "Who is the typical pt?"
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  "Does the inflammatory component tend to be mild, or severe?"
  Mild

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

--Secondary glaucoma
  "How severe?"
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
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- *What is the noneponymous name for Posner-Schlossman?*
  Glaucomatocyclitic crisis

--Increased episcleral venous pressure

*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
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---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
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*How long do the crises last?*
Unilateral ↑ IOP associated with:

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  --Anterior uveitis
  --Trabeculitis
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  Glaucomatocyclitic crisis

--Increased episcleral venous pressure

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Mild

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

--Secondary glaucoma

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
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Open

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

---Do they recur?
Yes
Unilateral \( \uparrow \) IOP associated with:

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

--- Corneal endothelial abnormality

--- Increased episcleral venous pressure
  --- **ICE**

--- Secondary glaucoma

--- Sturge-Weber

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

--- What is the noneponymous name for Posner-Schlossman?
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--- Who is the typical pt?
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  - Mild

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

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

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

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  --Anterior uveitis
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--Secondary glaucoma

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

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

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

4. Is the angle open, or closed?
   Open

5. How long do the crises last?
   Hours to weeks

6. 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

--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

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

-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?
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
  - *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

--- Increased episcleral venous pressure

--- Secondary glaucoma
  - 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

--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 non-eponymous 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

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
- PPMD
- ICE
- Increased episcleral venous pressure
- CCF
- SVC syndrome
- Sturge-Weber
- Orbital inflammation
- Cellulitis
- Pseudotumor
- TED
- Secondary glaucoma

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

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

Infection with what entity has been implicated in the pathogenesis of P-S syndrome?
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Does the inflammatory component tend to be mild, or severe?
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Does the IOP elevation tend to be mild, or severe?
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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
In a nutshell, what is abnormal about the endothelial cells in PPMD?
Unilateral ↑ IOP associated with:

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

--Corneal endothelial abnormality
  --PPMD

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

--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.
Intraocular inflammation

Anterior uveitis
Trabeculitis
Fuchs heterochromic iridocyclitis
Posner-Schlossman

Corneal endothelial abnormality

PPMD

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.

Increased episcleral venous pressure

CCF
SVC syndrome
Sturge-Weber
Orbital inflammation
Cellulitis
Pseudotumor
TED

Secondary glaucoma

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
- Increased episcleral venous pressure
  - CC
  - SVC syndrome
- Orbital inflammation
  - Cellulitis
  - Pseudotumor
  - TED
- Secondary glaucoma

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They ‘behave’ like epithelial cells and/or fibroblasts. Specifically and problematically, the endothelial cells prolif erate, 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
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--Corneal endothelial abnormality
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PPMD presents with three posterior-K slit-lamp findings. What are they?

--

--

--Secondary glaucoma
Unilateral ↑ IOP associated with:

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

--Secondary glaucoma

--Increased episcleral venous pressure

--CCF

--SVC syndrome

--Sturge-Weber

--Orbital inflammation

--Cellulitis

--Pseudotumor

--TED
Unilateral ↑ IOP associated with:

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

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
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--- Diffuse opacities

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

--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
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--Corneal endothelial abnormality
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In a nutshell, what is abnormal about the endothelial cells in PPMD?
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--Linear band-shaped opacities
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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
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- Corneal endothelial abnormality
  - PPMD

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Intraocular inflammation

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

Corneal endothelial abnormality

- PPMD

Increased episcleral venous pressure

CCF

SVC syndrome

Sturge-Weber

Orbital inflammation

Cellulitis

Pseudotumor

TED

Secondary glaucoma

Unilateral ↑ IOP associated with:

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

- Corneal endothelial abnormality
  - PPMD

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

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

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

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Given this, how can it cause unilateral IOP elevation?

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Unilateral \( \uparrow \) IOP associated with:

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  - Trabeculitis
  - Fuchs heterochromic iridocyclitis
  - Posner-Schlossman
- Corneal endothelial abnormality
  - PPMD
  - ICE
- Increased episcleral venous pressure
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- Sturge-Weber
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  - Cellulitis
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  - TED
- Secondary glaucoma

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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

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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:

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  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

Increased episcleral venous pressure

Briefly, what is ICE?
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:

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

Increased episcleral venous pressure: 'CCF'

SVC syndrome

Sturge-Weber

Orbital inflammation

Cellulitis

Pseudotumor

TED

Secondary glaucoma

Lens-related: Phacolytic, phacomorphic

PXS

PDS

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

--?

--?

--?

A young-to-middle-aged adult female

How will a pt with ICE present?

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--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
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- Corneal endothelial abnormality
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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; 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?
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--Corneal endothelial abnormality
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  --ICE

Increased episcleral venous pressure

Ice syndrome

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

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ICE: PAS
Unilateral ↑ IOP associated with:

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

A young-to-middle-aged, incidentally female

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

Abnormal endothelium
Abnormal endothelium
Abnormal endothelium

The pt will c/o changes in the eye's appearance, and/or pain, and/or decreased VA

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

A young-to-middle-aged-until female

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

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
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--The cornea of the affected eye will have abnormal endothelium, and may be edematosus
--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
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One variant is less likely than the others to manifest elevated IOP—which one?

Chandler syndrome

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)?
Unilateral ↑ IOP associated with:

--Intraocular inflammation
  --Anterior uveitis
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 Increased episcleral venous pressure

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--**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

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**Chandler syndrome**

How will a pt with ICE present?

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---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; ?
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--Essential iris atrophy: Abnormal endothelium; PAS; elevated IOP

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  --ICE

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

--Sturge-Weber
--Orbital inflammation
--cellulitis
--pseudotumor
--TED

Secondary glaucoma
--Lens-related: Phacolytic, phacomorphic

PXS
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Increased episcleral venous pressure

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A young-to-middle-aged adult female
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  --Anterior uveitis
  --Trabeculitis
  --Fuchs heterochromic iridocyclitis
  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

Increased episcleral venous pressure
--CCF
--SVC syndrome

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

--Secondary glaucoma
--Lens-related: Phacolytic, phacomorphic

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  --Posner-Schlossman

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure

--Secondary glaucoma

How common is glaucoma in each?
--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
Unilateral ↑ IOP associated with:

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

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

- ICE:
- PPMD:
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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

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  --cellulitis
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--Secondary glaucoma

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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
   --CCF
   --SVC syndrome
   --Sturge-Weber

---Orbital inflammation
   --Inflammation
   --Cellulitis
   --Pseudotumor
   --TED

---Secondary glaucoma

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  - 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

How common is glaucoma in each?
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For more on PPMD, see slide-set K45
For more ICE, see slide-set K26
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--Intraocular inflammation
  --Anterior uveitis
  --Trabeculitis
  --Fuch's 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…
  --Second term...

\[ IOP = \text{First term} + \text{Second term} \]
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

--Secondary glaucoma

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

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

--Corneal endothelial abnormality
  --PPMD
  --ICE

--Increased episcleral venous pressure

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

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

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  --Anterior uveitis
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  --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 (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)}
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Unilateral ↑ IOP associated with:

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  --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

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

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IOP = \frac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)}
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- Increased episcleral venous pressure
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  - 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.

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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?**

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**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
- Cellulitis
- Pseudotumor
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Demographically speaking, who gets:

- Traumatic CCF?
- Spontaneous CCF?

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What two classes of mechanism account for the formation of CCF?
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Demographically speaking, who gets:
- Traumatic CCF? Young persons
- Spontaneous CCF? Middle-aged to elderly females

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

What is the formula for intraocular pressure (IOP)?
IOP = Aqueous Formation Rate (µL/min) + Episcleral Venous Pressure (mmHg)

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They can be traumatic or spontaneous.
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:

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

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IOP = \[
\begin{align*}
\text{Aqueous Formation Rate (} \mu \text{L/min)} & + \\
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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.

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Outflow Facility ($\mu$L/min/mmHg)

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

--- Intraocular inflammation
--- Anterior uveitis
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--- Corneal endothelial abnormality
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What is the formula for IOP?
\[ IOP = \text{Aqueous Formation Rate (µL/min)} + \text{Outflow Facility (µL/min/mmHg)} + \text{Episcleral Venous Pressure (mmHg)} \]
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Corkscrewing of conj vessels 2ndry to CCF
Unilateral \( \uparrow \) IOP associated with:

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--Corneal endothelial abnormality

--Increased 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).

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 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; \( \sim 9-12 \text{ mmHg} \)). 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

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

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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:

--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

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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?
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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)

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:

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  - Anterior uveitis
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  - 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

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

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 (beware of the little old lady with ‘chronic conjunctivitis’ that never seems to get any better!)
- Chemosis
- Proptosis

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

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 fundamental anatomical abnormality in CCF?
**Unilateral ↑ IOP associated with:**

<table>
<thead>
<tr>
<th>Condition</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intraocular inflammation</td>
</tr>
<tr>
<td>Anterior uveitis</td>
</tr>
<tr>
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<td>TED</td>
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---

**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

---

**What is the formula for IOP?**

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

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- PPMD
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- Increased episcleral venous pressure
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- SVC syndrome
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- Orbital inflammation
- Cellulitis
- Pseudotumor
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- Secondary glaucoma

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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.

\[ IOP = \frac{\text{Aqueous Formation Rate (µL/min)}}{\text{Outflow Facility (µL/min/mmHg)}} + \text{Episcleral Venous Pressure (mmHg)} \]
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
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- Orbital inflammation
- Cellulitis
- Pseudotumor
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$IOP = \frac{\text{Aqueous Formation Rate (\(\mu L/min\))}}{\text{Outflow Facility (\(\mu L/min/mmHg\))}} + \text{Episcleeral 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

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  --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

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Unilateral ↑ IOP associated with:

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  --Anterior uveitis
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  --Fuchs heterochromic iridocyclitis
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  --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
2) Orbital congestion mechanically compresses the vortex veins

Unilateral ↑ IOP associated with:

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  --PPMD
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  --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

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  - SVC syndrome
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  - Orbital inflammation
    - cellulitis
    - pseudotumor
    - TED
- Secondary glaucoma

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--Increased episcleral venous pressure
  --CCF
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  --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-Webber
  --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

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
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)
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
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 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**
  - 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 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**
    -- 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
    --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
  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 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
  -- Orbital inflammation
    -- cellulitis
    -- pseudotumor
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-- Secondary glaucoma
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  -- Lens-related: Phacolytic
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A cortical cataract that has advanced to the point that the entire lens is opaque

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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

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

--Secondary glaucoma
  --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?
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

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--Secondary glaucoma

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 one complaint in an eye that has had a different complaint

Lens-related: Phacolytic

--PXS
--PDS
Unilateral ↑ IOP associated with:

-- Intraocular inflammation
  -- Anterior uveitis
  -- Trabeculitis
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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
    --PXS
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To make the diagnosis of phacolytic glaucoma, what two clinical features must be present?
  --Elevated IOP (duh)
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Is the angle open or closed?

Open
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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
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What will cytologic evaluation of an aqueous aspirate reveal?
(This is another classic feature of phacolytic glaucoma.)

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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
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Is phacomorphic glaucoma a form of open-angle, or angle-closure glaucoma?

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
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What is the status of the lens capsule in these conditions?
Unilateral ↑ IOP associated with:

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What is the status of the lens capsule in these conditions? By definition, intact
Intraocular inflammation
--Anterior uveitis
--Trabeculitis
--Fuchs heterochromic iridocyclitis
--Posner-Schlossman

Corneal endothelial abnormality
--PPMD
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Increased episcleral venous pressure
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Orbital inflammation
--cellulitis
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--TED

Secondary glaucoma
--Lens-related: Phacolytic, phacomorphic
--PXS
--PDS

Unilateral ↑ IOP associated with:

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
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What lens condition associated with uveitis has, by definition, a capsule that has been traumatically or surgically breached? Phacoantigenic uveitis

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PXS and PDS are compared and contrasted in detail in slide-set G4
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There are many other causes of secondary glaucoma—see both G13 (2ndry open-angle glaucoma) and G16 (2ndry angle-closure glaucoma)

- TED

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