Before you begin: This is a big topic, and big topics beget big slide-sets. There’s a couple of natural breaks (around slide 235, and again at 399); I placed *break time!* slides at those locations.
Define glaucoma.
Define glaucoma.
A group of optic neuropathies that present with progressive ONH damage and characteristic VF loss

Open-angle Glaucoma: Secondary
Define glaucoma.
A group of optic neuropathies that present with progressive ONH damage and characteristic VF loss

Why isn’t elevated IOP mentioned above?
Define glaucoma.
A group of optic neuropathies that present with progressive ONH damage and characteristic VF loss

Why isn’t elevated IOP mentioned above?
Elevated IOP is a strong risk factor for glaucoma, but it need not be present—IOP can be normal, or even low
Define glaucoma. A group of optic neuropathies that present with progressive ONH damage and characteristic VF loss.

Why isn’t elevated IOP mentioned above? Elevated IOP is a strong risk factor for glaucoma, but it need not be present—IOP can be normal, or even low.

In addition to being the strongest risk factor for glaucoma, IOP has another quality that renders it unique—what is it?
Define glaucoma.
A group of optic neuropathies that present with progressive ONH damage and characteristic VF loss

Why isn’t elevated IOP mentioned above?
Elevated IOP is a strong risk factor for glaucoma, but it need not be present—IOP can be normal, or even low

In addition to being the strongest risk factor for glaucoma, IOP has another quality that renders it unique—what is it?
It is the only risk factor that is modifiable in a manner proven to influence the risk of glaucoma progression
Define glaucoma.
A group of optic neuropathies that present with progressive ONH damage and characteristic VF loss

Why isn’t elevated IOP mentioned above?
Elevated IOP is a strong risk factor for glaucoma, but it need not be present—IOP can be normal, or even low.

In addition to being the strongest risk factor for glaucoma, IOP has another quality that renders it unique—what is it?
It is the only risk factor that is modifiable in a manner proven to influence the risk of glaucoma progression.

That’s why glaucoma management concerns nothing but IOP-lowering maneuvers!
The first thought you should have when encountering a pt you suspect has glaucoma is…
The first thought you should have when encountering a pt you suspect has glaucoma is…

*What is the status of the angle?*
Glaucoma

Open-angle Glaucoma: Secondary

Closed- or narrow-angle

The first thought you should have when encountering a pt you suspect has glaucoma is...

What is the status of the angle?

How does one determine the status of the angle?
The first thought you should have when encountering a pt you suspect has glaucoma is…

What is the status of the angle?

How does one determine the status of the angle?

Gonioscopy. Don’t assume your glaucoma pt has open angles—prove it by gonioing them!
How does one determine the status of the angle?

Gonioscopy

Don't assume your glaucoma pt has open angles—prove it by gonioing them!

Glaucoma

Open-angle Glaucoma:

Open-angle

Closed- or narrow-angle

The first thought you should have when encountering a pt you suspect has glaucoma is...

What is the status of the angle?

Angle-closure glaucoma is covered in multiple slide-sets; see the Table of Contents
Once you have determined a pt has open-angle glaucoma, the next ‘first thought’ is to ask…
Once you have determined a pt has open-angle glaucoma, the next ‘first thought’ is to ask…

Is it high-pressure OAG, or low (aka normal) tension OAG?
Untreated IOP consistently above # mmHg

Normal-tension glaucoma (NTG)

Untreated IOP consistently below # mmHg
Untreated IOP consistently above 22 mmHg

Normal-tension glaucoma (NTG)

Untreated IOP consistently below 22 mmHg

(Note that this distinction is somewhat controversial, as some glaucomalogists contend NTG is not a separate condition.)
Untreated IOP consistently above 22 mmHg

Open-angle Glaucoma: Secondary

OAG

Normal-tension glaucoma (NTG)

↑IOP

Untreated IOP consistently below 22 mmHg

(Note that this distinction is somewhat controversial, as some glaucomalogists contend NTG is not a separate condition.)

Normal-tension glaucoma is covered in its own slide-set (G21)
Once you have determined a pt has high-pressure open-angle glaucoma, the next ‘first thought’ is to ask…
Once you have determined a pt has high-pressure open-angle glaucoma, the next ‘first thought’ is to ask…

*Is it primary open-angle glaucoma (POAG), or secondary OAG?*
There are many forms of secondary open-angle glaucoma!
Open-angle Glaucoma: Secondary

↑ IOP OAG

Primary

Secondary

Drug-Induced

General category

↑ EVS

Trauma-Related

General category

Schwartz syndrome

Specific condition

PXS Pigmentary

Tumor-Induced

Lens-Induced

Inflammation-Induced

Specific conditions

There are many forms of secondary open-angle glaucoma!
Note that primary open-angle glaucoma (POAG) is a diagnosis of exclusion—it can only be made by first determining that the angle is open, and then ruling out the myriad causes of secondary OAG.
Open-angle Glaucoma: Secondary

↑ IOP OAG

Primary

Secondary

PXS Pigmentary

Tumor-induced

Lens-induced

Inflammation-Induced

↑ EVP

Trauma-Related

Drug-Induced

Schwartz syndrome

PXS and pigmentary glaucoma are addressed in slide-set G4
Let’s take a look at secondary OAG owing to the lens

- Primary
- Secondary

↑ IOP OAG

- PXS Pigmentary
- Tumor-induced
- Inflammation-Induced
- Drug-Induced
- Trauma-Related
- Schwartz syndrome
- EVP

Lens-induced
The only one described in the *Glaucoma* book as ‘rare’:
The only one described in the *Glaucoma* book as ‘rare’:

Phacoantigenic
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer):

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic
- Mediated by inflammatory response to lens proteins in AC:
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

1. The only one described in the *Glaucoma* book as ‘rare’:
   - Phacoantigenic

2. Mediated by inflammatory response to lens proteins in AC:
   - Phacoantigenic; phacolytic
| The only one described in the *Glaucoma* book as ‘rare’: | **Phacoantigenic** |
| Mediated by inflammatory response to lens proteins in AC: | **Phacoantigenic; phacolytic** |
| Mediated by IgG antibodies: | |
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic

Mediated by IgG antibodies: Phacoantigenic
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic

Mediated by IgG antibodies: Phacoantigenic

TM is clogged with macrophages:
• The only one described in the *Glaucoma* book as ‘rare’: **Phacoantigenic**
• Mediated by inflammatory response to lens proteins in AC: **Phacoantigenic; phacolytic**
• Mediated by IgG antibodies: **Phacoantigenic**
• TM is clogged with macrophages: **Phacolytic**
For each statement, identify the **lens-related secondary OAG with which it is associated** (some have more than one answer):

<table>
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<th>Phacoantigenic glaucoma</th>
<th>Lens-particle glaucoma</th>
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- The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic
- Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic
- Mediated by IgG antibodies: Phacoantigenic
- **TM is clogged with macrophages**: Phacolytic

‘**TM clogged with macrophages**’ applies also to another form of secondary OAG—which one?
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic

Mediated by IgG antibodies: Phacoantigenic

**TM is clogged with macrophages**: Phacolytic

‘TM clogged with macrophages’ applies also to another form of secondary OAG—which one?
Hemolytic glaucoma
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic

Mediated by IgG antibodies: Phacoantigenic

**TM is clogged with macrophages**: Phacolytic

‘*TM clogged with macrophages*’ applies also to another form of secondary OAG—*which one?*

Hemolytic glaucoma

*In phacolytic glaucoma, the macrophages are full of* [two words].
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to **lens proteins** in AC: Phacoantigenic; **phacolytic**

Mediated by IgG antibodies: Phacoantigenic

**TM is clogged with macrophages**: Phacolytic

‘**TM clogged with macrophages**’ applies also to another form of secondary OAG—which one?

Hemolytic glaucoma

*In phacolytic glaucoma, the macrophages are full of lens proteins.*
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to **lens proteins** in AC: Phacoantigenic; **phacolytic**

Mediated by IgG antibodies: Phacoantigenic

**TM is clogged with macrophages**: Phacolytic

‘*TM clogged with macrophages’ applies also to another form of secondary OAG—which one?*

Hemolytic glaucoma

*In phacolytic glaucoma, the macrophages are full of lens proteins.*

*What are they full of in hemolytic glaucoma?*
For each statement, identify the lens-related secondary OAG with which it is associated (some have more than one answer)

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic
- Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic
- Mediated by IgG antibodies: Phacoantigenic
- **TM is clogged with macrophages**: Phacolytic

‘**TM clogged with macrophages**’ applies also to another form of secondary OAG—which one?
Hemolytic glaucoma

*In phacolytic glaucoma, the macrophages are full of lens proteins.*
*What are they full of in hemolytic glaucoma?*
Hemoglobin
The only one described in the *Glaucoma* book as ‘rare’: **Phacoantigenic**

Mediated by inflammatory response to lens proteins in AC: **Phacoantigenic; phacolytic**

Mediated by IgG antibodies: **Phacoantigenic**

TM is clogged with macrophages: **Phacolytic**

Chunks of cortex may be visible in AC:
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic

Mediated by IgG antibodies: Phacoantigenic

TM is clogged with macrophages: Phacolytic

Chunks of cortex may be visible in AC: Lens particle
Lens-particle glaucoma
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer):

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- Mediated by IgG antibodies: **Phacoantigenic**
- TM is clogged with macrophages: **Phacolytic**
- Chunks of cortex may be visible in AC: **Lens particle**
- Is also known as [condition name] *uveitis*: **
A

For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic
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TM is clogged with macrophages: Phacolytic

Chunks of cortex may be visible in AC: Lens particle

Is also known as [condition name] uveitis: Phacoantigenic

Capsule is intact:
• The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

• Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic

• Mediated by IgG antibodies: Phacoantigenic

• TM is clogged with macrophages: Phacolytic

• Chunks of cortex may be visible in AC: Lens particle

• Is also known as [condition name] *uveitis*: Phacoantigenic

• Capsule is intact: Phacolytic
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic

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TM is clogged with macrophages: Phacolytic

Chunks of cortex may be visible in AC: Lens particle

Is also known as [condition name] *uveitis*: Phacoantigenic

**Capsule is intact:** Phacolytic

What does this imply about the status of the capsule in phacoantigenic and lens-particle glaucoma?
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic

Mediated by IgG antibodies: Phacoantigenic

TM is clogged with macrophages: Phacolytic

Chunks of cortex may be visible in AC: Lens particle

Is also known as [condition name] *uveitis*: Phacoantigenic

**Capsule is intact:** Phacolytic

What does this imply about the status of the capsule in phacoantigenic and lens-particle glaucoma?

It implies (correctly) that the capsule is open in these conditions.
The only one described in the *Glaucoma* book as ‘rare’:

**Phacoantigenic**

- Mediated by inflammatory response to lens proteins in AC:
  - Phacoantigenic; phacolytic
- Mediated by IgG antibodies: Phacoantigenic
- TM is clogged with macrophages: Phacolytic
- Chunks of cortex may be visible in AC: Lens-particle
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Is also known as [condition name] *uveitis*: Phacoantigenic

Capsule is intact: Phacolytic

AC reaction is granulomatous:
The only one described in the *Glaucoma* book as ‘rare’: **Phacoantigenic**

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Is also known as [condition name] uveitis: Phacoantigenic

Capsule is intact: Phacolytic

AC reaction is granulomatous: Phacoantigenic

Is a reaction to *normal* lens proteins:
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic

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TM is clogged with macrophages: Phacolytic

Chunks of cortex may be visible in AC: Lens particle

Is also known as [condition name] uveitis: Phacoantigenic

Capsule is intact: Phacolytic

AC reaction is granulomatous: Phacoantigenic

Is a reaction to *normal* lens proteins: Phacoantigenic
The only one described in the *Glaucoma* book as ‘rare’:

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Mediated by inflammatory response to lens proteins in AC:

- Phacoantigenic; phacolytic

Mediated by IgG antibodies:

- Phacoantigenic

TM is clogged with macrophages:

- Phacolytic

Chunks of cortex may be visible in AC: Lens particle

Is also known as [condition name] *uveitis*: Phacoantigenic

Capsule is intact:

- Phacolytic

AC reaction is granulomatous:

- Phacoantigenic

Is a reaction to *normal* lens proteins: **Phacoantigenic**

---

*Why is it significant that phacoantigenic glaucoma involves an immune reaction to ‘normal’ lens proteins?*
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic

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Is also known as [condition name] uveitis: Phacoantigenic

Capsule is intact: Phacolytic

AC reaction is granulomatous: Phacoantigenic

Is a reaction to *normal* lens proteins: Phacoantigenic

Why is it significant that phacoantigenic glaucoma involves an immune reaction to ‘normal’ lens proteins?

In phakic eyes, minute amounts of lens proteins make their way through the capsule and into the AC. Because of this, normal lens proteins enjoy a certain level of immunologic privilege and are well tolerated by the eye.
The only one described in the *Glaucoma* book as ‘rare’:

*Phacoantigenic glaucoma*  
Mediated by inflammatory response to lens proteins in AC:  
*Phacoantigenic*  
Mediated by IgG antibodies: *Phacoantigenic*  
TM is clogged with macrophages: *Phacolytic*  
Chunks of cortex may be visible in AC: *Lens-particle glaucoma*  
Is also known as [condition name] *uveitis*: *Phacoantigenic*  
Capsule is intact: *Phacolytic*  
AC reaction is granulomatous: *Phacoantigenic*  
Is a reaction to *normal* lens proteins: *Phacoantigenic*  

Why is it significant that *phacoantigenic glaucoma* involves an immune reaction to ‘normal’ lens proteins?  
In phakic eyes, minute amounts of lens proteins make their way through the capsule and into the AC. Because of this, normal lens proteins enjoy a certain level of immunologic privilege and are well tolerated by the eye. However, violation of the capsule results in massive amounts of lens proteins spilling into the AC. If this influx disrupts the privilege, severe inflammation, ie, phacoantigenic uveitis—and glaucoma—may result.
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic

Mediated by IgG antibodies: Phacoantigenic

TM is clogged with macrophages: Phacolytic

Is a reaction to *normal* lens proteins: Phacoantigenic

Chunks of cortex may be visible in AC: Lens particle

Is also known as [condition name] *uveitis*: Phacoantigenic

Capsule is intact: Phacolytic

AC reaction is granulomatous: Phacoantigenic

Is a reaction to *denatured* lens proteins:
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic

Mediated by IgG antibodies: Phacoantigenic

TM is clogged with macrophages: Phacolytic

Is a reaction to normal lens proteins: Phacoantigenic

Chunks of cortex may be visible in AC: Lens particle

Is also known as [condition name] uveitis: Phacoantigenic

Capsule is intact: Phacolytic

AC reaction is granulomatous: Phacoantigenic

Is a reaction to denatured lens proteins: Phacolytic
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

*What does it mean to say a protein has been ‘denatured’?*

Is a reaction to denatured lens proteins: Phacolytic
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Is a reaction to *denatured* lens proteins: Phacolytic

**What does it mean to say a protein has been ‘denatured’?**
It means the protein has been forced out of its native conformation. Because a protein’s function is inextricably tied to its shape, denatured proteins do not behave as they do in their native form.
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

What does it mean to say a protein has been ‘denatured’?
It means the protein has been forced out of its native conformation. Because a protein’s function is inextricably tied to its shape, denatured proteins do not behave as they do in their native form.

Can you give an example of protein denaturation?

Is a reaction to *denatured* lens proteins: Phacolytic
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

*What does it mean to say a protein has been ‘denatured’?*  
It means the protein has been forced out of its native conformation. Because a protein’s function is inextricably tied to its shape, denatured proteins do not behave as they do in their native form.

*Can you give an example of protein denaturation?*  
Consider egg albumin. In its native state, it’s a clear liquid. But if sufficient heat is applied, it becomes a white solid. (And if sufficient salsa is applied to the white solid, it becomes delish.)

Is a reaction to *denatured* lens proteins: Phacolytic

For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma
For each statement, identify the lens-related secondary OAG with which it is associated (some have more than one answer)

- Phacolytic glaucoma
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**Q**

- The only one described in the *Glaucoma* book as ‘rare’: **Phacoantigenic**

  *What does it mean to say a protein has been ‘denatured’?*
  It means the protein has been forced out of its native conformation. Because a protein’s function is inextricably tied to its shape, denatured proteins do not behave as they do in their native form.

  *Can you give an example of protein denaturation?*
  Consider egg albumin. In its native state, it’s a clear liquid. But if sufficient heat is applied, it becomes a white solid. (And if sufficient salsa is applied to the white solid, it becomes delish.)

  *What role does denaturation play in the inflammatory process?*

- Is a reaction to *denatured* lens proteins: **Phacolytic**
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

**What does it mean to say a protein has been ‘denatured’?**

It means the protein has been forced out of its native conformation. Because a protein’s function is inextricably tied to its shape, denatured proteins do not behave as they do in their native form.

**Can you give an example of protein denaturation?**

Consider egg albumin. In its native state, it’s a clear liquid. But if sufficient heat is applied, it becomes a white solid. (And if sufficient salsa is applied to the white solid, it becomes delish.)

**What role does denaturation play in the inflammatory process?**

Recall that normal lens proteins enjoy a degree of immunologic privilege. In contrast, *denatured* proteins enjoy no such privilege, and thus tend to attract macrophages in large numbers.

**Is a reaction to *denatured* lens proteins:** Phacolytic
Q

For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

- **Phacolytic glaucoma**
- **Phacoantigenic glaucoma**
- **Lens-particle glaucoma**

- The only one described in the *Glaucoma* book as ‘rare’: **Phacoantigenic**
- Mediated by inflammatory response to lens proteins in AC: **Phacoantigenic; phacolytic**
- Mediated by IgG antibodies: **Phacoantigenic**
- TM is clogged with macrophages: **Phacolytic**
- Chunks of cortex may be visible in AC: **Lens particle**
- Is also known as [condition name] *uveitis*: **Phacoantigenic**
- Capsule is intact: **Phacolytic**
- AC reaction is granulomatous: **Phacoantigenic**
- Is a reaction to *normal* lens proteins: **Phacoantigenic**
- Is a reaction to *denatured* lens proteins: **Phacolytic**
- The presence of KP is a key clinical finding:
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; Phacolytic

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Chunks of cortex may be visible in AC: Lens particle

Is also known as [condition name] uveitis: Phacoantigenic

Capsule is intact: Phacolytic

AC reaction is granulomatous: Phacoantigenic

Is a reaction to *normal* lens proteins: Phacoantigenic

Is a reaction to *denatured* lens proteins: Phacolytic

The presence of KP is a key clinical finding: Phacoantigenic
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic

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Is also known as [condition name] *uveitis*: Phacoantigenic

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AC reaction is granulomatous: Phacoantigenic

Is a reaction to *normal* lens proteins: Phacoantigenic

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The presence of KP is a key clinical finding: Phacoantigenic

Are the KP granulomatous, or nongranulomatous?
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic

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Is also known as [condition name] uveitis: Phacoantigenic

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Granulomatous
Phacoantigenic glaucoma: Granulomatous KP
The only one described in the *Glaucoma* book as ‘rare’: Phacoantigenic

Mediated by inflammatory response to lens proteins in AC: Phacoantigenic; phacolytic

Mediated by IgG antibodies: Phacoantigenic

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Is also known as [condition name] *uveitis*: Phacoantigenic

Capsule is intact: Phacolytic

AC reaction is granulomatous: Phacoantigenic

Is a reaction to *normal* lens proteins: Phacoantigenic

Is a reaction to *denatured* lens proteins: Phacolytic

The presence of KP is a key clinical finding: Phacoantigenic

The one most likely to have a very high IOP:
The only one described in the *Glaucoma* book as ‘rare’: **Phacoantigenic**

Mediated by inflammatory response to lens proteins in AC: **Phacoantigenic; phacolytic**

Mediated by IgG antibodies: **Phacoantigenic**

TM is clogged with macrophages: **Phacolytic**

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Is also known as [condition name] *uveitis*: **Phacoantigenic**

Capsule is intact: **Phacolytic**

AC reaction is granulomatous: **Phacoantigenic**

Is a reaction to *normal* lens proteins: **Phacoantigenic**

Is a reaction to *denatured* lens proteins: **Phacolytic**

The presence of KP is a key clinical finding: **Phacoantigenic**

The one most likely to have a very high IOP: **Phacolytic**
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer):

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- aka **phacoanaphylactic glaucoma**: 
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

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- Lens-particle glaucoma

**Q**

- aka phacoanaphylactic glaucoma: Phacoantigenic

Why is phacoanaphylactic glaucoma actually a misnomer?
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer):

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- **aka** *phacoanaphylactic glaucoma*: Phacoantigenic

*Why is phacoanaphylactic glaucoma actually a misnomer?* Because the condition is not a Type 1 (anaphylactic) reaction.
For each statement, identify the lens-related secondary OAG with which it is associated (some have more than one answer)

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

Q

- aka phacoanaphylactic glaucoma: Phacoantigenic

Why is phacoanaphylactic glaucoma actually a misnomer?
Because the condition is not a Type 1 (anaphylactic) reaction

What characteristics inherent to true anaphylaxis are missing in phacoantigenic glaucoma?
phacoanaphylactic glaucoma: Phacoantigenic

Why is phacoanaphylactic glaucoma actually a misnomer? Because the condition is not a Type 1 (anaphylactic) reaction

What characteristics inherent to true anaphylaxis are missing in phacoantigenic glaucoma? The involvement of IgE, mast cells and basophils
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer):

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- aka *phacoanaphylactic glaucoma*: **Phacoantigenic**
- Usually unilateral:
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

| Phacolytic glaucoma | Phacoantigenic glaucoma | Lens-particle glaucoma |

- aka *phacoanaphylactic glaucoma*: Phacoantigenic
- Usually unilateral: **All of them**
Q

For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer):

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- **aka phacoanaphylactic glaucoma**: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an *adaptive* immune response:
A

For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- **aka** *phacoanaphylactic glaucoma*: Phacoantigenic
- Usually unilateral: **All of them**
- Is mediated by an *adaptive* immune response: Phacoantigenic
Q For each statement, identify the lens-related secondary OAG with which it is associated (some have more than one answer)

Phacolytic glaucoma  Phacoantigenic glaucoma  Lens-particle glaucoma

- aka *phacoanaphylactic glaucoma*: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an *adaptive* immune response: Phacoantigenic
- Associated with mature/hypermature cataract:
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

**Phacolytic glaucoma**  **Phacoantigenic glaucoma**  **Lens-particle glaucoma**

- aka *phacoanaphylactic glaucoma*: **Phacoantigenic**
- Usually unilateral: **All of them**
- Is mediated by an *adaptive* immune response: **Phacoantigenic**
- Associated with mature/hypermature cataract: **Phacolytic**
Q

For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

Phacolytic glaucoma  Phacoantigenic glaucoma  Lens-particle glaucoma

- aka *phacoanaphylactic glaucoma*: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an *adaptive* immune response: Phacoantigenic
- Associated with *mature/hypermature cataract*: Phacolytic

*What is a mature cataract?*
Q/A
For each statement, identify the lens-related secondary OAG with which it is associated (some have more than one answer)
Phacolytic glaucoma  Phacoantigenic glaucoma  Lens-particle glaucoma

- aka phacoanaphylactic glaucoma: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an adaptive immune response: Phacoantigenic
- Associated with mature/hypermature cataract: Phacolytic

What is a mature cataract?
A cataract that has progressed to involve the entire lens
A aka phacoanaphylactic glaucoma: Phacoantigenic
● Usually unilateral: All of them
● Is mediated by an adaptive immune response: Phacoantigenic
● Associated with mature/hypermature cataract: Phacolytic

What is a mature cataract?
A cortical cataract that has progressed to involve the entire lens cortex
Mature cataract
For each statement, identify the lens-related secondary OAG with which it is associated (some have more than one answer)

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- **aka phacoanaphylactic glaucoma**: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an adaptive immune response: Phacoantigenic
- Associated with mature/hypermature cataract: Phacolytic

**What is a mature cataract?**
A cortical cataract that has progressed to involve the entire lens cortex

**What is a hypermature cataract?**
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

- **Phacolytic glaucoma**
- **Phacoantigenic glaucoma**
- **Lens-particle glaucoma**

- aka *phacoanaphylactic glaucoma*: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an *adaptive* immune response: Phacoantigenic
- Associated with mature/hypermature cataract: **hypermature cataract**

*What is a mature cataract?*
A cortical cataract that has progressed to involve the entire lens cortex

*What is a hypermature cataract?*
Mature cataracts may absorb water, transforming them into an *cortical cataract.*
For each statement, identify the lens-related secondary OAG with which it is associated (some have more than one answer):

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- aka phacoanaphylactic glaucoma: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an adaptive immune response: Phacoantigenic
- Associated with mature/hypermature cataract: Phacolytic

What is a mature cataract?
A cortical cataract that has progressed to involve the entire lens cortex

What is a hypermature cataract?
Mature cataracts may absorb water, transforming them into an intumescent cortical cataract.
A aka *phacoanaphylactic glaucoma*: Phacoantigenic
● Usually unilateral: All of them
● Is mediated by an *adaptive* immune response: Phacoantigenic
● Associated with mature/ hypermature cataract: Phacolytic

**For each statement, identify the lens-related secondary OAG with which it is associated (some have more than one answer)**

Phacolytic glaucoma  Phacoantigenic glaucoma  Lens-particle glaucoma

---

**What is a mature cataract?**

A cortical cataract that has progressed to involve the entire lens cortex

**What is a hypermature cataract?**

Mature cataracts may absorb water, transforming them into an *intumescent* cortical cataract. A *hypermature cataract* results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

Phacolytic glaucoma  Phacoantigenic glaucoma  Lens-particle glaucoma

- aka *phacoanaphylactic glaucoma*: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an *adaptive* immune response: Phacoantigenic
- Associated with mature/*hypermature cataract*: Phacolytic

---

**What is a mature cataract?**
A cortical cataract that has progressed to involve the entire lens cortex

**What is a hypermature cataract?**
Mature cataracts may absorb water, transforming them into an *intumescent cortical cataract*. A *hypermature cataract* results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

*Take note of the stages:*

Mature cataract → ?
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer).

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- **aka phacoanaphylactic glaucoma**: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an *adaptive* immune response: Phacoantigenic
- Associated with mature/hypermature cataract: Phacolytic

---

**What is a mature cataract?**
A cortical cataract that has progressed to involve the entire lens cortex

**What is a hypermature cataract?**
Mature cataracts may absorb water, transforming them into an *intumescent cortical cataract*. A *hypermature cataract* results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

**Take note of the stages:**

Mature cataract → intumescent cataract → ?
aka phacoanaphylactic glaucoma: Phacoantigenic
Usually unilateral: All of them
Is mediated by an adaptive immune response: Phacoantigenic
Associated with mature/hypermature cataract: Phacolytic

What is a mature cataract?
A cortical cataract that has progressed to involve the entire lens cortex

What is a hypermature cataract?
Mature cataracts may absorb water, transforming them into an intumescent cortical cataract. A hypermature cataract results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

Take note of the stages:
Mature cataract → intumescent cataract → hypermature cataract
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- aka **phacoanaphylactic glaucoma**: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an **adaptive immune response**: Phacoantigenic
- Associated with mature/hypermature cataract: Phacolytic

---

**What is a mature cataract?**
A cortical cataract that has progressed to involve the entire lens cortex

**What is a hypermature cataract?**
Mature cataracts may absorb water, transforming them into an **intumescent cortical cataract**. A **hypermature cataract** results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

---

**Take note of the stages:**

Mature cataract → intumescent cataract → hypermature cataract

Cataract absorbs water → Cataract leaks water
aka phacoanaphylactic glaucoma: Phacoantigenic
Usually unilateral: All of them
Is mediated by an adaptive immune response: Phacoantigenic
Associated with mature/hypermature cataract: Phacolytic

What is a **mature cataract**?
A cortical cataract that has progressed to involve the entire lens cortex

What is a hypermature cataract?
Mature cataracts may absorb water, transforming them into an intumescent cortical cataract. A hypermature cataract results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

Take note of the stages:

- Mature cataract
- Intumescent cataract
- Hypermature cataract

Cataract absorbs water

What happens
Q

For each statement, identify the **lens-related secondary OAG with which it is associated** (some have more than one answer)

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- **aka** *phacoanaphylactic glaucoma*: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an **adaptive** immune response: Phacoantigenic
- Associated with mature/*hypermature cataract*: Phacolytic

---

**What is a mature cataract?**
A cortical cataract that has progressed to involve the entire lens cortex.

**What is a hypermature cataract?**
Mature cataracts may **absorb water**, transforming them into an *intumescent cortical cataract*. A **hypermature cataract** results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

---

**Take note of the stages:**

Mature cataract → intumescent cataract → hypermature cataract

Cataract *absorbs* water  

*What happens*  

Cataract *leaks* water
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- aka **phacoanaphylactic glaucoma**: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an *adaptive* immune response: Phacoantigenic
- Associated with mature/*hypermature cataract**: Phacolytic

---

**What is a mature cataract?**

A cortical cataract that has progressed to involve the entire lens cortex.

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**What is a hypermature cataract?**

Mature cataracts may absorb water, transforming them into an *intumescent cortical cataract*. A *hypermature cataract* results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

---

**Take note of the stages:**

Mature cataract → intumescent cataract → hypermature cataract

Cataract *absorbs* water *← What happens →* Cataract *leaks* water*

*and proteins*
Q

For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

- **Phacolytic glaucoma**
- **Phacoantigenic glaucoma**
- **Lens-particle glaucoma**

- aka **phacoanaphylactic glaucoma**: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an **adaptive immune response**: Phacoantigenic
- Associated with mature/hypermature cataract: **Phacolytic**

---

**What is a mature cataract?**

A cortical cataract that has progressed to involve the entire lens cortex

**What is a hypermature cataract?**

Mature cataracts may absorb water, transforming them into an intumescent cortical cataract. A hypermature cataract results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

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**All three of these pose a particular challenge during an early, crucial step in cataract surgery. What step, and what challenge?**

---

**Take note of the stages:**

Mature cataract ➔ intumescent cataract ➔ hypermature cataract
A aka *phacoanaphylactic glaucoma*: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an *adaptive* immune response: Phacoantigenic
- Associated with mature/hypermature cataract: Phacolytic

---

What is a mature cataract?
A cortical cataract that has progressed to involve the entire lens cortex

What is a hypermature cataract?
Mature cataracts may absorb water, transforming them into an intumescent cortical cataract. A *hypermature cataract* results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

---

*All three of these pose a particular challenge during an early, crucial step in cataract surgery. What step, and what challenge?*

For all three stages, the red reflex is completely obscured. As most cataract surgeons rely on the red reflex to visualize the anterior capsule during capsulorrhexis, this step cannot be performed in a conventional manner.

---

*Take note of the stages:*

Mature cataract ➔ intumescent cataract ➔ hypermature cataract
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- **aka phacoanaphylactic glaucoma:** Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an *adaptive* immune response: Phacoantigenic
- Associated with mature/hypermature cataract: Phacolytic

---

**Q**

What is a mature cataract?

A  cortical  cataract that has progressed to involve the entire lens cortex

What is a hypermature cataract?

Mature cataracts may absorb water, transforming them into an intumescent cortical cataract. A hypermature cataract results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

---

**Q**

All three of these pose a particular challenge during an early, crucial step in cataract surgery. **What step, and what challenge?**

For all three stages, the red reflex is completely obscured. As most cataract surgeons rely on the red reflex to visualize the anterior capsule during capsulorrhexis, this step cannot be performed in a conventional manner.

**What step do most surgeons take to facilitate capsulorrhexis in these cases?**

Take note of the stages:

- Mature cataract
- Intumescent cataract
- Hypermature cataract
aka phacoanaphylactic glaucoma: Phacoantigenic
Usually unilateral: All of them
Is mediated by an adaptive immune response: Phacoantigenic
Associated with mature/hypermature cataract: Phacolytic

All three of these pose a particular challenge during an early, crucial step in cataract surgery. What step, and what challenge?
For all three stages, the red reflex is completely obscured. As most cataract surgeons rely on the red reflex to visualize the anterior capsule during capsulorrhexis, this step cannot be performed in a conventional manner.

What step do most surgeons take to facilitate capsulorrhexis in these cases?
They stain the anterior capsule with trypan blue.

Take note of the stages:
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer).

- **Phacolytic glaucoma**
- **Phacoantigenic glaucoma**
- **Lens-particle glaucoma**

- aka *phacoanaphylactic glaucoma*: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an *adaptive immune response*: Phacoantigenic
- Associated with mature/hypermature cataract: Phacolytic

**What is a mature cataract?**
A cortical cataract that has progressed to involve the entire lens cortex

**What is a hypermature cataract?**
Mature cataracts may absorb water, transforming them into an intumescent cortical cataract. A hypermature cataract results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

---

_A cauliflower makes more of a splash than a man_.

All three of these pose a particular challenge during an early, crucial step in cataract surgery. _What step, and what challenge?_ For all three stages, the red reflex is completely obscured. As most cataract surgeons rely on the red reflex to visualize the anterior capsule during capsulorrhexis, this step cannot be performed in a conventional manner.

_What step do most surgeons take to facilitate capsulorrhexis in these cases?_ They stain the anterior capsule with trypan blue

_Take note of the stages:_

- **Mature cataract**
- **intumescent cataract**
- **hypermature cataract**
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

**Phacolytic glaucoma**  **Phacoantigenic glaucoma**  **Lens-particle glaucoma**

- aka *phacoanaphylactic glaucoma*: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an *adaptive* immune response: Phacoantigenic
- Associated with mature/ **hypermature cataract**: Phacolytic

**Q**

What is a **mature cataract**?
- A cortical cataract that has progressed to involve the entire lens cortex

What is a **hypermature cataract**?
- Mature cataracts may absorb water, transforming them into an *intumescent* cortical cataract. A **hypermature cataract** results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

**Take note of the stages:**

- Mature cataract
- **intumescent cataract**
- Hypermature cataract

*Let’s drill down on intumescent cataracts for a moment. In this context, what does intumescent mean?*

*All three of these pose a particular challenge during an early, crucial step in cataract surgery. What step, and what challenge?*

For all three stages, the red reflex is completely obscured. As most cataract surgeons rely on the red reflex to visualize the anterior capsule during capsulorrhexis, this step cannot be performed in a conventional manner.

*What step do most surgeons take to facilitate capsulorrhexis in these cases?*
- They stain the anterior capsule with **trypan blue**

*Take note of the stages:*
For each statement, identify the lens-related secondary OAG with which it is associated (some have more than one answer).

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- aka *phacoanaphylactic glaucoma*: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an *adaptive* immune response: Phacoantigenic
- Associated with mature/ *hypermature cataract*: Phacolytic

**Phacolytic glaucoma**

Phacolytic glaucoma is a type of lens-related secondary open-angle glaucoma that occurs when a mature or hypermature cataract blocks the drainage of aqueous humor, leading to increased intraocular pressure. This condition is characterized by the presence of a large amount of proteinaceous fluid (phacolyte) within the anterior chamber, which can result in a decrease in corneal transparency and a red reflex that is difficult to visualize.

**Phacoantigenic glaucoma**

Phacoantigenic glaucoma is caused by the immunological reaction against phacoantigens released during cataract surgery. It usually presents as a unilateral condition and is characterized by an adaptive immune response. This type of glaucoma is associated with mature or hypermature cataracts.

**Lens-particle glaucoma**

Lens-particle glaucoma occurs when lens particles become trapped in the trabecular meshwork, blocking aqueous humor outflow and causing an increase in intraocular pressure. This condition is typically associated with mature or hypermature cataracts.

---

**What is a mature cataract?**

A cortical cataract that has progressed to involve the entire lens cortex.

**What is a hypermature cataract?**

Mature cataracts may absorb water, transforming them into an intumescent cortical cataract. A hypermature cataract results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

---

**Take note of the stages:**

- Mature cataract
- Intumescent cataract
- Hypermature cataract

---

**What step do most surgeons take to facilitate capsulorrhexis in these cases?**

They stain the anterior capsule with *trypan blue*.

---

*Let’s drill down on intumescent cataracts for a moment. In this context, what does intumescent mean?*

It means ‘swollen.’ As mentioned a few slides ago, the event that transforms a *mature* cataract into an *intumescent* cataract is absorption of water, and this absorption results in swelling of the lens.

---

*What effect does swelling have on the internal dynamics of the lens?*

It increases the pressure within the lens.
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

- **aka phacoanaphylactic glaucoma:** Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an *adaptive* immune response: Phacoantigenic
- Associated with mature/hypermature cataract: Phacolytic

---

**Q**

1. What is a mature cataract?
   - A cortical cataract that has progressed to involve the entire lens cortex

2. What is a hypermature cataract?
   - Mature cataracts may absorb water, transforming them into an intumescent cortical cataract. A hypermature cataract results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

---

*Let’s drill down on intumescent cataracts for a moment. In this context, what does intumescent mean?*

It means ‘swollen.’ As mentioned a few slides ago, the event that transforms a mature cataract into an intumescent cataract is absorption of water, and this absorption results in swelling of the lens.

---

*What effect does swelling have on the internal dynamics of the lens?*

It increases the pressure within the lens.

---

*What step do most surgeons take to facilitate capsulorrhexis in these cases?*

They stain the anterior capsule with trypan blue.

---

*Take note of the stages:*

Mature cataract → **intumescent cataract** → hypermature cataract
A

For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

| Phacolytic glaucoma | Phacoantigenic glaucoma | Lens-particle glaucoma |

- **aka** *phacoanaphylactic glaucoma*: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an *adaptive* immune response: Phacoantigenic
- Associated with mature/ **hypermature cataract**: Phacolytic

---

Let’s drill down on intumescent cataracts for a moment. *In this context, what does intumescent mean?*

It means ‘swollen.’ As mentioned a few slides ago, the event that transforms a mature cataract into an intumescent cataract is absorption of water, and this absorption results in swelling of the lens.

What effect does swelling have on the internal dynamics of the lens?

It increases the pressure within the lens.

---

What step do most surgeons take to facilitate capsulorrhexis in these cases?

They stain the anterior capsule with trypan blue.

---

**Take note of the stages:**

Mature cataract → **intumescent cataract** → hypermature cataract
aka *phacoanaphylactic glaucoma*: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an *adaptive* immune response: Phacoantigenic
- Associated with mature/hypermature cataract: Phacolytic

For each statement, identify the *lens-related secondary OAG* with which it is associated (some have more than one answer)

- Phacolytic glaucoma
- Phacoantigenic glaucoma
- Lens-particle glaucoma

**Q**

What is a mature cataract?
- A cortical cataract that has progressed to involve the entire lens cortex

What is a hypermature cataract?
- Mature cataracts may absorb water, transforming them into an intumescent cortical cataract. A hypermature cataract results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

Take note of the stages:

- Mature cataract
- Intumescent cataract
- Hypermature cataract

For all three stages, the red reflex is completely obscured. As most cataract surgeons rely on the red reflex to visualize the anterior capsule during capsulorrhexis, this step cannot be performed in a conventional manner.

What step do most surgeons take to facilitate capsulorrhexis in these cases?
- They stain the anterior capsule with trypan blue

Let’s drill down on intumescent cataracts for a moment. In this context, what does intumescent mean?

As if obscuration of the red reflex wasn’t enough, the increased intralenticular pressure of an intumescent cataract poses an additional challenge during capsulorrhexis—what is it?

What effect does swelling have on the internal dynamics of the lens?
- It increases the pressure within the lens

As if obscuration of the red reflex wasn’t enough, the increased intralenticular pressure of an intumescent cataract poses an additional challenge during capsulorrhexis—what is it?

What step do most surgeons take to facilitate capsulorrhexis in these cases?
- They stain the anterior capsule with trypan blue
Phacoanaphylactic glaucoma: Phacoantigenic
Usually unilateral: All of them
Is mediated by an adaptive immune response: Phacoantigenic
Associated with mature/hypermature cataract: Phacoanaphylactic

Let's drill down on intumescent cataracts for a moment. In this context, what does intumescent mean?

As if obscuration of the red reflex wasn’t enough, the increased intralenticular pressure of an intumescent cataract poses an additional challenge during capsulorrhexis—what is it?

When the surgeon makes the initial rent in the capsule, the increased pressure within an intumescent cataract may cause the rent to suddenly and uncontrollably extend to the periphery of the lens.

What effect does swelling have on the internal dynamics of the lens?

It increases the pressure within the lens.

What step do most surgeons take to facilitate capsulorrhexis in these cases?
They stain the anterior capsule with trypan blue.

Take note of the stages:

Mature cataract → intumescent cataract → hypermature cataract
aka *phacoanaphylactic glaucoma*: Phacoantigenic

Usually unilateral: All of them

Is mediated by an *adaptive* immune response: Phacoantigenic

Associated with mature/hypermature cataract: Phacolytic

---

**What is a mature cataract?**

A cortical cataract that has progressed to involve the entire lens cortex

**What is a hypermature cataract?**

Mature cataracts may absorb water, transforming them into an intumescent cortical cataract. A hypermature cataract results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

Take note of the stages:

- Mature cataract
- Intumescent cataract
- Hypermature cataract

---

**Let's drill down on intumescent cataracts for a moment. In this context, what does intumescent mean?**

As if obscuration of the red reflex wasn't enough, the increased intralenticular pressure of an intumescent cataract poses an additional challenge during capsulorrhexis—what is it?

When the surgeon makes the initial rent in the capsule, the increased pressure within an intumescent cataract may cause the rent to suddenly and uncontrollably extend to the periphery.

*If/when the rent runs peripherally, what is the resulting appearance of the lens?*

Recall that, because of red-reflex obscuration, they stain the anterior capsule with *trypan blue*. Thus, after the rent runs out, the surgeon sees a white stripe (the cataract) between two areas of blue (the undisturbed, trypan blue-stained capsule).
For each statement, identify the lens-related secondary OAG with which it is associated (some have more than one answer)

Phacolytic glaucoma  Phacoantigenic glaucoma  Lens-particle glaucoma

- aka phacoanaphylactic glaucoma: Phacoantigenic
- Usually unilateral: All of them
- Is mediated by an adaptive immune response: Phacoantigenic
- Associated with mature/hypermature cataract: Phacolytic

Let’s drill down on intumescent cataracts for a moment. In this context, what does intumescent mean?

As if obscuration of the red reflex wasn’t enough, the increased intralenticular pressure of an intumescent cataract poses an additional challenge during capsulorrhexis—what is it?

When the surgeon makes the initial rent in the capsule, the increased pressure within an intumescent cataract may cause the rent to suddenly and uncontrollably extend to the periphery. What effect does swelling have on the internal dynamics of the lens?

It increases the pressure within the lens.

As if obscuration of the red reflex wasn’t enough, the increased intralenticular pressure of an intumescent cataract poses an additional challenge during capsulorrhexis—what is it?

If/when the rent runs peripherally, what is the resulting appearance of the lens?

Recall that, because of red-reflex obscuration, trypan blue is used in all these cases.

Trypan blue

What step, and what challenge?

For all three stages, the red reflex is completely obscured. As most cataract surgeons rely on the red reflex to visualize the anterior capsule during capsulorrhexis, this step cannot be performed in a conventional manner.

What step do most surgeons take to facilitate capsulorrhexis in these cases?

They stain the anterior capsule with trypan blue.
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

**Phacoanaphylactic glaucoma**  
**Phacoantigenic glaucoma**  
**Lens-particle glaucoma**

**Phacolytic glaucoma**  
**Phacoantigenic glaucoma**  
**Lens-particle glaucoma**

**Phacoantigenic glaucoma**

Usually unilateral: All of them

Is mediated by an **adaptive immune response**

Associated with mature/hypermature cataract: Phacolytic

**Phacolytic glaucoma**

**Phacoantigenic glaucoma**

**Lens-particle glaucoma**

---

What is a **mature cataract**?

A cortical cataract that has progressed to involve the entire lens cortex

What is a **hypermature cataract**?

Mature cataracts may absorb water, transforming them into an intumescent cortical cataract. A **hypermature cataract** results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

---

Take note of the stages:

Mature cataract  
**Intumescent cataract**  
Hypermature cataract

---

A **intumescent cataract**

All three of these pose a particular challenge during an early, crucial step in cataract surgery. What step, and what challenge?

For all three stages, the red reflex is completely obscured. As most cataract surgeons rely on the red reflex to visualize the anterior capsule during capsulorrhexis, this step cannot be performed in a conventional manner.

What step do most surgeons take to facilitate capsulorrhexis in these cases?

They stain the anterior capsule with **trypan blue**.

---

Let's drill down on **intumescent cataracts** for a moment. In this context, what does **intumescent** mean?

It means 'swollen.' As mentioned a few slides ago, the event that transforms a mature cataract into an intumescent cataract is absorption of water, and this absorption results in swelling of the lens.

What effect does swelling have on the internal dynamics of the lens?

It increases the pressure within the lens.

As if obscuration of the red reflex wasn’t enough, the increased intralenticular pressure of an intumescent cataract poses an additional challenge during capsulorrhexis—what is it?

When the surgeon makes the initial rent in the capsule, the increased pressure within an intumescent cataract may cause the rent to suddenly and uncontrollably extend to the periphery.

If/when the rent runs peripherally, what is the resulting appearance of the lens?

Recall that, because of red-reflex obscuration, **trypan blue** is used in all these cases. Thus, after the rent runs out, the surgeon sees a white stripe (the cataract) between two areas of blue (the undisturbed, trypan blue-stained capsule). They stain the anterior capsule with trypan blue.
Phacoanaphylactic glaucoma: Phacoantigenic

Usually unilateral: All of them

Is mediated by an adaptive immune response: Phacoantigenic

Associated with mature/hypermature cataract: Phacolytic

For each statement, identify the lens-related secondary OAG with which it is associated (some have more than one answer)

Phacolytic glaucoma       Phacoantigenic glaucoma    Lens-particle glaucoma

What is a mature cataract?

A cortical cataract that has progressed to involve the entire lens cortex

What is a hypermature cataract?

Mature cataracts may absorb water, transforming them into an intumescent cortical cataract. A hypermature cataract results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

Take note of the stages:

Mature cataract  intumescent cataract  hypermature cataract

Q

intumescent cataract

All three of these pose a particular challenge during an early, crucial step in cataract surgery. What step, and what challenge?

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This appearance has led to a memorable name for this finding. What is it?

It is known as 'Argentinian flag sign'.
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer):

**Phacoanaphylactic glaucoma**

**Phacoantigenic glaucoma**

**Lens-particle glaucoma**

Phacolytic glaucoma       Phacoantigenic glaucoma    Lens-particle glaucoma

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**What is a mature cataract?**

A cortical cataract that has progressed to involve the entire lens cortex.

**What is a hypermature cataract?**

Mature cataracts may absorb water, transforming them into an intumescent cortical cataract. A hypermature cataract results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.

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Recall that, because of red-reflex obscuration, trypan blue is used in all these cases. They stain the anterior capsule with trypan blue. When the rent runs out, the surgeon sees a white stripe between two areas of blue (the cataract) between the blue-stained capsule.

This appearance has led to a memorable name for this finding. What is it?

It is known as ‘**Argentinian flag sign**’.
Q: For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

- aka *phacoanaphylactic glaucoma*: Phacoantigenic
- Usually unilateral: All of them
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**Phacolytic glaucoma**  **Phacoantigenic glaucoma**  **Lens-particle glaucoma**

---

*When faced with an intumescent cataract, what can the surgeon do to minimize the likelihood of seeing an Argentinian flag?*

---

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*They stain the anterior capsule with* **trypan blue**

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*Take note of the stages:*

- Mature cataract
- **intumescent cataract**
- Hypermature cataract
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

**Phacolytic glaucoma**  **Phacoantigenic glaucoma**  **Lens-particle glaucoma**

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When faced with an intumescent cataract, what can the surgeon do to minimize the likelihood of seeing an Argentinian flag?

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-- Counteract the positive pressure within the lens by filling the AC with a high-viscosity OVD
-- Reduce intralenticular pressure by aspirating cortical material immediately upon creating the initial rent

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What is a mature cataract?
A cortical cataract that has progressed to involve the entire lens cortex

What is a hypermature cataract?
Mature cataracts may absorb water, transforming them into an intumescent cortical cataract. A hypermature cataract results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule. Finally: What stage occurs after the hypermature stage?

Take note of the stages:
Mature cataract → intumescent cataract → hypermature cataract
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

| Phacolytic glaucoma | Phacoantigenic glaucoma | Lens-particle glaucoma |

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**Finally: What stage occurs after the hypermature stage?**

**Take note of the stages:**

Mature cataract → intumescent cataract → hypermature cataract

*Morganian cataract*
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

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Take note of the stages:

- Mature cataract
- Intumescent cataract
- Hypermature cataract

---

What change occurs as a cortical cataract progresses from the hypermature to the morgagnian stage?

Further and extensive liquefaction of the cortical material

---

The dense brown nuclear cataract is observed to be freely mobile within the liquified remnants of the cortical cataract
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**What change occurs as a cortical cataract progresses from the hypermature to the morgagnian stage?**
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**What is the slit-lamp appearance of a morgagnian cataract?**
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For each statement, identify the lens-related secondary OAG with which it is associated (some have more than one answer)

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*Take note of the stages:*
- Mature cataract
- Intumescent cortical cataract
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Morgagnian cataract
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What effect does the leaking of water and proteins have on the volume of the cataract?
It reduces it significantly

A mature cataract may become *intumescent*, meaning it has the appearance of an *intumescent cortical cataract*. A *hypermature cataract* results when an intumescent cataract begins leaking water and denatured proteins through its intact anterior capsule.
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

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*This reduction in cataract volume is responsible for a classic finding in hypermature cataracts. What is it?*

A hypermature cataract results when an intumescent cortical cataract begins **leaking water and denatured proteins through its intact anterior capsule.**
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<tr>
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**What is it?**

The anterior capsule is *sign and sign*

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Hypermature cataract. Note the capsular wrinkling.
Q

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How should lens-particle glaucoma be managed?
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

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*How should lens-particle glaucoma be managed?*

If possible, medical management should be employed to control the inflammation and IOP until the eye can absorb the inciting lens material.
For each statement, identify the lens-related secondary OAG with which it is associated (some have more than one answer)

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*If medical management proves inadequate, what is the next step?*
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*How should lens-particle glaucoma be managed?*
If possible, medical management should be employed to control the inflammation and IOP until the eye can absorb the inciting lens material

*If medical management proves inadequate, what is the next step?*
Surgical removal of the offending material
Q

For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

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There are two broad categories of immune response—what are they?
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer)

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There are two broad categories of immune response—**what are they?**

**Innate** and **adaptive**
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There are two broad categories of immune response—what are they?

**Innate** and **adaptive**

In general, what is the nature of each, and how do they differ?
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There are two broad categories of immune response—what are they?  **Innate** and **adaptive**

*In general, what is the nature of each, and how do they differ?*

The adaptive immune response involves ‘education,’ with surveillance cells learning to recognize and remember foreign material.
Phacoanaphylactic glaucoma (aka phacoantigenic glaucoma)

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**There are two broad categories of immune response—what are they?**

**Innate** and **adaptive**

**In general, what is the nature of each, and how do they differ?**

The adaptive immune response involves ‘education,’ with surveillance cells learning to recognize and remember foreign material. OTOH, the innate (or natural) immune response does not require education—it relies on ‘preprogrammed’ immune cells to recognize foreign material encountered in tissue or blood.
Q

For each statement, identify the lens-related secondary OAG with which it is associated (some have more than one answer)

- Phacolytic glaucoma
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**There are two broad categories of immune response—what are they?**

**Innate** and **adaptive**

**In general, what is the nature of each, and how do they differ?**

The adaptive immune response involves ‘education,’ with surveillance cells learning to recognize and remember foreign material. OTOH, the innate (or **natural**) immune response does not require education—it relies on ‘preprogrammed’ immune cells to recognize foreign material encountered in tissue or blood.

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**What are the two main effector cell types of innate immunity?**

Neutrophils and macrophages
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What are the two main effector cell types of innate immunity? Neutrophils and macrophages

And now, an overly long sidebar regarding immunology and the lens-related secondary OAGs: Some clinicians reserve the term immune response for clinical situations in which an adaptive immune response is involved. That is, if the clinical situation involves only an innate response, such clinicians opt to use the more general term inflammation in describing the clinical picture. Take the Lens book. It states that "phacoantigenic uveitis" (it does not use the term phacoantigenic glaucoma) is "immune-mediated." However, it pointedly states that phacolytic glaucoma does not elicit an immune response. This is an example of the immune response = adaptive response mindset mentioned above. Likewise, the Path book addresses phacoantigenic uveitis in a section entitled Inflammations, but not phacolytic glaucoma—it is discussed under Secondary Glaucoma with Material in the Trabecular Meshwork. (The term lens-particle glaucoma does not appear in the Path book's index.) In contrast, the Uveitis book eschews the term phacoantigenic uveitis/glaucoma entirely, using instead the term lens-induced uveitis. It goes on to describe phacolytic glaucoma in a manner consistent with the other books. (Lens-particle glaucoma does not appear in its index either.) Finally, the Glaucoma book groups all three conditions together under the heading Lens-Induced Glaucoma, and does not address the issue of innate vs adaptive immunity. Instead, it refers simply to 'inflammation' in the description of all three conditions. The term phacoantigenic uveitis does not appear. TLDR When studying the lens-related secondary OAGs, make sure to read about them in all four books that address them: Glaucoma, Uveitis, Lens and Path, and be prepared to encounter inconsistencies when doing so.
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Given that their cataracts can’t get much worse, what accounts for the fact that phacolytic glaucoma pts c/o an acute worsening of VA coinciding with the onset of their pain and ocular injection?
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**How is it the fellow eye can become involved in phacoantigenic glaucoma?**
Recall that it is normal for minute amounts of lens protein to be found in the AC of a phakic eye.
For each statement, identify the **lens-related secondary OAG** with which it is associated (some have more than one answer).

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The *absence* of KP is a key clinical finding:
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Next let’s take a look at some inflammatory causes of secondary OAG.
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In general terms, what is the mechanism of steroid-induced IOP elevation?
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--Clogging of Schlemm’s canal with inflammatory debris
--And lest we forget, the classic iatrogenic cause of elevated IOP in uveitis: 
Steroid-induced reduced TM outflow

In general terms, what is the mechanism of steroid-induced IOP elevation?
While still uncertain, it likely stems from impeded outflow at the TM owing to remodeling of the TM induced by the steroid
Is elevated IOP a common manifestation of uveitis?
No—in fact, a lower-than-normal IOP is expected

Why does the IOP tend to be lower in an inflamed eye?
Inflammation causes the ciliary body to ‘shut down’ to some extent, resulting in aqueous hyposecretion

When elevated IOP does occur in the setting of uveitis, a number of different mechanisms may be responsible. What are they?
--Inflammation of the TM causing it to swell
--Blocking of the angle by inflammatory material
--Clogging of Schlemm’s canal with inflammatory debris
--And lest we forget, the classic iatrogenic cause of elevated IOP in uveitis:

**Steroid-induced reduced TM outflow**

In general terms, what is the mechanism of steroid-induced IOP elevation?

**Steroid-induced IOP elevation will be addressed in greater detail later in the slide-set**
While there are many causes of OAG secondary to intraocular inflammation, the Glaucoma book sees fit to mention seven by name. What are they?
While there are many causes of OAG secondary to intraocular inflammation, the Glaucoma book sees fit to mention seven by name. What are they?
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

Posner-Schlossman syndrome?  Fuchs heterochromic iridocyclitis?

VZV uveitis?           HSV uveitis?

Toxo/plasmosis?         Pars planitis?

Juvenile idiopathic arthritis?

Of the seven, the Glaucoma book addresses two in detail—so in this slide-set, we will follow suit. Which ones?
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

Posner-Schlossman syndrome

- VZV uveitis
- Toxoplasmosis
- Juvenile idiopathic arthritis

Fuchs heterochromic iridocyclitis

- HSV uveitis
- Pars planitis

Of the seven, the Glaucoma book addresses two in detail—so in this slide-set, we will follow suit. Which ones?

(Note: All of the others are covered extensively in other slide-sets; see the Table of Contents)
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

Posner-Schlossman syndrome (aka)

Fuchs heterochromic iridocyclitis

By what noneponymous name is P-S syndrome also known?
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

- Posner-Schlossman syndrome
  (aka glaucomatocyclitic crisis)
- Fuchs heterochromic iridocyclitis

By what noneponymous name is P-S syndrome also known?
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

- Posner-Schlossman syndrome (aka glaucomatocyclitic crisis)
- Fuchs heterochromic iridocyclitis

Does each tend to present unilaterally, or bilaterally?
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

- Posner-Schlossman syndrome (aka glaucomatocyclitic crisis)
  - Unilateral

- Fuchs heterochromic iridocyclitis
  - Unilateral

Does each tend to present unilaterally, or bilaterally?
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

Posner-Schlossman syndrome (aka glaucomatocyclitic crisis)
  --Unilateral
  --?

Fuchs heterochromic iridocyclitis
  --Unilateral
  --?

What demographic is typically affected?
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

Posner-Schlossman syndrome (aka glaucomatocyclitic crisis)
--Unilateral
--Young to middle-aged adults

Fuchs heterochromic iridocyclitis
--Unilateral
--Young to middle-aged adults

What demographic is typically affected?
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

Posner-Schlossman syndrome (aka glaucomatocyclitic crisis)
--Unilateral
--Young to middle-aged adults
--Inflammation is...

Fuchs heterochromic iridocyclitis
--Unilateral
--Young to middle-aged adults
--Inflammation is...

Is inflammation typically mild, or severe?
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

Posner-Schlossman syndrome (aka glaucomatocyclitic crisis)
--Unilateral
--Young to middle-aged adults
--Inflammation is...mild

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Is inflammation typically mild, or severe?
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--Is a chronic condition

Is it an acute, chronic, or recurrent condition?
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What does it mean to say a uveitic condition is acute, recurrent or chronic?
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

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What does it mean to say a uveitic condition is acute, recurrent or chronic?
  --An acute uveitis…
  --A recurrent uveitis…
  --A chronic uveitis…
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Is it an acute, chronic, or recurrent condition?

What does it mean to say a uveitic condition is acute, recurrent or chronic?
--An acute uveitis…comes on suddenly, and resolves fairly quickly
--A recurrent uveitis…
--A chronic uveitis…
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

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Q/A

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Is it an acute, chronic, or recurrent condition?

What does it mean to say a uveitic condition is acute, recurrent or chronic?
--An acute uveitis...comes on suddenly, and resolves fairly quickly
--A recurrent uveitis...eventually relapses, but is quiescent off-treatment for at least a period of time
--A chronic uveitis...
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

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Is it an acute, chronic, or recurrent condition?

What does it mean to say a uveitic condition is acute, recurrent or chronic?
-- An acute uveitis...comes on suddenly, and resolves fairly quickly
-- A recurrent uveitis...eventually relapses, but is quiescent off-treatment for at least 3 months
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Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

Posner-Schlossman syndrome (aka glaucomatocyclitic crisis)
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- Young to middle-aged adults
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- IOP elevation usually...severe
- Is a **recurrent** condition

Fuchs heterochromic iridocyclitis
- Unilateral
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- IOP elevation usually...mild (or absent)
- Is a **chronic** condition

Is it an **acute**, **chronic**, or **recurrent** condition?

What does it mean to say a uveitic condition is acute, recurrent or chronic?
- An acute *uveitis*...comes on suddenly, and resolves fairly quickly
- A recurrent *uveitis*...eventually relapses, but is quiescent off-treatment for at least 3 months
- A chronic *uveitis*...
Q/A

Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

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Is it an acute, chronic, or recurrent condition?

What does it mean to say a uveitic condition is acute, recurrent or chronic?
-- An acute uveitis...comes on suddenly, and resolves fairly quickly
-- A recurrent uveitis...eventually relapses, but is quiescent off-treatment for at least 3 months
-- A chronic uveitis...also relapses, but its quiescent periods off-treatment last more than 3 months
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

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Take special note of the difference between recurrent and chronic uveitis, a commonly misunderstood distinction
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Does inflammation in each respond well, or poorly to steroids?
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

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*Does inflammation in each respond well, or poorly to steroids?*
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- KP are ‘white and round’

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- Poor response to steroids
- KP are ‘white and stellate’

*What is the classic descriptor of the shape of KP for each?*
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Are the KP distributed diffusely, or limited to Arlt’s triangle?
**Two forms of uveitic 2ndry OAG addressed in the Glaucoma book**

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*Are the KP distributed diffusely, or limited to Arlt’s triangle?*
They are diffusely distributed in both conditions
FHI: Stellate KP. Note the diffuse distribution
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Are the KP distributed diffusely, or limited to Arlt’s triangle?
They are diffusely distributed in both conditions.

Where is Arlt’s triangle located?

Arlt’s triangle
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Are the KP distributed diffusely, or limited to Arlt’s triangle? They are diffusely distributed in both conditions.

Where is Arlt’s triangle located? Its apex is at the corneal center, and base in the inferior cornea.
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Which is associated with heterochromia iridis?
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Is the affected eye the darker eye or the lighter eye?
Which is associated with heterochromia iridis?
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Is the affected eye the darker eye or the lighter eye?
The lighter (with one exception)

Which is associated with heterochromia iridis?
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--- Associated with heterochromia iridis

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

The lighter eye with one exception

heterochromia iridis

Which is associated with heterochromia iridis?
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What is the exception; ie, under what circumstances is the darker eye the one with FHI?
In individuals with light-blue eyes...heterochromia iridis

The lighter eye with one exception
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

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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

The lighter (with one exception)

heterochromia iridis
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

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*Which is associated with NVI and NVA?*
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

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- Associated with heterochromia iridis
- Associated with NVI and NVA

*Which is associated with NVI and NVA?*
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

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--Associated with heterochromia iridis
--Associated with NVI and NVA

Which is associated with NVI and NVA in FHI lead to PAS and NVG?
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

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-- Young to middle-aged adults
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-- IOP elevation usually...mild (or absent)
-- Is a chronic condition
-- Poor response to steroids
-- KP are ‘white and stellate’
-- Associated with heterochromia iridis
-- Associated with NVI and NVA, but PAS and/or NVG rarely develop

Which is associated with

Do NVI and NVA in FHI lead to PAS and NVG? Only rarely
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

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--Associated with NVI and NVA, but PAS and/or NVG rarely develop

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?
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

Posner-Schlossman syndrome (aka glaucomatocyclitic crisis)

--Unilateral
--Young to middle-aged adults
--Inflammation is…mild
--IOP elevation usually…severe
--Is a recurrent condition
--Good response to steroids
--KP are ‘white and round’

Fuchs heterochromic iridocyclitis

--Unilateral
--Young to middle-aged adults
--Inflammation is…mild
--IOP elevation usually…mild (or absent)
--Is a chronic condition
--Poor response to steroids
--KP are ‘white and stellate’
--Associated with heterochromia iridis
--Associated with NVI and NVA, but PAS and/or NVG rarely develop

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 (or Amsler-Verrey) sign
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

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(aka *glaucamatocyclitic crisis*)
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Which has a strong association with cataract, and with what type of cataract is it associated?
Two forms of uveitic 2ndry OAG addressed in the Glaucoma book

Posner-Schlossman syndrome (aka glaucomatocyclitic crisis)

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-- Associated with NVI and NVA, but PAS and/or NVG rarely develop
-- Associated with PSC

Which has a strong association with cataract, and with what type of cataract is it associated?
Open-angle Glaucoma: Secondary

FHI: Note the cataract
(This is a good point in the set to take a break)
Next let’s take a look at OAG secondary to increased EVP
What does EVP stand for in this context?
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Episcleral venous pressure, ie, the BP in the episcleral venous plexus
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What is the episcleral venous plexus?
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What is the episcleral venous plexus?

The episcleral venous plexus is the network of venous channels located in the episclera in the peri-limbal region
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What is the episcleral venous plexus?
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What is the episclera?
What does EVP stand for in this context?
Episceral venous pressure, ie, the BP in the episcleral venous plexus

What is the episcleral venous plexus?

The episcleral venous plexus is the network of venous channels located in the episclera in the peri-limbal region

What is the episclera?
The outermost of the three layers of the sclera
What does EVP stand for in this context?
Episcleral venous pressure, ie, the BP in the episcleral venous plexus

What is the episcleral venous plexus?

The episcleral venous plexus is the network of venous channels located in the episclera in the peri-limbal region

What is the episclera?
The outermost of the three layers of the sclera

Out to in, what are the other two layers?
--Episclera
--
What does EVP stand for in this context?
Episceral venous pressure, ie, the BP in the episcleral venous plexus

What is the episcleral venous plexus?
The episcleral venous plexus is the network of venous channels located in the peri-limbal region.

What is the episclera?
The outermost of the three layers of the sclera

Out to in, what are the other two layers?
--Episclera
--Stroma
--Lamina fusca
Open-angle Glaucoma: Secondary

Sclera: Anatomy

The * indicates the lamina fusca
What does EVP stand for in this context?
Episcleral venous pressure, ie, the BP in the episcleral venous plexus

What is the episcleral venous plexus? From where does it receive its intake?

The episcleral venous plexus is the network of venous channels located in the episclera in the peri-limbal region.
OAG 2ndry to ↑ EVP

What does EVP stand for in this context?
Episceral venous pressure, ie, the BP in the episcleral venous plexus

What is the episcleral venous plexus? From where does it receive its intake?

The episcleral venous plexus is the network of venous channels located in the episclera in the peri-limbal region. So-called *two words* carry aqueous to it that drained from Schlemm’s canal via collector channels.
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What is the episcleral venous plexus? From where does it receive its intake?

The episcleral venous plexus is the network of venous channels located in the episclera in the peri-limbal region. So-called aqueous veins carry aqueous to it that drained from Schlemm’s canal via collector channels.
Open-angle Glaucoma: Secondary

Conventional aqueous outflow pathway
Aqueous vein. Note that the first portion (black arrowhead) contains only aqueous, whereas upon emptying into a venule, laminar flow consisting of separate aqueous and blood columns can be seen (blue arrowhead)
What does EVP stand for in this context?
Episceral venous pressure, ie, the BP in the episcleral venous plexus

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There are two main pathways for blood leaving the globe. What are they?
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There are two main pathways for blood leaving the globe. What are they?
The central retinal vein (CRV), and the vortex veins
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Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

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The central retinal vein (CRV), and the vortex veins. How many vortex veins are there?
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There are two main pathways for blood leaving the globe. What are they?
The central retinal vein (CRV), and the vortex veins. How many vortex veins are there? 4-7 (can be more)
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There are two main pathways for blood leaving the globe. What are they?
The central retinal vein (CRV), and the vortex veins.

How many vortex veins are there?
4-7 (can be more)

Where are they located?
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Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

There are two main pathways for blood leaving the globe. What are they?
The central retinal vein (CRV), and the vortex veins.

How many vortex veins are there?
4-7 (can be more)

Where are they located?
The pierce the sclera roughly at the equator of the globe. There is at least one (often more) in each quadrant of the eye.
Open-angle Glaucoma: **Secondary**

![Diagram of the eye showing vortex veins](image)

**Vortex veins**
Vortex veins. Their ampullae are visible during DFE (the large circle is approximating the equator of the globe)
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There are two main pathways for blood leaving the globe. What are they?
The central retinal vein (CRV), and the vortex veins.

What ocular structures does each pathway drain?
--The CRV:
--The vortex veins:
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There are two main pathways for blood leaving the globe. What are they? The central retinal vein (CRV), and the vortex veins.

What ocular structures does each pathway drain?
--The CRV: The retina
--The vortex veins: Pretty much everything else
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There are two main pathways for blood leaving the globe. What are they?
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What ocular structures does each pathway drain? 
--The CRV: The retina
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What ocular structure comprises the lion’s share of ‘everything else’?
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What ocular structures does each pathway drain?
--The CRV: The retina
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What ocular structure comprises the lion’s share of ‘everything else’?
The uvea, ie, the three parts of the uvea
What does EVP stand for in this context? Episcleral venous pressure, ie, the BP in the episcleral venous plexus.

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What ocular structures does each pathway drain? The CRV: The retina
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What ocular structure comprises the lion’s share of ‘everything else’? The uvea, ie, the choroid, ciliary body and iris.
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What venous structure receives the majority of CRV and vortex veins outflow?
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The superior ophthalmic vein (SOV)
Open-angle Glaucoma: Secondary

Superior ophthalmic vein

Superior ophthalmic vein
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

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Into what structure does the SOV empty?
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What venous structure receives the majority of CRV and vortex veins outflow?
The superior ophthalmic vein (SOV)

Into what structure does the SOV empty?
The cavernous sinus.
Open-angle Glaucoma: Secondary

Superior ophthalmic vein

Cavernous sinus
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What is the normal range for EVP?
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What is the normal range for EVP?
5-9 mmHg—same as CVP
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Open-angle Glaucoma: Secondary

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What does CVP stand for in this context?
Central venous pressure, ie, the blood pressure in the
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What is the normal range for EVP?
5-9 mmHg—same as CVP

What does CVP stand for in this context?
Central venous pressure, ie, the blood pressure in the right atrium
What does EVP stand for in this context?
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What is the normal range for EVP?
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What happens if EVP increases significantly?
Open-angle Glaucoma: Secondary

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What is the normal range for EVP?
5-9 mmHg—same as CVP

What happens if EVP increases significantly?
If EVP is high, back-pressure will be transmitted to the AC via the aqueous veins, collector channels, Schlemm’s canal and TM, causing a proportional increase in IOP.
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What happens if EVP increases significantly?
If EVP is high, back-pressure will be transmitted to the AC via the aqueous veins, collector channels, Schlemm’s canal and TM, causing a proportional increase in IOP. And if IOP is elevated high enough for long enough, the pt will develop OAG 2ndry to the increased EVP.
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Open-angle Glaucoma: Secondary

OAG 2ndry to $\uparrow$ EVP

In addition to elevated IOP, what other sign/symptoms might result from elevated EVP? If EVP is high, the episcleral veins will be 'prominent'—dilated and/or tortuous.
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Open-angle Glaucoma: Secondary

In addition to elevated IOP, what other sign/symptoms might result from elevated EVP?
The pt may c/o and present with a chronic ‘red eye’
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What happens if EVP is high?

In addition to elevated IOP, what other sign/symptoms might result from elevated EVP? The pt may c/o and present with a chronic ‘red eye’

Two aspects of such a red-eye presentation—one symptom-related, one sign-related—will suggest it’s not a run-of-the-mill red eye. What are they?

--Symptom-wise…

--Sign-wise…
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In addition to elevated IOP, what other sign/symptoms might result from elevated EVP?
The pt may c/o and present with a chronic ‘red eye’

Two aspects of such a red-eye presentation—one symptom-related, one sign-related—will suggest it’s not a run-of-the-mill red eye. What are they?
--Symptom-wise…The presentation will lack c/o related to ocular surface dz—no FBS, itching, tearing, etc
--Sign-wise…
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5-9 mmHg—same as CVP

In addition to elevated IOP, what other sign/symptoms might result from elevated EVP?
The pt may c/o and present with a chronic ‘red eye’

Two aspects of such a red-eye presentation—one symptom-related, one sign-related—will suggest it’s not a run-of-the-mill red eye. What are they?
--Symptom-wise…The presentation will lack c/o related to ocular surface dz—no FBS, itching, tearing, etc
--Sign-wise…

If EVP is high.

Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP
What does EVP stand for in this context?
Episceral venous pressure, ie, the BP in the episcleral venous plexus

What is the episcleral venous plexus? From where does it receive its intake? To where does it subsequently discharge that intake?
The episcleral venous plexus is the network of venous channels located in the episclera in the peri-limbal region. So-called aqueous veins carry aqueous to it that drained from Schlemm’s canal via collector channels. The plexus eventually discharges into uveal vessels that subsequently empty into the vortex veins.

What is the normal range for EVP?
5-9 mmHg—same as CVP

What happens if EVP increases significantly?
If EVP is high, back-pressure will be transmitted to the AC via the aqueous veins, collector channels and Schlemm’s canal, resulting in a proportional increase in IOP. And if IOP is elevated high enough for long enough, the pt will develop OAG 2ndry to the increased EVP.

In addition to elevated IOP, what other sign/symptoms might result from elevated EVP?
The pt may c/o and present with a chronic ‘red eye’

Two aspects of such a red-eye presentation—one symptom-related, one sign-related—will suggest it’s not a run-of-the-mill red eye. What are they? 
--Symptom-wise…The presentation will lack c/o related to ocular surface dz—no FBS, itching, tearing, etc
--Sign-wise…The episcleral veins will be ‘prominent’—dilated and/or tortuous
Open-angle Glaucoma: Secondary

Prominent episcleral veins 2ndry to increased EVP
In cases for which a cause can be identified, what are the two basic mechanisms of increased EVP?
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

Venous obstruction

In cases for which a cause can be identified, what are the two basic mechanisms of increased EVP?
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

Venous obstruction

*Note for those of you thought ‘AV malformation’ was the correct answer here…*
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

Venous obstruction

Note for those of you thought ‘AV malformation’ was the correct answer here...

This is one of very few times where I’m substituting my judgment for that of a BCSC book. The Glaucoma book does indeed label this category “AV malformations,” but that term clearly does not apply to all of the conditions the Glaucoma book hangs under it (TBH, I don’t think it applies to any of them). This is why I decided to go with my own term—the anodyne-but-accurate Arterial and/or venous abnormalities.
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

- Idiopathic
- Arterial and/or venous abnormalities
  - Umbrella term for several conditions
  - Fairly specific condition, but can present several ways
  - Very specific condition
- Venous obstruction

*The Glaucoma book mentions three arterial/venous abnormalities as causes—what are they?*
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

- Idiopathic
- Arterial and/or venous abnormalities
  - A-V fistula *Umbrella term for several conditions*
  - Orbital varix *Fairly specific condition, but can present several ways*
  - Sturge-Weber syndrome *Very specific condition*

Venous obstruction

The Glaucoma book mentions three arterial/venous abnormalities as causes—what are they?
It mentions two causes of venous obstruction—what are they?
It mentions two causes of venous obstruction—what are they?
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic?

Arterial and/or venous abnormalities
  - A-V fistula?
  - Orbital varix?
  - Sturge-Weber syndrome?

Venous obstruction
  - Retrobulbar tumor?
  - Thyroid eye dz?

Finally: Of the conditions known to cause 2ndry OAG owing to increased EVP, which is most common?
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix
- Sturge-Weber syndrome

Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

Finally: Of the conditions known to cause 2ndry OAG owing to increased EVP, which is most common? Idiopathic
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

The A-V fistulas implicated in OAG 2ndry to increased EVP: Where are they located?
The A-V fistulas implicated in OAG 2ndry to increased EVP: Where are they located?
The cavernous sinus (CS)
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

A-V fistula

Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

Orbital varix

The A-V fistulas implicated in OAG 2ndry to increased EVP: Where are they located?
The cavernous sinus (CS)

An aspect of CS anatomy makes it uniquely vulnerable to the development of A-V fistulas. What is it?
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

A-V fistula

Orbital varix

Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

The A-V fistulas implicated in OAG 2ndry to increased EVP: Where are they located?
The cavernous sinus (CS)

An aspect of CS anatomy makes it uniquely vulnerable to the development of A-V fistulas. What is it?
It is the configuration--unique in the human body--of having an arterial structure (the internal carotid artery and its branches) wholly within the confines of a venous structure (ie, the CS itself)
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

A-V fistula

Orbital varix

The A-V fistulas implicated in OAG 2ndry to increased EVP: Where are they located?
The cavernous sinus (CS)

An aspect of CS anatomy makes it uniquely vulnerable to the development of A-V fistulas. What is it? It is the configuration--unique in the human body--of having an arterial structure (the internal carotid artery and its dural branches) wholly within the confines of a venous structure (ie, the CS itself).
Open-angle Glaucoma: Secondary

Relationship between the internal carotid artery and the CS
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

A-V fistula

Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

The A-V fistulas implicated in OAG 2ndry to increased EVP: Where are they located?
The cavernous sinus (CS)

An aspect of CS anatomy makes it uniquely vulnerable to the development of A-V fistulas. What is it?
It is the configuration—unique in the human body—of having an arterial structure (the internal carotid artery and its dural branches) wholly within the confines of a venous structure (ie, the CS itself)

What is the fundamental problem that results from a fistula within the CS?
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

- A-V fistula
- Orbital varix

Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

The A-V fistulas implicated in OAG 2ndry to increased EVP: Where are they located?

The cavernous sinus (CS)

An aspect of CS anatomy makes it uniquely vulnerable to the development of A-V fistulas. What is it?
It is the configuration--unique in the human body--of having an arterial structure (the internal carotid artery and its dural branches) wholly within the confines of a venous structure (ie, the CS itself)

What is the fundamental problem that results from a fistula within the CS?
It’s a pressure thing. A fistula allows high-pressure blood from the arterial tree to flow into the low-pressure, venous-sided CS. The subsequent increase in blood pressure within the CS impedes venous flow into the CS, leading to congestion of the eye and orbit.
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

- A-V fistula

Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

The A-V fistulas implicated in OAG 2ndry to increased EVP: Where are they located?

The cavernous sinus (CS)

An aspect of CS anatomy makes it uniquely vulnerable to the development of A-V fistulas. What is it?

It is the configuration—unique in the human body—of having an arterial structure (the internal carotid artery and its dural branches) wholly within the confines of a venous structure (ie, the CS itself)

What is the fundamental problem that results from a fistula within the CS?

It’s a pressure thing. A fistula allows high-pressure blood from the arterial tree to flow into the low-pressure, venous-sided CS. The subsequent increase in blood pressure within the CS impedes venous flow into the CS, leading to congestion of the eye and orbit. Further, if the pressure increase within the CS is significant enough, reversal of blood flow through the venous structures that drain into the CS will occur—that is, blood will circulate from the CS to the eye and orbit.
Open-angle Glaucoma: Secondary

Carotid-CS fistula
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

- A-V fistula

Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

Earlier in the slide-set, the main venous conduit from the eye and orbit to the CS was identified. What was it again?

Superior ophthalmic vein

In a pt with a CS fistula, what is the appearance of the superior ophthalmic vein on orbital imaging studies?

It is enlarged. This is an important sign to search for when reviewing imaging studies in cases of suspected CS fistulas!

The A-V fistula implicated in OAG 2ndry to increased EVP: Where are they located?

Cavernous sinus (CS)

An aspect of CS anatomy makes it uniquely vulnerable to the development of A-V fistulas. What is it?

It is the configuration—unique in the human body—of having an arterial structure (the internal carotid artery and its dural branches) wholly within the confines of a venous structure (i.e., the CS itself). What is the fundamental problem that results from a fistula within the CS?

It’s a pressure thing. A fistula allows high-pressure blood from the arterial tree to flow into the low-pressure, venous-sided CS. The subsequent increase in blood pressure within the CS impedes venous flow into the CS, leading to congestion of the eye and orbit. Further, if the pressure increase within the CS is significant enough, reversal of blood flow through the venous structures that drain into the CS will occur—that is, blood will circulate from the CS to the eye and orbit.
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

- A-V fistula

Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

The A-V fistulas implicated in OGA 2ndry to increased EVP: Where are they located?

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Earlier in the slide-set, the main venous conduit from the eye and orbit to the CS was identified. What was it again?

The superior ophthalmic vein (SOV)

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Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

- A-V fistula

Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

The A-V fistulas implicated in OGA 2ndry to increased EVP: Where are they located?

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Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

A-V fistula

Venous obstruction

- Retrobulbar tumor
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Open-angle Glaucoma: Secondary

Carotid-CS fistula: Enlarged SOV on MRA
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

A-V fistula

Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

The A-V fistulas implicated in OGA 2ndry to increased EVP: Where are they located?

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For more on A-V fistulas, see slide-set N19
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

- A-V fistula
- Orbital varix
- Sturge-Weber syndrome

Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

Orbital varices are known also by what other name?

Q
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
  - A-V fistula
  - Orbital varix
  - Sturge-Weber syndrome

Venous obstruction
  - Retrobulbar tumor
  - Thyroid eye dz

Orbital varices are known also by what other name?
Orbital venous malformations
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
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Venous obstruction
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Orbital varices are known also by what other name?
Orbital venous malformations

What is the classic presenting sign?
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix
- Sturge-Weber syndrome

Venous obstruction
- Retrobulbar tumor
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Orbital venous malformations

What is the classic presenting sign?
Proptosis that occurs or worsens when the pt’s head is action or when the pt action
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix
- Sturge-Weber syndrome

Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

Orbital varices are known also by what other name? Orbital venous malformations

What is the classic presenting sign? Proptosis that occurs or worsens when the pt’s head is dependent, or when the pt Valsalvas
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
  - A-V fistula
  - Orbital varix
  - Sturge-Weber syndrome

Venous obstruction
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Orbital varices are known also by what other name?
Orbital venous malformations

What is the classic presenting sign?
Proptosis that occurs or worsens when the pt's head is dependent, or when the pt Valsalva-ing

Do these pts always have proptosis at rest, ie, when not Valsalva-ing?

No; in fact, it is not uncommon for the affected eye to be enophthalmic at rest
Open-angle Glaucoma: Secondary

**OAG 2ndry to ↑ EVP**

- **Idiopathic**
- **Arterial and/or venous abnormalities**
  - A-V fistula
  - Orbital varix
  - Sturge-Weber syndrome
- **Venous obstruction**
  - Retrobulbar tumor
  - Thyroid eye dz

**Orbital varices are known also by what other name?**
Orbital venous malformations

**What is the classic presenting sign?**
Proptosis that occurs or worsens when the pt's head is dependent, or when the pt Valsalvas

**What is the best means if diagnosing an orbital varix?**
Perform contrast-enhanced spiral CT while the pt Valsalvas

**How should orbital varices be managed?**
Conservatively, if at all possible—complete excision is difficult, and significant intra-op bleeding is a concern

**Do these pts always have proptosis at rest, ie, when not Valsalva-ing?**
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Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

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Venous obstruction
- Retrobulbar tumor
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Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

- Venous obstruction
  - Retrobulbar tumor
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Open-angle Glaucoma: Secondary

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Orbital varix. A 70-year-old woman who presented due to an orbital mass incidentally discovered on an outside brain MR imaging. Axial (A and C) and coronal (B and D) CT images of the orbits without (A and B) and with (C and D) a Valsalva maneuver demonstrate inducible enlargement of a lobular structure in the right orbit (arrows), consistent with a varix. The patient subsequently reported right-eye fullness when bending over.
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

- A-V fistula
- Orbital varix
- Sturge-Weber syndrome

Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

Orbital varices are known also by what other name? Orbital venous malformations

What is the classic presenting sign? Proptosis that occurs or worsens when the pt Valsalvas

Why spiral CT?

What is the best means of diagnosing an orbital varix? Perform contrast-enhanced spiral CT while the pt Valsalvas
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
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Venous obstruction
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Orbital varices are known also by what other name?
Orbital venous malformations

What is the classic presenting sign?
Proptosis that occurs or worsens when the pt Valsalvas

What is the best means of diagnosing an orbital varix?
Perform contrast-enhanced spiral CT while the pt Valsalvas

Why spiral CT?
Because it's a relatively fast imaging modality
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

- A-V fistula
- Orbital varix
- Sturge-Weber syndrome

Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

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Why spiral CT?
Because it’s a relatively fast imaging modality

Why is speed important?

Why spiral CT?
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

- A-V fistula
- Orbital varix
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Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

Orbital varices are known also by what other name? Orbital venous malformations

What is the classic presenting sign? Proptosis that occurs or worsens when the pt Valsalvas

Why spiral CT? Because it's a relatively fast imaging modality

Why is speed important? Because the pt is holding her breath!

What is the best means of diagnosing an orbital varix? Perform contrast-enhanced spiral CT while the pt Valsalvas
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
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Venous obstruction
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Orbital varices are known also by what other name?
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How should orbital varices be managed?
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

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How should orbital varices be managed?
Conservatively, if at all possible—complete excision is difficult, and significant intra-op bleeding is a concern
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
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Orbital varices are known also by what other name? Orbital venous malformations

What is the classic presenting sign? Proptosis that occurs or worsens when the pt’s head is dependent, or when the pt. Valsalvas

Per the Orbit book, in what two circumstances should excision of an orbital varix be considered?

How should orbital varices be managed? Conservatively, if at all possible—complete excision is difficult, and significant intra-op bleeding is a concern
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

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Orbital varices are known also by what other name? Orbital venous malformations

What is the classic presenting sign? Proptosis that occurs or worsens when the pt’s head is dependent, or when the pt Valsalvas

Per the Orbit book, in what two circumstances should excision of an orbital varix be considered?

-- If the varix is causing severe pain
-- If optic-nerve compression is threatening vision

How should orbital varices be managed? Conservatively, if at all possible—complete excision is difficult, and significant intra-op bleeding is a concern
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

- A-V fistula
- Orbital varix
- Sturge-Weber syndrome

Venous obstruction

- Retrobulbar tumor
- Thyroid eye dz

In one word, what sort of condition is Sturge-Weber?
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix

Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

Sturge-Weber syndrome

In one word, what sort of condition is Sturge-Weber?
A phakomatosis
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix
- Sturge-Weber syndrome

Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

In one word, what sort of condition is Sturge-Weber? A phakomatosis

Briefly, what is a phakomatosis?
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
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Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

In one word, what sort of condition is Sturge-Weber?
A phakomatosis

Briefly, what is a phakomatosis?
A congenital condition involving hamartomatous lesions of multiple organ systems, usually including the CNS, eyes, and skin.
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
  - A-V fistula
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  - Retrobulbar tumor
  - Thyroid eye dz

In one word, what sort of condition is Sturge-Weber?
A phakomatosis

Briefly, what is a phakomatosis?
A congenital condition involving hamartomatous lesions of multiple organ systems, usually including the CNS, eyes and skin
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix
- Sturge-Weber syndrome

Venous obstruction
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Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

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As neuro-oculocutaneous syndromes
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix

Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

Sturge-Weber syndrome

In one word, what sort of condition is Sturge-Weber?
A phakomatosis

By what noneponymous name is Sturge-Weber known?
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
  - A-V fistula
  - Orbital varix
  - Sturge-Weber syndrome

Venous obstruction
  - Retrobulbar tumor
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In one word, what sort of condition is Sturge-Weber?
A phakomatosis

By what noneponymous name is Sturge-Weber known?
Encephalotrigeminal angiomatosis
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix

Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

Sturge-Weber syndrome

What facial lesion is a hallmark of Sturge-Weber syndrome?
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix

Sturge-Weber syndrome

Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

What facial lesion is a hallmark of Sturge-Weber syndrome?
The port wine stain, aka 'nevus flammeus'.
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
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Venous obstruction
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What facial lesion is a hallmark of Sturge-Weber syndrome? The port wine stain, aka ‘nevus flammeus’
Open-angle Glaucoma: Secondary

Sturge-Weber: Port-wine stain
What facial lesion is a hallmark of Sturge-Weber syndrome? The port wine stain, aka ‘nevus flammeus’

When does it present?
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
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Venous obstruction
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The port wine stain, aka ‘nevus flammeus’

When does it present?
At birth
Open-angle Glaucoma: Secondary

Sturge-Weber: Port-wine stain
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OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
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Venous obstruction
- Retrolbulbar tumor
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Sturge-Weber syndrome

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When does it present?
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Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

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Venous obstruction
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What facial lesion is a hallmark of Sturge-Weber syndrome? The port wine stain, aka 'nevus flammeus'.

When does it present? At birth

What is the typical pattern of distribution? It comports to the distribution of one or more divisions of CN5

A phakomatosis

By what noneponymous name is Sturge-Weber known? Encephalotrigeminal angiomatosis

All infants with Sturge-Weber syndrome have a port wine stain.

Do all infants with a port wine stain have Sturge-Weber syndrome? No
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

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All infants with Sturge-Weber syndrome have a port wine stain. Do all infants with a port wine stain have Sturge-Weber syndrome?
**Open-angle Glaucoma: Secondary**

**OAG 2ndry to ↑ EVP**

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix

Sturge-Weber syndrome

Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

---

**What facial lesion is a hallmark of Sturge-Weber syndrome?**

The port wine stain, aka ‘nevus flammeus’

**When does it present?**

At birth

**What is the typical pattern of distribution?**

It comports to the distribution of one or more divisions of CN5

---

**All infants with Sturge-Weber syndrome have a port wine stain.**

**Do all infants with a port wine stain have Sturge-Weber syndrome?**

No
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix

Venous obstruction
- Retrobulbar tumor
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Sturge-Weber syndrome

What facial lesion is a hallmark of Sturge-Weber syndrome? The port wine stain, aka 'nevus flammeus'

When does it present? At birth

What is the typical pattern of distribution? It comports to the distribution of one or more divisions of CN5

All infants with Sturge-Weber syndrome have a port wine stain.

Do all infants with a port wine stain have Sturge-Weber syndrome? No

What posterior-pole lesion is a hallmark of Sturge-Weber syndrome? The diffuse choroidal hemangioma

Diffuse choroidal hemangioma is present in about 50%

Can the choroidal hemangioma be present bilaterally? Yes, but it's uncommon

Does the choroidal hemangioma have malignant potential? No
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix
- Sturge-Weber syndrome

Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

What facial lesion is a hallmark of Sturge-Weber syndrome? The port wine stain, aka ‘nevus flammeus’.

When does it present? At birth

What is the typical pattern of distribution? It comports to the distribution of one or more divisions of CN5.

All infants with Sturge-Weber syndrome have a port wine stain. Do all infants with a port wine stain have Sturge-Weber syndrome? No

What posterior-pole lesion is a hallmark of Sturge-Weber syndrome? The diffuse choroidal hemangioma

Diffuse choroidal hemangioma is present in about 50% of cases. Can the choroidal hemangioma be present bilaterally? Yes, but it’s uncommon. Does the choroidal hemangioma have malignant potential? No.
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix
- Sturge-Weber syndrome

Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

What facial lesion is a hallmark of Sturge-Weber syndrome? The port wine stain, aka 'nevus flammeus'.

When does it present? At birth.

What is the typical pattern of distribution? It comports to the distribution of one or more divisions of CN5.

All infants with Sturge-Weber syndrome have a port wine stain. Do all infants with a port wine stain have Sturge-Weber syndrome? No.

What posterior-pole lesion is a hallmark of Sturge-Weber syndrome? The diffuse choroidal hemangioma.

By what food-based name is the DFE appearance of a diffuse choroidal hemangioma known? 'Tomato catsup fundus'.
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix
- Sturge-Weber syndrome

Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

What facial lesion is a hallmark of Sturge-Weber syndrome?
The port wine stain, aka ‘ nevus flammeus ’.

When does it present?
At birth.

What posterior-pole lesion is a hallmark of Sturge-Weber syndrome?
The diffuse choroidal hemangioma.

By what food-based name is the DFE appearance of a diffuse choroidal hemangioma known?
‘Tomato catsup fundus’.
Open-angle Glaucoma: Secondary

Sturge-Weber: Tomato catsup fundus OD
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix
- Sturge-Weber syndrome

Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

What facial lesion is a hallmark of Sturge-Weber syndrome? The port wine stain, aka 'nevus flammeus'.

When does it present? At birth.

What is the typical pattern of distribution? It comports to the distribution of one or more divisions of CN5.

All infants with Sturge-Weber syndrome have a port wine stain. Do all infants with a port wine stain have Sturge-Weber syndrome? No.

What posterior-pole lesion is a hallmark of Sturge-Weber syndrome? Diffuse choroidal hemangioma.

Diffuse choroidal hemangioma is present in what percent of SWS? About 50%.

Can the choroidal hemangioma be present bilaterally? Yes, but it's uncommon.

Does the choroidal hemangioma have malignant potential? No.
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix

Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

Sturge-Weber syndrome

What facial lesion is a hallmark of Sturge-Weber syndrome?
The port wine stain, aka ‘nevus flammeus’

When does it present?
At birth

What is the typical pattern of distribution?
It comports to the distribution of one or more divisions of CN5

Sturge-Weber syndrome, also known as Encephalotrigeminal angioma.

What posterior-pole lesion is a hallmark of Sturge-Weber syndrome?
The diffuse choroidal hemangioma

Diffuse choroidal hemangioma is present in what percent of SWS?
About 50%

All infants with Sturge-Weber syndrome have a port wine stain.
Do all infants with a port wine stain have Sturge-Weber syndrome?
No
Open-angle Glaucoma: Secondary

**OAG 2ndry to ↑ EVP**

- Idiopathic
- Arterial and/or venous abnormalities
  - A-V fistula
  - Orbital varix
  - Sturge-Weber syndrome
- Venous obstruction
  - Retrobulbar tumor
  - Thyroid eye dz

**What facial lesion is a hallmark of Sturge-Weber syndrome?**
The port wine stain, aka 'nevus flammeus'.

**When does it present?**
At birth.

**What is the typical pattern of distribution?**
It comports to the distribution of one or more divisions of CN 5.

**All infants with Sturge-Weber syndrome have a port wine stain.**

No

**What posterior-pole lesion is a hallmark of Sturge-Weber syndrome?**
The diffuse choroidal hemangioma.

**Diffuse choroidal hemangioma is present in what percent of SWS?**
About 50%.

**Can the choroidal hemangioma be present bilaterally?**
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix
- Sturge-Weber syndrome

Venous obstruction
- Retrobulbar tumor
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What facial lesion is a hallmark of Sturge-Weber syndrome?
The port wine stain, aka ‘nevus flammeus’

When does it present?
At birth

What is the typical pattern of distribution?
It comports to the distribution of one or more divisions of CN5

All infants with Sturge-Weber have a port wine stain.

Do all infants with a port wine stain have Sturge-Weber syndrome?
No

What posterior-pole lesion is a hallmark of Sturge-Weber syndrome?
The diffuse choroidal hemangioma

Diffuse choroidal hemangioma is present in what percent of SWS?
About 50%

Can the choroidal hemangioma be present bilaterally?
Yes, but it’s uncommon
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix

Sturge-Weber syndrome

Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

What facial lesion is a hallmark of Sturge-Weber syndrome? The port wine stain, aka 'nevus flammeus'.

When does it present? At birth.

What is the typical pattern of distribution? It comports to the distribution of one or more divisions of CNV.

All infants with Sturge-Weber syndrome have a port wine stain.

Do all infants with a port wine stain have Sturge-Weber syndrome? No.

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Does the choroidal hemangioma have malignant potential? No.
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Arterial and/or venous abnormalities
- A-V fistula
- Orbital varix
- Sturge-Weber syndrome

Venous obstruction
- Retrobulbar tumor
- Thyroid eye dz

What facial lesion is a hallmark of Sturge-Weber syndrome?
- The port wine stain, aka ‘nevus flammeus’

When does it present?
- At birth

What is the typical pattern of distribution?
- It comports to the distribution of one or more divisions of CN5

All infants with Sturge-Weber syndrome have a port wine stain.
- No

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Does the choroidal hemangioma have malignant potential?
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What is a phakomatosis?
- Sturge-Weber

By what noneponymous name is Sturge-Weber known?
- Encephalotrigeminal angiomatosis

The port wine stain, aka ‘nevus flammeus’

All infants with Sturge-Weber syndrome have a port wine stain.
- No
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

Venous obstruction abnormalities

There is another DFE finding that is a hallmark of Sturge-Weber syndrome.

What is it?

Sturge-Weber syndrome

What facial lesion is a hallmark of Sturge-Weber syndrome?

The port wine stain, aka 'nevus flammeus'

When does it present?

At birth

What is the typical pattern of distribution?

It comports to the distribution of one or more divisions of CN5

All infants with Sturge-Weber have a port wine stain.

Do all infants with a port wine stain have Sturge-Weber syndrome?

No

What posterior-pole lesion is a hallmark of Sturge-Weber syndrome?

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Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

Venous obstruction abnormalities

There is another DFE finding that is a hallmark of Sturge-Weber syndrome. What is it?
Glaucomatous cupping of the ONH, aka the reason we’re talking about it!

Sturge-Weber syndrome

What facial lesion is a hallmark of Sturge-Weber syndrome?
The port wine stain, aka ‘nevus flammeus’

When does it present?
At birth

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It comports to the distribution of one or more divisions of CN5

All infants with Sturge-Weber have a port wine stain.

Do all infants with a port wine stain have Sturge-Weber syndrome?
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Yes, but it’s uncommon

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No
Open-angle Glaucoma: Secondary

Sturge-Weber: Same pic, but this time take note the glaucomatous cupping on the affected side
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

Venous obstruction abnormalities

There is another DFE finding that is a hallmark of Sturge-Weber syndrome. What is it?

**Glaucmatous cupping** of the ONH, aka the reason we’re talking about it!

What facial lesion is a hallmark of Sturge-Weber syndrome?

The port wine stain, aka ‘nevus flammeus’

When does it present?

At birth

What is the typical pattern of distribution?

It comports to the distribution of one or more divisions of CN5

All infants with Sturge-Weber syndrome have a port wine stain.

Do all infants with a port wine stain have Sturge-Weber syndrome?

No

What posterior-pole lesion is a hallmark of Sturge-Weber syndrome?

The diffuse choroidal hemangioma

What percent of Sturge-Weber syndrome pts develop glaucoma?

Estimates run as high as 70%

Is there a relationship between the port-wine stain and risk of glaucoma?

Yes—if the port-wine stain involves the eyelid, the risk is increased

Does the choroidal hemangioma have malignant potential?

No
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

Venous obstruction abnormalities

There is another DFE finding that is a hallmark of Sturge-Weber syndrome. What is it?

Glaucmatous cupping of the ONH, aka the reason we're talking about it!

Sturge-Weber syndrome

What facial lesion is a hallmark of Sturge-Weber syndrome?
The port wine stain, aka 'nevus flammeus'

When does it present?
At birth

What is the typical pattern of distribution?
It comports to the distribution of one or more divisions of CN5

All infants with Sturge-Weber syndrome have a port wine stain.

Do all infants with a port wine stain have Sturge-Weber syndrome?
No

What posterior-pole lesion is a hallmark of Sturge-Weber syndrome?
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Glaucomatous cupping of the ONH, aka the reason we're talking about it!
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

Venous obstruction abnormalities

There is another DFE finding that is a hallmark of Sturge-Weber syndrome. What is it?

Glaucmatous cupping of the ONH, aka the reason we’re talking about it!

Sturge-Weber syndrome

What facial lesion is a hallmark of Sturge-Weber syndrome?

The port wine stain, aka ‘nevus flammeus’

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The diffuse choroidal hemangioma

What percent of Sturge-Weber syndrome pts develop glaucoma?

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Is there a relationship between the port-wine stain and risk of glaucoma?

All infants with Sturge-Weber syndrome do not have glaucoma. Do all infants with a port wine stain develop glaucoma? No

Does the choroidal hemangioma have malignant potential?

No
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

Venous obstruction abnormalities

There is another DFE finding that is a hallmark of Sturge-Weber syndrome. What is it?

Glaucmatous cupping of the ONH, aka the reason we’re talking about it!

Sturge-Weber syndrome

What facial lesion is a hallmark of Sturge-Weber syndrome? The port wine stain, aka 'nevus flammeus'

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Is there a relationship between the port-wine stain and risk of glaucoma? Yes—if the port-wine stain involves the eyelid, the risk is increased

All infants with Sturge-Weber syndrome have a port wine stain.

Do all infants with a port wine stain have Sturge-Weber syndrome? No

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Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

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There is another DFE finding that is a hallmark of Sturge-Weber syndrome. What is it? Glaucmatous cupping of the ONH, aka the reason we’re talking about it!

Sturge-Weber syndrome

What facial lesion is a hallmark of Sturge-Weber syndrome? The port wine stain, aka ‘nevus flammeus’

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What percent of Sturge-Weber syndrome pts develop glaucoma? Estimates run as high as 70

Is there a relationship between the port-wine stain and risk of glaucoma? Yes—if the port-wine stain involves the eyelid, the risk is increased

All infants with Sturge-Weber syndrome develop port wine stain

Do all infants with a port wine stain have Sturge-Weber syndrome? No

Does the choroidal hemangioma have malignant potential? No
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Idiopathic

Arterial and/or venous abnormalities

Venous obstruction abnormalities

There is another DFE finding that is a hallmark of Sturge-Weber syndrome. What is it?

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What facial lesion is a hallmark of Sturge-Weber syndrome?

The port wine stain, aka ‘nevus flammeus’

What posterior-pole lesion is a hallmark of Sturge-Weber syndrome?

The diffuse choroidal hemangioma

What percent of Sturge-Weber syndrome pts develop glaucoma?

Estimates run as high as 70%

Is there a relationship between the port-wine stain and risk of glaucoma?

Yes—if the port-wine stain involves the eyelid, the risk is increased

For more on Sturge-Weber syndrome, see slide-set P10
In a nutshell, what causes increased EVP in thyroid eye dz (TED)?
In a nutshell, what causes increased EVP in thyroid eye dz (TED)?
Recall that TED involves congestion of the orbit owing to both infiltrating inflammatory cells as well as increased secretion of ground substance (eg, glycosaminoglycans). This increase in orbital mass leads to proptosis (TED is the #1 cause of both uni- and bilateral proptosis in adults).
In a nutshell, what causes increased EVP in thyroid eye dz (TED)?
Recall that TED involves congestion of the orbit owing to both infiltrating inflammatory cells as well as increased secretion of ground substance (eg, glycosaminoglycans). This increase in orbital mass leads to proptosis (TED is the #1 cause of both uni- and bilateral proptosis in adults). Further, the orbital congestion often compresses the vortex veins, which in turn increases EVP.
In a nutshell, what causes increased EVP in thyroid eye dz (TED)?
Recall that TED involves congestion of the orbit owing to both infiltrating inflammatory cells as well as increased secretion of ground substance (eg, glycosaminoglycans). This increase in orbital mass leads to proptosis (TED is the #1 cause of both uni- and bilateral proptosis in adults). Further, the orbital congestion often compresses the vortex veins, which in turn increases EVP.
In a nutshell, what causes increased EVP in thyroid eye dz (TED)?

For more on TED, see slide-set O5

Recall that TED involves congestion of the orbit owing to both infiltrating inflammatory cells as well as increased secretion of ground substance (e.g., glycosaminoglycans). This increase in orbital mass leads to proptosis (TED is the #1 cause of both uni- and bilateral proptosis in adults). Further, the orbital congestion often compresses the vortex veins, which in turn increases EVP.
Is OAG 2ndry to increased EVP amenable to medical management?
Is OAG 2ndry to increased EVP amenable to medical management?
Yes, provided the clinician keep in mind that treatments which work by are unlikely to be effective
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Is OAG 2ndry to increased EVP amenable to medical management? Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Is OAG 2ndry to increased EVP amenable to medical management?
Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective

Why don’t treatments directed at increasing TM outflow work?
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Is OAG 2ndry to increased EVP amenable to medical management?
Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective.

Why don’t treatments directed at increasing TM outflow work?
Because of the pathophysiology of the condition—the mechanism by which IOP is elevated.
Is OAG 2ndry to increased EVP amenable to medical management?
Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective.

Why don’t treatments directed at increasing TM outflow work?
Because of the pathophysiology of the condition—the mechanism by which IOP is elevated. Remember: High EVP produces back-pressure that is transmitted to the AC via a path that directly involves the TM. Because of this, the TM is not amenable to therapeutic maneuvers intended to enhance its functionality.
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Is OAG 2ndry to increased EVP amenable to medical management?
Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective.

Why don’t treatments directed at increasing TM outflow work?
Because of the pathophysiology of the condition—the mechanism by which IOP is elevated. Remember: High EVP produces back-pressure that is transmitted to the AC via a path that directly involves the TM. Because of this, the TM is not amenable to therapeutic maneuvers intended to enhance its functionality.

Which two therapies are off the table?

-- Hints forthcoming…
Open-angle Glaucoma: Secondary

OAG Secondary to ↑ EVP

Is OAG Secondary to increased EVP amenable to medical management?
Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective.

Why don’t treatments directed at increasing TM outflow work?
Because of the pathophysiology of the condition—the mechanism by which IOP is elevated. Remember: High EVP produces back-pressure that is transmitted to the AC via a path that directly involves the TM. Because of this, the TM is not amenable to therapeutic maneuvers intended to enhance its functionality.

Which two therapies are off the table?
-- (this one is NBD, because you never use it anyway)
-- (this one you use a lot)
Is OAG 2ndry to increased EVP amenable to medical management?
Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective.

Why don’t treatments directed at increasing TM outflow work?
Because of the pathophysiology of the condition—the mechanism by which IOP is elevated. Remember: High EVP produces back-pressure that is transmitted to the AC via a path that directly involves the TM. Because of this, the TM is not amenable to therapeutic maneuvers intended to enhance its functionality.

Which two therapies are off the table?
--Topical tx with…
--Surgical tx with…
Is OAG 2ndry to increased EVP amenable to medical management?
Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective.

Why don’t treatments directed at increasing TM outflow work?
Because of the pathophysiology of the condition—the mechanism by which IOP is elevated. Remember: High EVP produces back-pressure that is transmitted to the AC via a path that directly involves the TM. Because of this, the TM is not amenable to therapeutic maneuvers intended to enhance its functionality.

Which two therapies are off the table?
--Topical tx with…pilo
--Surgical tx with…SLT
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Is OAG 2ndry to increased EVP amenable to medical management?
Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective.

Why don’t treatments directed at increasing TM outflow work?
Because of the pathophysiology of the condition—the mechanism by which IOP is elevated. Remember: High EVP produces back-pressure that is transmitted to the AC via a path that directly involves the TM. Because of this, the TM is not amenable to therapeutic maneuvers intended to enhance its functionality.

Which two therapies are off the table?
--Topical tx with…pilo
--Surgical tx with…MIGS?

If you answered MIGS…
Open-angle Glaucoma: Secondary

OAG 2ndry to ↑ EVP

Is OAG 2ndry to increased EVP amenable to medical management?
Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective.

Why don’t treatments directed at increasing TM outflow work?
Because of the pathophysiology of the condition—the mechanism by which IOP is elevated. Remember: High EVP produces back-pressure that is transmitted to the AC via a path that directly involves the TM. Because of this, the TM is not amenable to therapeutic maneuvers intended to enhance its functionality.

Which two therapies are off the table?
--Topical tx with…pilo
--Surgical tx with…MIGS?

If you answered MIGS…The Glaucoma book in my possession does not address these procedures specifically in the context of elevated EVP. But it would certainly stand to reason that procedures intended to facilitate TM outflow (as is the case for all FDA-approved MIGS at the time of this writing) would be ineffective as well.
Is OAG 2ndry to increased EVP amenable to medical management?
Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective.

Why don’t treatments directed at increasing TM outflow work?
Because of the pathophysiology of the condition—the mechanism by which IOP is elevated. Remember: High EVP produces back-pressure that is transmitted to the AC via a path that directly involves the TM. Because of this, the TM is not amenable to therapeutic maneuvers intended to enhance its functionality.

OK, so what treatments should be employed?
Is OAG 2ndry to increased EVP amenable to medical management? Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective.

Why don’t treatments directed at increasing TM outflow work? Because of the pathophysiology of the condition—the mechanism by which IOP is elevated. Remember: High EVP produces back-pressure that is transmitted to the AC via a path that directly involves the TM. Because of this, the TM is not amenable to therapeutic maneuvers intended to enhance its functionality.

OK, so what treatments should be employed? Drops that should be used, along with those that
Is OAG 2ndry to increased EVP amenable to medical management?
Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective.

Why don’t treatments directed at increasing TM outflow work?
Because of the pathophysiology of the condition—the mechanism by which IOP is elevated. Remember: High EVP produces back-pressure that is transmitted to the AC via a path that directly involves the TM. Because of this, the TM is not amenable to therapeutic maneuvers intended to enhance its functionality.

OK, so what treatments should be employed?
Drops that decrease aqueous production should be used, along with those that increase uveoscleral outflow.
Is OAG 2ndry to increased EVP amenable to medical management?
Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective.

Why don’t treatments directed at increasing TM outflow work?
Because of the pathophysiology of the condition—the mechanism by which IOP is elevated. Remember: High EVP produces back-pressure that is transmitted to the AC via a path that directly involves the TM. Because of this, the TM is not amenable to therapeutic maneuvers intended to enhance its functionality.

OK, so what treatments should be employed?
Drops that decrease aqueous production should be used, along with those that increase uveoscleral outflow.

If medical management fails, should filtering surgery be pursued?
Is OAG 2ndry to increased EVP amenable to medical management? Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective.

Why don’t treatments directed at increasing TM outflow work? Because of the pathophysiology of the condition—the mechanism by which IOP is elevated. Remember: High EVP produces back-pressure that is transmitted to the AC via a path that directly involves the TM. Because of this, the TM is not amenable to therapeutic maneuvers intended to enhance its functionality.

OK, so what treatments should be employed? Drops that decrease aqueous production should be used, along with those that increase uveoscleral outflow.

If medical management fails, should filtering surgery be pursued? While not strictly contraindicated, it is best not attempted by surgeons who are faint of heart, because the presence of elevated EVP significantly increases the risk of intra-op effusion of the uvea.
Is OAG 2ndry to increased EVP amenable to medical management?
Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective.

Why don’t treatments directed at increasing TM outflow work?
Because of the pathophysiology of the condition—the mechanism by which IOP is elevated. Remember: High EVP produces back-pressure that is transmitted to the AC via a path that directly involves the TM. Because of this, the TM is not amenable to therapeutic maneuvers intended to enhance its functionality.

OK, so what treatments should be employed?
Drops that decrease aqueous production should be used, along with those that increase uveoscleral outflow.

If medical management fails, should filtering surgery be pursued?
While not strictly contraindicated, it is best not attempted by surgeons who are faint of heart, because the presence of elevated EVP significantly increases the risk of intra-op effusion and/or hemorrhage of the uvea.
Is OAG 2ndry to increased EVP amenable to medical management?
Yes, provided the clinician keep in mind that treatments which work by increasing TM outflow are unlikely to be effective.

Why don’t treatments directed at increasing TM outflow work?
Because of the pathophysiology of the condition—the mechanism by which IOP is elevated. Remember: High EVP produces back-pressure that is transmitted to the AC via a path that directly involves the TM. Because of this, the TM is not amenable to therapeutic maneuvers intended to enhance its functionality.

OK, so what treatments should be employed?
Drops that decrease aqueous production should be used, along with those that increase uveoscleral outflow.

If medical management fails, should filtering surgery be pursued?
While not strictly contraindicated, it is best not attempted by surgeons who are faint of heart, because the presence of elevated EVP significantly increases the risk of intra-op effusion and/or hemorrhage of the uvea. Filtering surgeries on these eyes can get real sporty real fast.
(This is a good point in the set to take a break)
Open-angle Glaucoma: Secondary

↑ IOP OAG

Primary

Secondary

Drug-Induced

Trauma-Related

Schwartz syndrome

Next let’s take a look at certain trauma-related causes of secondary OAG, along with Schwartz syndrome
One of these things is not like the others. Which two belong together, which one stands alone, and why?
One of these things is not like the others. Which two belong together, which one stands alone, and why?

- Glaucoma 2ndry to hyphema
  - Follows AC bleed

- Hemolytic glaucoma
  - Follow vitreous bleed

- Ghost-cell glaucoma
Open-angle Glaucoma: Secondary

Glaucoma 2ndry to hyphema
Follows AC bleed

Hemolytic glaucoma
Ghost-cell glaucoma

Follow vitreous bleed

Hyphema is covered in its own slide-set (FELT12)
Glaucoma 2ndry to hyphema
Follows AC bleed

The remainder of this set will focus on hemolytic- and ghost-cell glaucoma
Open-angle Glaucoma: Secondary

Hemolytic glaucoma

Ghost-cell glaucoma

Follow vitreous bleed

THIS IS IMPORTANT! Take a moment to file a mental note before proceeding:
Hemolytic- and ghost-cell glaucoma follow a vitreous bleed, not an AC bleed!
Glaucoma 2ndry to hyphema

Follows AC bleed

Hemolytic glaucoma
Ghost-cell glaucoma

Follow vitreous bleed

What causes of vitreous hemorrhage are involved?
What causes of vitreous hemorrhage are involved?
The usual suspects—PDR, CRVO, etc, as well as trauma
What causes of vitreous hemorrhage are involved? The usual suspects—PDR, CRVO, etc, as well as trauma

How does the blood get from the vitreous cavity to the AC?
Open-angle Glaucoma: Secondary

- Hemolytic glaucoma
- Ghost-cell glaucoma

What causes of vitreous hemorrhage are involved?
The usual suspects—PDR, CRVO, etc, as well as trauma

How does the blood get from the vitreous cavity to the AC?
It can occur spontaneously, but more commonly there’s a hx of anterior hyaloid face disruption from trauma or surgery (eg, cataract; PPV) that provides a ready pathway for cells to reach the AC
Open-angle Glaucoma: Secondary

Glaucoma 2ndry to hyphema
Follows AC bleed

Hemolytic glaucoma

Ghost-cell glaucoma

In a nutshell, what is the mechanism underlying both hemolytic and ghost-cell glaucomas?
Glaucoma 2ndry to hyphema
Follows AC bleed

**Open-angle Glaucoma: Secondary**

- **Hemolytic glaucoma**
  - TM clogged
- **Ghost-cell glaucoma**
  - TM clogged

*In a nutshell, what is the mechanism underlying both hemolytic and ghost-cell glaucomas?*

TM clogging → impeded aqueous outflow → increased IOP
In each condition, what is clogging the TM?

--Hemolytic glaucoma: ?
--Ghost-cell glaucoma: ?

Glaucoma 2ndry to hyphema
Follows AC bleed

Open-angle Glaucoma: Secondary

Hemolytic glaucoma
TM clogged with…
?

Ghost-cell glaucoma
TM clogged with…
?
Hemolytic glaucoma

- TM clogged with...
- Hgb-laden macrophages

Ghost-cell glaucoma

- TM clogged with...
- Degenerated RBCs

In each condition, what is clogging the TM?
- Hemolytic glaucoma: Hgb-laden macrophages
- Ghost-cell glaucoma: Degenerated RBCs

Glaucoma 2ndry to hyphema

Follows AC bleed
Ghost-cell glaucoma

Follows AC bleed

Hemolytic glaucoma

TM clogged with...

Hgb-laden macrophages

degenerated RBCs

Ghost-cell glaucoma

Glaucoma 2ndry to hyphema

Make another mental note: While ghost-cell glaucoma involves RBCs as would be expected in a hemorrhage-related condition, in hemolytic glaucoma the culprit is not RBCs—it’s macrophages
Open-angle Glaucoma: Secondary

- Glaucoma 2ndry to hyphema
  - Follows AC bleed

- Hemolytic glaucoma
  - TM clogged with...
  - Hgb-laden macrophages

- Ghost-cell glaucoma
  - TM clogged with...
  - Degenerated RBCs

What’s up with the macrophages? How do they figure in all this?
What’s up with the macrophages? How do they figure in all this?

RBCs in a vitreous hemorrhage start to break down after a week or two. The degeneration of these cells attracts macrophages, which consume both the effete RBCs as well as the hemoglobin-related material they release. Heavy-laden with globules of degenerated Hgb and other RBC detritus, these macrophages end up in the AC, and ultimately the angle.
Hemolytic glaucoma. The anterior chamber angle contains macrophages with erythrocytic debris and rust-colored intracytoplasmic material (arrows).
What’s up with the macrophages? How do they figure in all this? RBCs in a vitreous hemorrhage start to break down after a week or two. The degeneration of these cells attracts macrophages, which consume both the effete RBCs as well as the hemoglobin-related material they release. Heavy laden with globules of degenerated Hgb and other RBC detritus, these macrophages end up in the AC, and ultimately the angle.

‘Globules of degenerated Hgb’ are known by what eponymous name? Heinz bodies

Globules of degenerated Hgb
What’s up with the macrophages? How do they figure in all this?
RBCs in a vitreous hemorrhage start to break down after a week or two. The degeneration of these cells attracts macrophages, which consume both the effete RBCs as well as the hemoglobin-related material they release. Heinz bodies are known by what eponymous name?

*Globules of degenerated Hgb* are known by what eponymous name? Heinz bodies
Hemolytic glaucoma. The degenerating hemoglobin is present as small globules known as Heinz bodies (arrows).
What’s up with the macrophages? How do they figure in all this? RBCs in a vitreous hemorrhage start to break down after a week or two. The degeneration of these cells attracts ‘Globules of degenerated Hgb’ are known by what eponymous name? ‘Heinz bodies’? Bruh, the BCSC Glaucoma book does not mention Heinz bodies. Why are you including details we don’t need to know?
Glaucoma 2ndry to hyphema

Follows AC bleed

Hemolytic glaucoma

TM clogged with...

Hgb-laden macrophages

Ghost-cell glaucoma

TM clogged with...

degenerated RBCs

What’s up with the macrophages? How do they figure in all this?

RBCs in a vitreous hemorrhage start to break down after a week or two. The degeneration of these cells attracts ‘Globules of degenerated Hgb’ are known by what eponymous name?

Heinz bodies

‘Heinz bodies’? Bruh, the BCSC Glaucoma book does not mention Heinz bodies. Why are you including details we don’t need to know? I wouldn’t do you like that bruh—the Pathology book mentions Heinz bodies in its discussion of hemolytic- and ghost-cell glaucoma, so it’s fair game for the OKAP
Open-angle Glaucoma: Secondary

- **Glaucoma 2ndry to hyphema**
  - Follows AC bleed

- **Hemolytic glaucoma**
  - TM clogged with...
    - **macrophages**

- **Ghost-cell glaucoma**
  - TM clogged with...
    - degenerated RBCs

**Finally:** ‘Macrophages clogging the TM’ should bring to mind another form of 2ndry OAG—what is it?
Open-angle Glaucoma: Secondary

Glaucoma 2ndry to hyphema
Follows AC bleed

Hemolytic glaucoma
TM clogged with...
*macrophages*

Ghost-cell glaucoma
TM clogged with...
degenerated RBCs

Finally: ‘Macrophages clogging the TM’ should bring to mind another form of 2ndry OAG—what is it?
Phacolytic glaucoma (addressed earlier in the slide-set)
Open-angle Glaucoma: Secondary

- Glaucoma 2ndry to hyphema
  - Follows AC bleed

- Hemolytic glaucoma
  - TM clogged with...
  - Hgb-laden macrophages

- Ghost-cell glaucoma
  - TM clogged with...
  - degenerated RBCs

*Degenerated RBCs pose a special problem for the TM--why?*
Open-angle Glaucoma: Secondary

- Glaucoma 2ndry to hyphema
  Follows AC bleed

- Hemolytic glaucoma
  TM clogged with...
  Hgb-laden macrophages

- Ghost-cell glaucoma
  TM clogged with...
  **degenerated RBCs**

*Degenerated RBCs pose a special problem for the TM--why?*
Healthy RBCs are very pliable, and gloop through the TM fairly easily. In contrast, degenerated RBCs become spherical and stiff, and do not pass easily through it; instead, they pile up in and clog the angle, preventing aqueous egress.
Open-angle Glaucoma: Secondary

Glaucoma 2ndry to hyphema
- Follows AC bleed

Hemolytic glaucoma
- TM clogged with...
- Hgb-laden macrophages

Ghost-cell glaucoma
- TM clogged with...
- degenerated RBCs

Degenerated RBCs pose a special problem for the TM—why?
Healthy RBCs are very pliable, and gloop through the TM fairly easily. In contrast, degenerated RBCs become spherical and stiff, and do not pass easily through it; instead, they pile up in and clog the angle, preventing aqueous egress.

‘RBCs that do not pass easily through the TM’—what other clinical scenario does that sound like?
Glaucoma 2ndry to hyphema
Follows AC bleed

Hemolytic glaucoma
TM clogged with...
Hgb-laden macrophages

Ghost-cell glaucoma
TM clogged with...
degenerated RBCs

Degenerated RBCs pose a special problem for the TM--why?
Healthy RBCs are very pliable, and gloop through the TM fairly easily. In contrast, degenerated RBCs become spherical and stiff, and do not pass easily through it; instead, they pile up in and clog the angle, preventing aqueous egress.

'RBCs that do not pass easily through the TM'—what other clinical scenario does that sound like?
Hyphema in a sickle-cell pt. Recall that the relatively basic v acidic nature of aqueous promotes RBC sickling. Sickled RBCs are significantly stiffer, and thus unable to pass easily through the TM.
Glaucoma 2ndry to hyphema

Hemolytic glaucoma

Ghost-cell glaucoma

Follows AC bleed

TM clogged with...

Hgb-laden macrophages

Degenerated RBCs

Degenerated RBCs pose a special problem for the TM--why? Healthy RBCs are very pliable, and gloop through the TM fairly easily. In contrast, degenerated RBCs become spherical and stiff, and do not pass easily through it; instead, they pile up in and clog the angle, preventing aqueous egress.

‘RBCs that do not pass easily through the TM’—what other clinical scenario does that sound like?

Hyphema in a sickle-cell pt. Recall that the relatively acidic nature of aqueous promotes RBC sickling. Sickled RBCs are significantly stiffer, and thus unable to pass easily through the TM.
Glaucoma 2ndry to hyphema
Follows AC bleed

Open-angle Glaucoma: Secondary

Hemolytic glaucoma

- TM clogged with...
- Hgb-laden macrophages

(Classic clinical description)

Ghost-cell glaucoma

- TM clogged with...
- Degenerated RBCs

(Classic clinical description)

What does examination of the AC reveal?
Glaucoma 2ndry to hyphema

Follows AC bleed

What does examination of the AC reveal?

Hemolytic glaucoma

TM clogged with...
Hgb-laden macrophages

Red-tinged cells

in AC

Ghost-cell glaucoma

TM clogged with...
degenerated RBCs

Tan-colored cells

in AC

Open-angle Glaucoma: Secondary
Open-angle Glaucoma: Secondary

Glaucoma 2ndry to hyphema
Follows AC bleed

Hemolytic glaucoma
- TM clogged with...
  - Hgb-laden macrophages
  - Red-tinged cells in AC

Ghost-cell glaucoma
- TM clogged with...
  - Degenerated RBCs
  - Tan-colored cells in AC

What does examination of the AC reveal?
Glaucoma 2ndry to hyphema
Follows AC bleed

Hemolytic glaucoma
TM clogged with...
Hgb-laden macrophages

Ghost-cell glaucoma
TM clogged with...
degenerated RBCs

Red-tinged cells in AC

Tan-colored cells in AC

Would these ‘red-tinged cells’ be Hgb-laden macrophages?
Open-angle Glaucoma: Secondary

Glaucoma 2ndry to hyphema
Follows AC bleed

Hemolytic glaucoma
TM clogged with...
Hgb-laden macrophages

Red-tinged cells in AC

Ghost-cell glaucoma
TM clogged with...
degenerated RBCs

Tan-colored cells in AC

Would these ‘red-tinged cells’ be Hgb-laden macrophages?
Indeed they would
Glaucoma 2ndry to hyphema

Follows AC bleed

Hemolytic glaucoma

TM clogged with...
Hgb-laden macrophages

Red-tinged cells in AC

Ghost-cell glaucoma

TM clogged with...
degenerated RBCs

Tan-colored cells in AC

Would these ‘tan-colored cells’ be the ghost cells after which the condition was named?
Glaucoma secondary to hyphema
Follows AC bleed

Hemolytic glaucoma
TM clogged with...
Hgb-laden macrophages

Ghost-cell glaucoma
TM clogged with...
degenerated RBCs

Red-tinged cells in AC

Would these ‘tan-colored cells’ be the ghost cells after which the condition was named?
Indeed they would
Ghost-cell glaucoma. Copious tan-colored cells in the AC.
Open-angle Glaucoma: Secondary

- **Glaucoma 2ndry to hyphema**
  - Follows AC bleed

- **Hemolytic glaucoma**
  - TM clogged with...
  - Hgb-laden macrophages

- **Ghost-cell glaucoma**
  - TM clogged with...
  - Degenerated RBCs

**Red-tinged cells in AC**

**Tan-colored cells in AC**

*Would these ‘tan-colored cells’ be the ghost cells after which the condition was named? Indeed they would*

*What’s the origin story of the ghost cells?*
A

Open-angle Glaucoma: Secondary

Glaucoma 2ndry to hyphema

Follows AC bleed

Hemolytic glaucoma

TM clogged with...
Hgb-laden macrophages

Red-tinged cells in AC

Ghost-cell glaucoma

TM clogged with...
Degenerated RBCs

Tan-colored cells in AC

Would these ‘tan-colored cells’ be the ghost cells after which the condition was named?
Indeed they would

What’s the origin story of the ghost cells?
They are RBCs from the vitreous bleed that have lost their hemoglobin
Ghost-cell glaucoma

Hemolytic glaucoma

Glaucoma secondary to hyphema

Follows AC bleed

Hemolytic glaucoma

TM clogged with...

Hgb-laden macrophages

Red-tinged cells in AC

Ghost-cell glaucoma

TM clogged with...

degenerated RBCs

Tan-colored cells in AC

Would these ‘tan-colored cells’ be the ghost cells after which the condition was named? Indeed they would.

How long does it take for RBCs to turn into ghost cells?

What’s the origin story of the ghost cells? They are RBCs from the vitreous bleed that have lost their hemoglobin.
Glaucoma 2ndry to hyphema

Follows AC bleed

Hemolytic glaucoma

TM clogged with...

Hgb-laden macrophages

Red-tinged cells in AC

Ghost-cell glaucoma

TM clogged with...

degenerated RBCs

Tan-colored cells in AC

Would these ‘tan-colored cells’ be the ghost cells after which the condition was named? Indeed they would.

How long does it take for RBCs to turn into ghost cells? 1-3 months

What’s the origin story of the ghost cells? They are RBCs from the vitreous bleed that have lost their hemoglobin.
Open-angle Glaucoma: Secondary

Glaucoma 2ndry to hyphema

Follows AC bleed

Hemolytic glaucoma
- TM clogged with...
  - Hgb-laden macrophages
- Red-tinged cells in AC

Ghost-cell glaucoma
- TM clogged with...
  - degenerated RBCs
- Tan-colored cells in AC

What does examination of the vitreous cavity reveal?

(Classic clinical description)
Hemolytic glaucoma:
- TM clogged with Hgb-laden macrophages
- Red-tinged cells in AC

Ghost-cell glaucoma:
- TM clogged with degenerated RBCs
- Tan-colored cells in AC

What does examination of the vitreous cavity reveal?
- Khaki-colored hemorrhage in the vitreous
Glaucoma 2ndry to hyphema
Follows AC bleed

Hemolytic glaucoma
- TM clogged with...
  - Hgb-laden macrophages
- **Red-tinged** cells in AC

Ghost-cell glaucoma
- TM clogged with...
  - degenerated RBCs
- **Tan-colored** cells in AC

Khaki-colored hemorrhage in the vitreous

What does examination of the vitreous cavity reveal?
Open-angle Glaucoma: Secondary

Glaucoma 2ndry to hyphema

Follows AC bleed

Hemolytic glaucoma

Ghost-cell glaucoma

Do hemolytic- and ghost-cell glaucoma resolve spontaneously?

Khaki-colored hemorrhage in the vitreous
Glaucoma 2ndry to hyphema

Follows AC bleed

Hemolytic glaucoma

Ghost-cell glaucoma

Do hemolytic- and ghost-cell glaucoma resolve spontaneously?
Usually, once the instigating vitreous hemorrhage has cleared

Khaki-colored hemorrhage in the vitreous
Open-angle Glaucoma: Secondary

Hemolytic glaucoma

Ghost-cell glaucoma

Do hemolytic- and ghost-cell glaucoma resolve spontaneously?

Usually, once the instigating vitreous hemorrhage has cleared

In the interim, how should the IOP be managed?

Khaki-colored hemorrhage in the vitreous
Open-angle Glaucoma: Secondary

Glaucoma 2ndry to hyphema

Follows AC bleed

Hemolytic glaucoma

- Do hemolytic- and ghost-cell glaucoma resolve spontaneously?
  - Usually, once the instigating vitreous hemorrhage has cleared

- In the interim, how should the IOP be managed?
  - With aqueous suppressants if possible

Ghost-cell glaucoma

Khaki-colored hemorrhage in the vitreous
Hemolytic glaucoma

Do hemolytic- and ghost-cell glaucoma resolve spontaneously?
Usually, once the instigating vitreous hemorrhage has cleared

In the interim, how should the IOP be managed?
With aqueous suppressants if possible

If medical management fails, what is the next step?

Ghost-cell glaucoma
Do hemolytic- and ghost-cell glaucoma resolve spontaneously? Usually, once the instigating vitreous hemorrhage has cleared.

In the interim, how should the IOP be managed? With aqueous suppressants if possible.

If medical management fails, what is the next step? AC washout.

Khaki-colored hemorrhage in the vitreous.
Open-angle Glaucoma: Secondary

Glaucoma 2ndry to hyphema

Follows AC bleed

Hemolytic glaucoma

Do hemolytic- and ghost-cell glaucoma resolve spontaneously?
 Usually, once the instigating vitreous hemorrhage has cleared

In the interim, how should the IOP be managed?
 With aqueous suppressants if possible

If medical management fails, what is the next step?
 AC washout

And if the AC washout fails?

Ghost-cell glaucoma

Khaki-colored hemorrhage in the vitreous
Glaucoma 2ndry to hyphema
Follows AC bleed

Open-angle Glaucoma: Secondary

Hemolytic glaucoma
Ghost-cell glaucoma

*Do hemolytic- and ghost-cell glaucoma resolve spontaneously?*
Usually, once the instigating vitreous hemorrhage has cleared

*In the interim, how should the IOP be managed?*
With aqueous suppressants if possible

*If medical management fails, what is the next step?*
AC washout

*And if the AC washout fails?*
PPV (if the hemorrhage is persistent) vs filtering surgery should be considered

Khaki-colored hemorrhage in the vitreous
Changing gears slightly…There is another form of secondary OAG called Schwartz syndrome that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but not a bleed. What event does it follow?
Changing gears slightly…There is another form of secondary OAG called Schwartz syndrome that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but not a bleed. What event does it follow? Rhegmatogenous retinal detachment (RRD)
Schwartz syndrome
Follows…**RRD**

**Open-angle Glaucoma: Secondary**

Hemolytic glaucoma
Ghost-cell glaucoma

Follow vitreous bleed

Changing gears slightly…There is another form of secondary OAG called **Schwartz syndrome** that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but not a bleed. What event does it follow?

**Rhegmatogenous retinal detachment (RRD)**

_Huh? I thought RRD was associated with reduced IOP. What gives?_
Changing gears slightly... There is another form of secondary OAG called Schwartz syndrome that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but not a bleed. What event does it follow?

Rhegmatogenous retinal detachment (RRD)

Huh? I thought RRD was associated with reduced IOP. What gives?

Acute RRD is indeed associated with reduced IOP. Schwartz syndrome is associated with chronic RRD.
What is the mechanism of reduced IOP in acute RRD?

Acute RRD is indeed associated with reduced IOP. Huh? I thought RRD was associated with reduced IOP. What gives?

Acute RRD is indeed associated with reduced IOP. Schwartz syndrome is associated with chronic RRD.
Changing gears slightly…There is another form of secondary OAG called Schwartz syndrome, which follows a posterior-segment event—but not a bleed. What event does it follow? Rhegmatogenous retinal detachment (RRD).

Why is it called Schwartz syndrome? Acute RRD is indeed associated with reduced IOP. Why? Recall that one function of the RPE is to deturgesc the subretinal space by actively pumping fluid out of it. RRD allows intraocular fluid to pass into the subretinal space, where the RPE attempts to remove it. If a significant portion of this fluid is removed, it will be reflected in a reduced IOP.

Huh? I thought RRD was associated with reduced IOP. What gives? Acute RRD is indeed associated with reduced IOP. Schwartz syndrome is associated with chronic RRD.
Schwartz syndrome  
Follows…**RRD**

Hemolytic glaucoma

Ghost-cell glaucoma

Follow vitreous bleed

**What is the mechanism of reduced IOP in acute RRD?**

Recall that one function of the RPE is to deturgesc the subretinal space by actively pumping fluid out of it. RRD allows intraocular fluid to pass into the subretinal space, where the RPE attempts to remove it. If a significant enough portion of this fluid is removed, IOP will go down.

**Acute RRD is indeed associated with reduced IOP**

*Huh? I thought RRD was associated with reduced IOP. What gives?*

Secondary OAG called Schwartz syndrome, follows a posterior-segment event—but not a bleed. What event does it follow?

Rhegmatogenous retinal detachment (RRD)

Acute RRD is indeed associated with reduced IOP.

Huh? I thought RRD was associated with reduced IOP. What gives?

Secondary OAG called Schwartz syndrome is associated with chronic RRD.
Changing gears slightly... There is another form of secondary OAG called Schwartz syndrome that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but not a bleed. What event does it follow?

Rhegmatogenous retinal detachment (RRD)

In both hemolytic and ghost-cell glaucomas, increased IOP results from clogging of the TM that impedes aqueous egress.
Changing gears slightly, there is another form of secondary open-angle glaucoma—Schwartz syndrome— that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but not a bleed. What event does it follow?

Rhegmatogenous retinal detachment (RRD)

In both hemolytic and ghost-cell glaucomas, increased IOP results from clogging of the TM that impedes aqueous egress.

What is the mechanism of IOP elevation in Schwartz syndrome?
Changing gears slightly, there is another form of secondary OAG called Schwartz syndrome. This syndrome is characterized by increased IOP, much like hemolytic- and ghost-cell glaucoma. However, it does not follow a bleed. What event does it follow?

- Rhegmatogenous retinal detachment (RRD)

What is the mechanism of IOP elevation in Schwartz syndrome? The same thing—TM clogging.

In both hemolytic and ghost-cell glaucomas, increased IOP results from clogging of the TM that impedes aqueous egress.
Changing gears slightly… There is another form of secondary OAG called Schwartz syndrome that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but not a bleed. What event does it follow?

- Rhegmatogenous retinal detachment (RRD)
- Ghost-cell glaucoma
- Hemolytic glaucoma
- Schwartz syndrome

In hemolytic glaucoma, the TM is clogged with macrophages; in ghost-cell glaucoma, it’s degenerated RBCs.

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- Ghost-cell glaucoma
- Hemolytic glaucoma

In hemolytic glaucoma, the TM is clogged with macrophages; in ghost-cell glaucoma, it’s degenerated RBCs.

What is clogging the TM in Schwartz syndrome?

Photoreceptor outer segments

TM clogged with…

Hgb-laden macrophages

degenerated RBCs
Changing gears slightly… There is another form of secondary OAG called Schwartz syndrome that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but not a bleed. What event does it follow?

- Rhegmatogenous retinal detachment (RRD)
- Ghost-cell glaucoma
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What is clogging the TM in Schwartz syndrome? Photoreceptor outer segments.

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Changing gears slightly... There is another form of secondary OAG called Schwartz syndrome that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but not a bleed. What event does it follow?

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- Ghost-cell glaucoma
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What is clogging the TM in Schwartz syndrome?

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In hemolytic glaucoma, the TM is clogged with macrophages; in ghost-cell glaucoma, it’s degenerated RBCs.

To sum it up: In Schwartz syndrome, chronic RRD allows enough time for liberated PR outer segs to migrate into the AC, where their accumulation in the angle ends up clogging the TM and elevating IOP.
Changing gears slightly… There is another form of secondary OAG called Schwartz syndrome that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but not a bleed. What event does it follow?

- Rhegmatogenous retinal detachment (RRD)
- Ghost-cell glaucoma
- Hemolytic glaucoma
- Schwartz syndrome

TM clogged with...
- Photoreceptor outer segments
- Hgb-laden macrophages
- Degenerated RBCs

What is clogging the TM in Schwartz syndrome?

Photoreceptor outer segments

In hemolytic glaucoma, the TM is clogged with macrophages; in ghost-cell glaucoma, it’s degenerated RBCs.

To sum it up: In Schwartz syndrome, chronic RRD allows enough time for liberated PR outer segs to migrate into the AC, where their accumulation in the angle ends up clogging the TM and elevating IOP.

All these PR outer segs floating around the AC—can they be mistaken for inflammatory cells?
Changing gears slightly… There is another form of secondary OAG called Schwartz syndrome that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but not a bleed. What event does it follow?

- Rhegmatogenous retinal detachment (RRD)
- Ghost-cell glaucoma
- Hemolytic glaucoma
- Schwartz syndrome

TM clogged with...
- Hgb-laden macrophages
- Degenerated RBCs
- PR outer segments

What is clogging the TM in Schwartz syndrome? Photoreceptor outer segments

In hemolytic glaucoma, the TM is clogged with macrophages; in ghost-cell glaucoma, it’s degenerated RBCs.

To sum it up: In Schwartz syndrome, chronic RRD allows enough time for liberated PR outer segs to migrate into the AC, where their accumulation in the angle ends up clogging the TM and elevating IOP.

All these PR outer segs floating around the AC—can they be mistaken for inflammatory cells?
Yes, uveitic glaucoma is a common misdiagnosis in Schwartz syndrome.
Changing gears slightly…There is another form of secondary OAG called **Schwartz syndrome** that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but not a bleed. What event does it follow? Rhegmatogenous retinal detachment (RRD)

What’s the best way to manage Schwartz syndrome?
Changing gears slightly…There is another form of secondary OAG called **Schwartz syndrome** that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but not a bleed. What event does it follow? Rhegmatogenous retinal detachment (RRD)

**What’s the best way to manage Schwartz syndrome?**
Repair the RRD
Next let’s take a look at other trauma-related causes of secondary OAG.
Three types of post-trauma angle changes:

1) one type of angle change

2) another type of angle change

3) a third type of angle change
Three types of post-trauma angle changes:

1) *Angle recession*

2) *Cyclodialysis cleft*

3) *Iridodialysis*
Three types of post-trauma angle changes:

1) **Angle recession**
   - Tear between longitudinal and circular CB fibers

2) **Cyclodialysis cleft**

3) **Iridodialysis**

(CB = ciliary body)
Three types of post-trauma angle changes:

1) **Angle recession**
   - Tear between **longitudinal and circular** CB fibers

2) **Cyclodialysis cleft**

3) **Iridodialysis**
Three types of post-trauma angle changes:

1) **Angle recession**
   - Tear between **longitudinal and circular** CB fibers
   - Classic description on gonio: **adjective** CBB
     
2) **Cyclodialysis cleft**

3) **Iridodialysis**
Three types of post-trauma angle changes:

1) **Angle recession**
   - Tear between *longitudinal and circular* CB fibers
   - Classic description on gonio: *Wide* CBB

2) **Cyclodialysis cleft**

3) **Iridodialysis**
Angle recession. Note the portion of normal angle with narrow CBB (black arrows), the point at which the recession starts (arrowhead), and the subsequent segment of recessed angle with widened CBB (red arrows).
Three types of post-trauma angle changes:

1) **Angle recession**
   - Tear between **longitudinal and circular** CB fibers
   - Classic description on gonio: **Wide** CBB

2) **Cyclodialysis cleft**
   - Structure separates from structure

3) **Iridodialysis**

Open-angle Glaucoma: *Secondary*
Three types of post-trauma angle changes:

1) *Angle recession*
   - Tear between *longitudinal and circular* CB fibers
   - Classic description on gonio: Wide CBB

2) *Cyclodialysis cleft*
   - CB separates from SS (SS = scleral spur)

3) *Iridodialysis*
Three types of post-trauma angle changes:

1) *Angle recession*
   - Tear between *longitudinal and circular* CB fibers
   - Classic description on gonio: *Wide* CBB

2) *Cyclodialysis cleft*
   - CB separates from SS
   - Classic description on gonio: *adjective* SS

3) *Iridodialysis*
Three types of post-trauma angle changes:

1) **Angle recession**
   - Tear between longitudinal and circular CB fibers
   - Classic description on gonio: **Wide** CBB

2) **Cyclodialysis cleft**
   - CB separates from SS
   - Classic description on gonio: **Glistening** SS

3) **Iridodialysis**

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Open-angle Glaucoma: **Secondary**
Cyclodialysis cleft. Racquetball vs eye. The broad white band is the cleft. Note the presence of angle recession on either side of the cleft.
Three types of post-trauma angle changes:

1) **Angle recession**
   - Tear between **longitudinal and circular** CB fibers
   - Classic description on gonio: **Wide** CBB

2) **Cyclodialysis cleft**
   - CB separates from SS
   - Classic description on gonio: **Glistening** SS

3) **Iridodialysis**
   - Tear at structure
Three types of post-trauma angle changes:

1) **Angle recession**
   - Tear between longitudinal and circular CB fibers
   - Classic description on gonio: **Wide** CBB

2) **Cyclodialysis cleft**
   - CB separates from SS
   - Classic description on gonio: **Glistening** SS

3) **Iridodialysis**
   - Tear at **iris root**

Open-angle Glaucoma: **Secondary**
Open-angle Glaucoma: Secondary

Iridodialysis
1) Angle recession glaucoma?

2) Cyclodialysis cleft glaucoma?

3) Iridodialysis glaucoma?

All three can be associated with the subsequent development of glaucoma, but for which of them is the association especially strong?
Three types of post-trauma angle changes:

1) Angle recession glaucoma

All three can be associated with the subsequent development of glaucoma, but for which of them is the association especially strong?

Angle recession
Angle recession glaucoma

What is the temporal relationship between the inciting trauma and the subsequent development of angle-recession glaucoma?
**Angle recession glaucoma**

*What is the temporal relationship between the inciting trauma and the subsequent development of angle-recession glaucoma?*

It can be immediate, or delayed by months to many years.
Angle recession glaucoma

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What is the classic presentation?
Three types of post-trauma angle changes:

1) Angle recession glaucoma

What is the temporal relationship between the inciting trauma and the subsequent development of angle-recession glaucoma?
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What is the classic presentation?
A pt with what seems to be unilateral POAG

**Angle recession glaucoma**

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A pt with what seems to be unilateral POAG
**Angle recession glaucoma**

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Is there a correlation between the extent of angle recession and the risk of developing glaucoma?
Angle recession glaucoma

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What is the classic presentation?
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Is there a correlation between the extent of angle recession and the risk of developing glaucoma?
Yes

Is there a correlation between angle-recession glaucoma in one eye and the development of elevated IOP in the fellow eye?
Angle recession glaucoma

What is the temporal relationship between the inciting trauma and the subsequent development of angle-recession glaucoma?
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A pt with what seems to be unilateral POAG

Is there a correlation between the extent of angle recession and the risk of developing glaucoma?
Yes

Is there a correlation between angle-recession glaucoma in one eye and the development of elevated IOP in the fellow eye?
Yes—it will occur in as many as [50%] of fellow eyes
Angle recession glaucoma

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What is the classic presentation?
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Is there a correlation between angle-recession glaucoma in one eye and the development of elevated IOP in the fellow eye?
Yes—it will occur in as many as half of fellow eyes
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What does this fact suggest about eyes with angle-recession glaucoma?
Angle recession glaucoma

*What is the temporal relationship between the inciting trauma and the subsequent development of angle-recession glaucoma?*
It can be immediate, or delayed by months to many years

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Yes

*Is there a correlation between angle-recession glaucoma in one eye and the development of elevated IOP in the fellow eye?*
Yes—*it will occur in as many as half of fellow eyes*

*What does this fact suggest about eyes with angle-recession glaucoma?*
It suggests they live in the head of a person who was predisposed to develop glaucoma in the first place
Angle recession glaucoma

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How should angle-recession glaucoma be managed?
Angle recession glaucoma

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How should angle-recession glaucoma be managed?
With the standard complement of topical hypotensives
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How should angle-recession glaucoma be managed?
With the standard complement of topical hypotensives, or SLT?
Three types of post-trauma angle changes:

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How should angle-recession glaucoma be managed?
With the standard complement of topical hypotensives, or SLT? Nah
Next let’s look at drug-induced secondary OAG.
Open-angle Glaucoma: Secondary

Which commonly-used compound is notorious for its propensity to elevate IOP?
Which commonly-used compound is notorious for its propensity to elevate IOP?
Corticosteroids
Open-angle Glaucoma: Secondary

Which commonly-used compound is notorious for its propensity to elevate IOP? Corticosteroids

Which of the common routes of ocular administration have been implicated in IOP elevation?
--Topical?
--Intravitreal injection?
--Intravitreal implant?
--Sub-Tenon’s/periocular depot?
--Periocular injection?
Open-angle Glaucoma: Secondary

Which commonly-used compound is notorious for its propensity to elevate IOP?
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Which of the common routes of ocular administration have been implicated in IOP elevation?
--Topical!
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--Sub-Tenon’s/periocular depot!
--Periocular injection!

All can cause elevated IOP!
Open-angle Glaucoma: Secondary

Which commonly-used compound is notorious for its propensity to elevate IOP?
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What proportion of IVit steroid injection pts will develop at least a transient IOP spike?

About 50%

Of the 50% of IVit steroid injection pts who experience an IOP spike, what proportion will require topical therapy to control their IOP?

About 25%

Of the 50% of IVit steroid injection pts who experience an IOP spike, what proportion will require surgical (incisional) intervention?

About 2%
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--Topical!
--Intravitreal injection!
--Intravitreal implant!
--Sub-Tenon's/periocular
--Periocular injection!

How do intravitreal implants compare to injections in terms of producing IOP elevation?

Open-angle Glaucoma: Secondary

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Which of the common routes of ocular administration have been implicated in IOP elevation?
--Topical!
--Intravitreal injection!
--**Intravitreal implant**!
--Sub-Tenon's/periocular
--Periocular injection!

How do intravitreal implants compare to injections in terms of producing IOP elevation? They're like intravitreal injections on steroids (if you'll pardon the expression). That is, everything is worse—a greater proportion of implant pts develop IOP spikes, a greater proportion need tx, and a greater proportion require incisional surgery.
Open-angle Glaucoma: Secondary

Which commonly-used compound is notorious for its propensity to elevate IOP? Corticosteroids

Which of the common routes of ocular administration have been implicated in IOP elevation? How about common routes of non-ocular administration?

--Topical!
--Intravitreal injection!
--Intravitreal implant!
--Sub-Tenon’s/periocular depot!
--Periocular injection!
--Systemic?
--Topical?
--Inhaled?
--Nasal?
Open-angle Glaucoma: Secondary

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Again, all can cause elevated IOP!
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--Nasal!

*What condition—very rare, but you know of it—is associated with inappropriately and chronically elevated corticosteroid levels?*
Open-angle Glaucoma: Secondary

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What condition—very rare, but you know of it—is associated with inappropriately and chronically elevated corticosteroid levels?
Cushing syndrome
Open-angle Glaucoma: Secondary

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--Periocular injection!
--Systemic!
--Topical!
--Inhaled!
--Nasal!
--Endogenous?

What condition—very rare, but you know of it—is associated with inappropriately and chronically elevated corticosteroid levels?
Cushing syndrome

Are Cushing pts at risk for developing endogenous steroid-response IOP elevation?
Which commonly-used compound is notorious for its propensity to elevate IOP? Corticosteroids

Which of the common routes of ocular administration have been implicated in IOP elevation? How about common routes of non-ocular administration?
--Topical!
--Intravitreal injection!
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--Periocular injection!
--Systemic!
--Topical!
--Inhaled!
--Nasal!
--Endogenous!

What condition—very rare, but you know of it—is associated with inappropriately and chronically elevated corticosteroid levels? Cushing syndrome

Are Cushing pts at risk for developing endogenous steroid-response IOP elevation?
Yes
Which commonly-used compound is notorious for its propensity to elevate IOP? Corticosteroids

Which of the common routes of ocular administration have been implicated in IOP elevation? How about common routes of non-ocular administration?
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--Systemic!
--Topical!
--Inhaled!
--Nasal!

Broadly, three sorts of factors determine whether a pt will develop elevated IOP in response to steroid therapy. What are they?
--Factors related to…
--Factors related to…
--Factors related to…
Open-angle Glaucoma: Secondary

*Which commonly-used compound is notorious for its propensity to elevate IOP?* Corticosteroids

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*Broadly, three sorts of factors determine whether a pt will develop elevated IOP in response to steroid therapy. What are they?*

--Factors related to…the steroid itself
--Factors related to…the administration of the steroid
--Factors related to…the pt
**Open-angle Glaucoma: Secondary**

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- Nasal!

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- Factors related to…the steroid itself
- Factors related to…the administration of the steroid
- Factors related to…the pt

Which two properties of a steroid are key in determining whether it will cause an IOP spike?

Dexamethasone

What proportion of pts on topical dex will develop an IOP >30 after 6 weeks of therapy? About 5%
Open-angle Glaucoma: Secondary

Which commonly-used compound is notorious for its propensity to elevate IOP? Corticosteroids

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Which two properties of a steroid are key in determining whether it will cause an IOP spike? Its potency, and its ability to reach the AC

About 5%
Which commonly-used compound is notorious for its propensity to elevate IOP?
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Which two properties of a steroid are key in determining whether it will cause an IOP spike?
Its potency, and its ability to reach the AC

Which steroid tops the list in this regard?
Dexamethasone

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--Factors related to…the pt

Which two properties of a steroid are key in determining whether it will cause an IOP spike? Its potency, and its ability to reach the AC

Which steroid tops the list in this regard? Dexamethasone

About 5%
Open-angle Glaucoma: Secondary

Which commonly-used compound is notorious for its propensity to elevate IOP? Corticosteroids

Which of the common routes of ocular administration have been implicated in IOP elevation? How about common routes of non-ocular administration?
--Topical!
--Intravitreal injection!
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--Sub-Tenon’s/periocular depot!
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--Systemic!
--Topical!
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Broadly, three sorts of factors determine whether a pt will develop elevated IOP in response to steroid therapy. What are they?
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What proportion of pts on topical dex will develop an IOP >30 after 6 weeks of therapy? About 5%
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Which route of admin is more likely to elevate IOP: Topical, or systemic? Topical
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What is the relationship between the commencement of steroid therapy and the subsequent development of elevated IOP?

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Which administration-related factors play a role in determining whether a steroid-induced IOP rise will occur?
--The route
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If IOP elevation does not occur by 6 weeks, is it reasonable to assume it isn't going to occur?
No! IOP elevation can occur at any point during extended steroid therapy.
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Factors related to…the administration of the steroid

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Is steroid-induced IOP elevation a reversible condition, ie, does IOP return to baseline with cessation of steroid use?

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If the steroid is stopped within 1 year of onset, IOP will likely normalize. OTH, if it is continued longer than 18 months, IOP elevation will likely be permanent.
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What proportion of people will manifest an IOP elevation of 6-15 mmHg if treated with a prolonged course of steroids?
About 33%

What proportion of people will have an IOP rise >15?
About 5%

What common ocular condition is a risk factor for developing elevated IOP as a result of steroid use?
POAG. Per the Glaucoma book, up to 95% of POAG pts are steroid responders

In terms of aqueous dynamics (ie, its production, outflow, etc), what is the cause of a steroid-induced IOP spike?
Increased resistance to outflow at the TM

--Factors related to...the pt

A family hx of POAG is a risk factor for developing POAG. Is it also a risk factor for steroid-induced IOP spike?
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What other pt factors increase the risk of a steroid-induced IOP spike?
--?
--?
--?

[Hints forthcoming]
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Which of the common routes of ocular administration have been implicated in IOP elevation? How about common routes of non-ocular administration?
--Topical!
--Intravitreal injection!
--Intravitreal implant!
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--Periocular injection!
--Systemic!
--Topical!
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--Nasal!

Broadly, three sorts of factors determine whether a pt will develop elevated IOP in response to steroid therapy. What are they?

--Factors related to…the steroid itself
--Factors related to…the administration of the steroid
--Factors related to…the pt

What proportion of people will manifest an IOP elevation of 6-15 mmHg if treated with a prolonged course of steroids?
About 33%!

What proportion of people will have an IOP rise >15?
About 5%

What common ocular condition is a risk factor for developing elevated IOP as a result of steroid use?
POAG. Per the Glaucoma book, up to 95% of POAG pts are steroid responders

In terms of aqueous dynamics (ie, its production, outflow, etc), what is the cause of a steroid-induced IOP spike?
Increased resistance to outflow at the TM

A family hx of POAG is a risk factor for developing POAG. Is it also a risk factor for steroid-induced IOP spike?
Indeed it is

What other pt factors increase the risk of a steroid-induced IOP spike?
--Pt age [but being very old, or very young?]
--?
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Open-angle Glaucoma: Secondary

↑ IOP OAG

Primary

Secondary

Pigmentary

Tumor-Induced

Lens-Induced

Inflammation-Induced

Trauma-Related

Schwartz syndrome

Drug-Induced

Steroids

Cycloplegics

↑ EVS

↑ IOP OAG

Summary slide of the major secondary open-angle glaucomas (no question)