Uveitis

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Uveitis

*Think of the eye as being composed of three layers or ‘tunics.’* The sclera and cornea comprise the tough outer tunic. The RPE and neurosensory retina comprise the inner retinal tunic. In between these two is the highly vascular, highly pigmented tunic known as the **uvea**. (The word *uvea* derives from the Greek word for ‘grape’—an acknowledgement of the deep-purple color characteristic of most of the uvea.)
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Uveal tissue. Note its deep purple hue
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With respect to uveitis, the most important aspects of a pt’s demographics are age, gender, and ethnicity. Other important factors include geographic history (ie, where they live/lived); social history (sexual behaviors, dietary habits, etc); and vocational/avocational activities (eg, exposure to farm animals).
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**The location of the uveitis**
--Anterior
--Intermediate
--Posterior
--Panuveitis

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**The location of the uveitis**
- **Anterior**
- **Intermediate**
- **Posterior**
- **Panuveitis**

**Anterior uveitis**

*If cell is located…*

- Exclusively in the AC
  - It is called: **Iritis**

- In the AC and the anterior vitreous
  - It is called: **Iridocyclitis**

- Exclusively in the AVit
  - It is called: **Anterior cyclitis**

In **anterior uveitis**, the primary location of inflammation is the anterior chamber and/or anterior vitreous.
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**Intermediate uveitis**

If condition is...

- Idiopathic
  - It is called: **Pars planitis**
- Not idiopathic
  - It is called: **Intermediate uveitis**

The location of the uveitis

- Anterior
- Intermediate
- Posterior
- Panuveitis

**Intermediate uveitis**

The primary location of inflammation is the main vitreous cavity, +/- the peripheral retina.
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**Posterior uveitis**

*If inflammation is located…*

- Exclusively in the choroid
  - It is called: **Choroiditis**

- In both the choroid and the retina
  - It is called: **Chorioretinitis or Retinochoroiditis**

- Exclusively in the retina
  - It is called: **Retinitis**

In **posterior uveitis**, the site of inflammation is the retina and/or choroid (the optic nerve head can be involved too).
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*Once a set of potential diagnoses have been identified via profiling and meshing, lab and other studies are obtained to identify the offending condition…After which the appropriate treatment can be instituted.*
Let’s drill down on anterior uveitis. Specifically, let’s look at how the BCSC organizes it by presentation.
Anterior uveitis is by far the most common form encountered clinically. The classic symptoms are pain and photophobia, along with some degree of reduced vision. Patients will also complain of surface injection (which presents often in a so-called ‘ciliary flush’ pattern).
In surface disorders (eg, conjunctivitis), redness is either distributed uniformly across the eye, or it tapers off near the limbus.
In surface disorders (eg, conjunctivitis), redness is either distributed uniformly across the eye, or it tapers off near the limbus. In contrast, redness associated with anterior uveitis is usually most intense at and just behind the limbus. This is because this area overlies the inflamed ciliary body—hence the term *ciliary flush* for this presentation.
Uveitis

Anterior uveitis is by far the most common form encountered clinically. The classic symptoms are pain and photophobia, along with some degree of reduced vision. Patients will also complain of surface injection (which presents often in a so-called ‘ciliary flush’ pattern). At the slit lamp, the classic signs of anterior uveitis are WBCs and inflammatory proteins in the AC (‘cell and flare’).
Uveitis

Aqueous cells and flare

- cells
- flare
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The *Uveitis* book employs an organizational tree on which it hangs the common causes of anterior uveitis. The first branch point in this tree is whether the inflammation is granulomatous or nongranulomatous. In clinical practice the term granulomatous refers to a particular slit-lamp appearance of KP—large, grayish, and ‘greasy.’
Uveitis

Granulomatous KP
Granulomatous is by far the most common form encountered clinically. The classic symptoms are pain and photophobia, along with some degree of reduced vision. Patients will also complain of surface injection (which presents often in a so-called ‘ciliary flush’ pattern). At the slit lamp, the classic signs of anterior uveitis are WBCs and inflammatory proteins in the AC (‘cell and flare’). Keratic precipitates (KP)—deposits of inflammatory debris on the endothelial surface of the cornea—may be present.

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In contrast, nongranulomatous KP are smaller, lighter in color, and do not look greasy. (Note: If no KP are present, the inflammation is considered nongranulomatous.)
Uveitis

Nongranulomatous KP
These are the common entities that can produce a granulomatous anterior uveitis. (Note: For some of these, the granulomatous anterior findings are part of an overall panuveitic presentation, ie, they typically do not present as an isolated anterior uveitis.)
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The rest of the anterior-uveitis classification tree concerns **nongranulomatous dz**. The first branch-point divides the etiologies into those that produce **acute dz** vs those producing **chronic dz**.
Acute Chronic

Granulomatous Nongranulomatous

The rest of the anterior-uveitis classification tree concerns nongranulomatous dz. The first branch-point divides the etiologies into those that produce acute dz vs those producing chronic dz.

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(FYI: If a uveitis eventually relapses but is quiescent off-treatment for longer than three months, it is termed a recurrent uveitis.)
The rest of the anterior-uveitis classification tree concerns nongranulomatous dz. The first branch-point divides the etiologies into those that produce acute dz vs those producing chronic dz.

Acute uveitis comes on suddenly and resolves fairly quickly. Chronic uveitis also resolves, but once treatment is withdrawn, it relapses within three months.
Acute uveitis comes on suddenly and resolves fairly quickly. Chronic uveitis also resolves, but once treatment is withdrawn, it relapses within three months.

(FYI: If a uveitis eventually relapses but is quiescent off-treatment for longer than three months, it is termed a recurrent uveitis.)
The rest of the anterior-uveitis classification tree concerns nongranulomatous dz. The first branch-point divides the etiologies into those that produce acute dz vs those producing chronic dz.

Finally, the acute uveitides are divided into those that present unilaterally vs those that tend to present bilaterally.
The rest of the anterior-uveitis classification tree concerns nongranulomatous dz. The first branch-point divides the etiologies into those that produce acute dz vs those that tend to present bilaterally. Take a good look at this slide—it represents how you should think about anterior uveitides encountered in the clinic or on the OKAP. (It wouldn’t be a bad idea to commit this to memory at this juncture.)
Just as an FYI, these are the anterior uveitides that are covered in detail in the *Uveitis* book. **Don’t try to memorize all this at this juncture!** (They will stick better if you learn them in their naturally-occurring groupings.)
Note that syphilis, sarcoid and TB show up everywhere on the tree. This is because all three can manifest in so many different ways.
Acute Chronic
Unilateral Bilateral
Nongranulomatous
Anterior Uveitis
HLA-B27 dz
Posner-Schlossman
Syphilis
Sarcoid
HSV/VZV
TB
TINU
Leptospirosis
Behçet
Drug rxn
IBD/PA
Syphilis
Sarcoid
TB
JIA
FHI
IBD/PA
Sarcoid
Syphilis
TB

Rule of thumb:
Syphilis, sarcoid and TB are on the DDx for every pt with any form of uveitis!

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Rule of thumb:
Syphilis, sarcoid and TB are on the DDx for every pt with any form of uveitis!
Next let’s look at intermediate uveitis
The hallmark of intermediate uveitis (IU) is inflammation in the anterior vitreous that involves the vitreous base.
The hallmark of **intermediate uveitis** (IU) is inflammation in the anterior vitreous that involves the vitreous base. The vitreous base is the primary attachment point of the vitreous; it forms a ~5 mm-wide band that straddles the ora serrata (the location where the anteriormost retina meets the posteriormost portion of the ciliary body).
Uveitis

The vitreous base

- Ciliary body
- Peripheral retina
The hallmark of intermediate uveitis (IU) is inflammation in the anterior vitreous that involves the vitreous base. The vitreous base is the primary attachment point of the vitreous; it forms a ~5 mm-wide band that straddles the ora serrata (the location where the anteriormost retina meets the posteriormost portion of the ciliary body). AC cell is typically absent; if present, it’s usually mild, and is generally believed to be ‘spillover,’ ie, vitreous cells that migrated anteriorly.
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Uveitis

Snowballs in intermediate uveitis
The hallmark of intermediate uveitis (IU) is inflammation in the anterior vitreous that involves the vitreous base. The vitreous base is the primary attachment point of the vitreous; it forms a ~5 mm-wide band that straddles the ora serrata (the location where the anteriormost retina meets the posteriormost portion of the ciliary body). AC cell is typically absent; if present, it’s usually mild, and is generally believed to be ‘spillover,’ ie, vitreous cells that migrated anteriorly. Snowballs are a classic finding in IU. Snowballs—so named because of their appearance—are clumps of inflammatory cells and detritus in the vitreous. When this material accumulates in broad swaths along the inferior pars plana, it is referred to as snowbanking.
Uveitis

Snowbanking in intermediate uveitis
The hallmark of intermediate uveitis (IU) is inflammation in the anterior vitreous that involves the vitreous base. The vitreous base is the primary attachment point of the vitreous; it forms a ~5 mm-wide band that straddles the ora serrata (the location where the anteriormost retina meets the posteriormost portion of the ciliary body). AC cell is typically absent; if present, it’s usually mild, and is generally believed to be ‘spillover,’ ie, vitreous cells that migrated anteriorly. Snowballs are a classic finding in IU. Snowballs—so named because of their appearance—are clumps of inflammatory cells and detritus in the vitreous. When this material accumulates in broad swaths along the inferior pars plana, it is referred to as snowbanking.

IU tends to be a dz of young people—teens through 40 or so. It is bilateral in most (80%) cases, although it can be quite asymmetric.
Uveitis

- Intermediate
  - Anterior
  - Posterior

- Panuveitis

IU is divided up into two categories.
IU is divvied up into two categories. If the inflammation is associated with an identifiable condition, the uveitis is called **IU**.
IU is divvied up into two categories. If the inflammation is associated with an identifiable condition, the uveitis is called **IU**. If it is idiopathic, ie, if no cause can be identified, it is called **pars planitis**.
Uveitis

- Anterior
- Intermediate
- Posterior

Intermediate
- Pars planitis
- Intermediate uveitis

Panuveitis

The entities most likely to produce IU:
- MS
- Lyme
- Toxocariasis
- Sarcoid
- Syphilis
- TB
Uveitis

- Anterior
  - Pars planitis
  - Intermediate uveitis

- Intermediate
  - Intermediate uveitis

- Posterior
  - Panuveitis

- Panuveitis

Note the appearance of these three on the IU DDx as well:

- Sarcoid
- Syphilis
- TB

- MS
- Lyme
- Toxocariasis
Let’s take a closer look at posterior uveitis
Posterior uveitis

*If inflammation is located…*

- Exclusively in the choroid
  - *It is called:*
    - Chorioretinitis or Retinochoroiditis

- In both the choroid and the retina
  - *It is called:*
    - Chorioretinitis or Retinochoroiditis

- Exclusively in the retina
  - *It is called:*
    - Retinitis

As presented previously, here are the ways *posterior uveitis* can manifest.
Uveitis

**Posterior uveitis**

*If inflammation is located…*

- Exclusively in the choroid
  - *It is called:* Choroiditis

- In both the choroid and the retina
  - *It is called:* Chorioretinitis or Retinochoroiditis

- Exclusively in the retina and ONH
  - *It is called:* Retinitis
    - **Neuroretinitis**

As presented previously, here are the ways *posterior uveitis* can manifest. One more—*neuroretinitis*, inflammation involving both the retina and optic nerve—should be added for completeness sake.
Uveitis

Posterior uveitis

*If inflammation is located...*

- Exclusively in the choroid
  - *It is called:* Chorioretinitis or Retinochoroiditis

- In both the choroid *and* the retina
  - *It is called:* Choroiditis

- Exclusively in the retina *and* ONH
  - *It is called:* Retinitis
  - Neuroretinitis

As presented previously, here are the ways **posterior uveitis** can manifest. One more—**neuroretinitis**, inflammation involving both the retina and optic nerve—should be added for completeness sake.

About 80% of **anterior** uveitis cases are **non**infectious in origin. *The opposite is true for posterior uveitis:* most cases are infectious—weirdly, also about 80%.
Toxoplasmosis is a common, classic cause of posterior uveitis. It is infectious, the bug being *Toxoplasma gondii*, an obligate intracellular parasite. The cat is its definitive host. *T gondii* has a worldwide distribution; an estimated one billion people are infected. Humans usually acquire the parasite via consumption of unwashed produce or undercooked meat.
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Uveitis

Toxoplasma gondii: Three infectious forms

**Oocyst form**
- Found in GI tract of cat (shed in feces)
- Acquired via ingestion of unwashed produce

**Tachyzoite form**
- Found in circulatory system of infected mother
- Responsible for transplacental infection

**Tissue cyst**
- Found in tissue of infected livestock
- Acquired via consumption of undercooked meat
Toxoplasmosis is a common, classic cause of posterior uveitis. It is infectious, the bug being *Toxoplasma gondii*, an obligate intracellular parasite. The cat is its definitive host. *T. gondii* has a worldwide distribution; an estimated one billion people are infected. Humans usually acquire the parasite via consumption of unwashed produce or undercooked meat. Another crucial mechanism of transmission is transplacentally, which leads to devastating congenital manifestations in affected infants (it is one of the TORCH syndrome etiologies).

The typical posterior-uveitis manifestation of toxoplasmosis is a retinochoroiditis accompanied by a dense overlying vitritis. Taken together, the appearance has been likened to a ‘headlight in the fog.’
Uveitis

Ocular toxoplasmosis: Headlight in the fog
Let's take a closer look at Panuveitis.
To qualify as a panuveitis, all compartments of the eye—the AC, vitreous, and retina/choroid—must be equally involved in the inflammatory process. Panuveitis is usually a bilateral condition, although it may be asymmetric.
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- Sarcoid
- Sympathetic ophthalmia
- Vogt-Koyanagi-Harada
- Behçet syndrome
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- Sympathetic ophthalmia
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- Syphilis
- TB
- Lyme
- Leptospirosis
- Whipple dz
To qualify as a panuveitis, all compartments of the eye—the AC, vitreous, and retina/choroid—must be equally involved in the inflammatory process. Panuveitis is usually a bilateral condition, although it may be asymmetric. The panuveitides are divvied into Noninfectious and Infectious causes.

Note that these three appear yet again:

Sarcoid
Sympathetic ophthalmia
Vogt-Koyanagi-Harada
Behçet syndrome

Syphilis
TB
Lyme
Leptospirosis
Whipple dz
Now we’ll change gears and look at endophthalmitis
Endophthalmitis

The *Uveitis* book defines **endophthalmitis** as an inflammatory process involving both the AC and vitreous cavities that is secondary to bacterial or fungal infection.
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Endogenous Endophthalmitis

The *Uveitis* book defines **endophthalmitis** as an inflammatory process involving both the AC and vitreous cavities that is secondary to bacterial or fungal infection. Endophthalmitis can be **posttraumatic, postoperative** or **endogenous**.

*Endogenous endophthalmitis* involves hematogenous spread of infection from a remote location to the eye. It is uncommon, accounting for less than 10% of all cases of endophthalmitis. Individuals at increased risk of endogenous endophthalmitis include those with impaired immune status, those who recently underwent an invasive medical procedure, and those subjected to chronic and/or repeated breaching of the body’s outer barrier.
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*The fact that the route is hematogenous indicates a nidus of infection is present somewhere in the body, and it is incumbent upon the pt’s care team to find and treat it!*
As mentioned, endogenous endophthalmitis can be bacterial or fungal.
As mentioned, endogenous endophthalmitis can be bacterial or fungal. **Bacterial endophthalmitis** presents with the expected ocular signs of pain, redness, and decreased vision. Additional ocular signs include periorbital/lid edema, a dense AC reaction (often with hypopyon), and vitreous inflammation. Retinal microabscesses may be present, including white-centered hemorrhages (aka Roth spots). Significantly, systemic findings of infection—fever, elevated white count, malaise, etc—will likely be present as well.
Bacterial endophthalmitis
Endogenous Endophthalmitis

Bacterial

Fungal

As mentioned, endogenous endophthalmitis can be bacterial or fungal. **Bacterial endophthalmitis** presents with the expected ocular signs of pain, redness, and decreased vision. Additional ocular signs include periorbital/lid edema, a dense AC reaction (often with hypopyon), and vitreous inflammation. Retinal microabscesses may be present, including white-centered hemorrhages (aka **Roth spots**). Significantly, systemic findings of infection—fever, elevated white count, malaise, etc—will likely be present as well. Both Gram(+) bugs (esp. **Staph, Strep, Bacillus**) and Gram(-) bugs (esp. **Neisseria, H flu, E coli** and **Klebsiella**) may be responsible. Often, the pt’s clinical and/or social situation provide important clues regarding the pathogen responsible.
As mentioned, endogenous endophthalmitis can be bacterial or fungal. **Bacterial endophthalmitis** presents with the expected ocular signs of pain, redness, and decreased vision. Additional ocular signs include periorbital/lid edema, a dense AC reaction (often with hypopyon), and vitreous inflammation. Retinal microabscesses may be present, including white-centered hemorrhages (aka Roth spots). Significantly, systemic findings of infection—fever, elevated white count, malaise, etc—will likely be present as well. Both Gram(+) bugs (esp. Staph, Strep, Bacillus) and Gram(-) bugs (esp. Neisseria, H flu, E coli and Klebsiella) may be responsible. **Often, the pt’s clinical and/or social situation provide important clues regarding the pathogen responsible.**

Some classic bacterial pathogen associations in endogenous endophthalmitis:
- **Endocarditis:** Strep
- **Skin infections:** Staph
- **IVDU:** Bacillus
- **Liver abscess:** Klebsiella
Endogenous Endophthalmitis

In contrast to the bacterial version, endogenous fungal endophthalmitis tends to be more insidious in onset. It generally progresses in a particular fashion. First, isolated choroidal metastatic lesions appear. With time, these break through Bruch’s membrane to involve the retina. Eventually, the bug reaches the vitreous, and (if still unchecked) the anterior segment.
Candida endophthalmitis: Choroidal lesions
Candida endophthalmitis: Classic ‘string of pearls’ vitreous involvement
In contrast to the bacterial version, endogenous **fungal** endophthalmitis tends to be more insidious in onset. It generally progresses in a particular fashion. First, isolated choroidal metastatic lesions appear. With time, these break through Bruch’s membrane to involve the retina. Eventually, the bug reaches the vitreous, and (if still unchecked) the anterior segment. Yeasts (esp. *Candida* and *Cryptococcus*), molds (*Aspergillus*) and dimorphic species (*Coccidioides*) may be responsible. As with bacterial, the pt’s clinical and/or social situation can provide important clues regarding the pathogen.
In contrast to the bacterial version, endogenous fungal endophthalmitis tends to be more insidious in onset. It generally progresses in a particular fashion. First, isolated choroidal metastatic lesions appear. With time, these break through Bruch’s membrane to involve the retina. Eventually, the bug reaches the vitreous, and (if still unchecked) the anterior segment. Yeasts (esp. *Candida* and *Cryptococcus*), molds (*Aspergillus*) and dimorphic species (*Coccidioides*) may be responsible. **As with bacterial, the pt’s clinical and/or social situation can provide important clues regarding the pathogen.**

**Some classic fungal pathogen associations in endogenous endophthalmitis:**

-- *Chronic indwelling lines/catheters*: Candida
-- *HIV/AIDS*: Cryptococcus
-- *Hx liver transplantation*: Aspergillus
-- *San Joaquin valley*: Coccidioides
Next let’s take a look at scleritis
Scleritis

Scleritis is an inflammatory condition characterized by painful infiltrative scleral edema and congestion of the deep episcleral plexus. It can be extremely painful, and can lead to blindness and loss of the eye. Women are more likely to be affected than are men. It is rare in children.
Scleritis

Scleritis is an inflammatory condition characterized by painful infiltrative scleral edema and congestion of the deep episcleral plexus. It can be extremely painful, and can lead to blindness and loss of the eye. Women are more likely to be affected than are men. It is rare in children.

To make matters worse, it can herald the presence or worsening of a systemic conditions that may be potentially lethal. About 40% of scleritis pts have an identifiable systemic inflammatory condition, the most common of which is rheumatoid arthritis.
Uveitis

Scleritis is divvied up with respect to whether the...
Uveitis

**Scleritis** is divvied up with respect to whether the...

Anterior sclera is affected, vs the Posterior sclera.
Uveitis

Scleritis

Anterior

Posterior

There are three classic signs of anterior scleritis:
--Scleral edema
--
Anterior scleritis: Scleral edema. Note the thickening of the limbal sclera (b) in comparison to the unaffected fellow eye (a)
There are three classic signs of anterior scleritis:
-- Scleral edema
-- Sclera has a violaceous hue
--
Anterior scleritis: Violaceous hue
Scleritis

There are three classic signs of anterior scleritis:
--Scleral edema
--Sclera has a violaceous hue
--Inflamed vasculature has a criss-cross pattern
‘Criss-cross’ injection of the deep vasculature in anterior scleritis. To see it, you have to look past the brighter injection of the inflamed overlying conj vessels.
Anterior scleritis comes in three forms: *Diffuse, nodular and necrotizing*
Uveitis

Diffuse anterior scleritis
Nodular anterior scleritis
Scleritis

Anterior
- Diffuse
- Nodular
- Necrotizing
  - w/ inflammation
  - w/o inflammation

Posterior

Necrotizing anterior scleritis comes in two forms: with and without inflammation.
Uveitis

Necrotizing anterior scleritis with inflammation
Contrary to the implications of the name, inflammation is present in *necrotizing scleritis w/o inflammation*. It is so named because, unlike its ‘with inflammation’ cousin, it is typically painless, and the eye does not *appear* inflamed.
Uveitis

Necrotizing anterior scleritis without inflammation
Scleritis

Uveitis

Contrary to the implications of the name, inflammation is present in *necrotizing scleritis w/o inflammation*. It is so named because, unlike its ‘with inflammation’ cousin, it is typically painless, and the eye does not appear inflamed. *Necrotizing scleritis w/o inflammation* is also known as *scleromalacia perforans*. It is strongly associated with RA.
Unlike anterior scleritis, posterior scleritis does not present with a red eye, and nodules are not present. Instead, posterior scleritis presents with:

- **Proptosis**
- **Disc edema**
- **Motility disorders**
- **Retinal/choroidal findings**

**Scleritis**

**Anterior**

- Diffuse
  - w/ inflammation
  - w/o inflammation

**Posterior**
Uveitis

Posterior scleral thickening with proptosis

Posterior scleritis: Proptosis
Unlike anterior scleritis, **posterior scleritis** does not present with a red eye, and nodules are not present. Instead, posterior scleritis presents with:

-- Proptosis
-- Disc edema

**Scleritis**

- Anterior
- Posterior
- Diffuse

**Uveitis**

- w/ inflammation
- w/o inflammation
Uveitis

Posterior scleritis OD: Optic nerve edema
Unlike anterior scleritis, **posterior scleritis** does not present with a red eye, and nodules are not present. Instead, posterior scleritis presents with:

- **Proptosis**
- **Disc edema**
- **Retinal/choroidal findings**
Posterior scleritis producing retinal folds
Unlike anterior scleritis, posterior scleritis does not present with a red eye, and nodules are not present. Instead, posterior scleritis presents with:

--- Proptosis
--- Disc edema
--- Retinal/choroidal findings
--- Motility disorders
The pain of posterior scleritis has three characteristics that should alert you to the diagnosis:

- Awakens the patient at night
- If you encounter descriptions such as this in the clinic (or on the OKAP), think posterior scleritis!
Scleritis

Anterior
  Diffuse
  Nodular

Posterior

The pain of posterior scleritis has three characteristics that should alert you to the diagnosis:
--The pain radiates to the brow
--The pain is aggravated by eye movements
--The pain awakens the pt at night

Uveitis
Scleritis

Anterior

Posterior

Diffuse

Nodular

w/ inflammation

The pain of posterior scleritis has three characteristics that should alert you to the diagnosis:
--The pain radiates to the brow
--The pain is aggravated by eye movements
--The pain awakens the pt at night
If you encounter descriptions such as this in the clinic (or on the OKAP), think posterior scleritis!
An easy-to-obtain imaging study for confirming the diagnosis of posterior scleritis is **B-scan ultrasonography**. B-scan will reveal choroidal thickening and sub-Tenon’s edema.
Uveitis

Posterior scleritis: Sub-Tenon’s edema
An easy-to-obtain imaging study for confirming the diagnosis of posterior scleritis is **B-scan ultrasonography**. B-scan will reveal choroidal thickening and sub-Tenon’s edema. When sub-Tenon’s edema involves the space around the optic nerve, the classic **T sign** finding will result.
Uveitis

Posterior scleritis: T-sign
Uveitis

Scleritis

Anterior
- Diffuse
- Nodular
- Necrotizing
  - w/ inflammation
  - w/o inflammation

Posterior

Scleritis requires systemic treatment. Diffuse scleritis might respond to PO NSAIDs—try these first. For the others, PO steroids are usually the first-line med, although NSAIDs may be tried. More powerful immunosuppression is frequently required.
Scleritis requires systemic treatment. Diffuse scleritis might respond to PO NSAIDs—try these first. For the others, PO steroids are usually the first-line med, although NSAIDs may be tried. More powerful immunosuppression is frequently required. Subconj depot steroids, long considered contraindicated, have recently gained wide acceptance as a treatment option.
Finally, we will look at masquerade syndromes
Uveitis

Masquerade Syndrome refers to entities that mimic immune-mediated dz.
Uveitis

Masquerade Syndrome refers to entities that mimic immune-mediated dz.

The entities can be broadly divided into Nonneoplastic and Neoplastic causes.
The most common entity to masquerade as intraocular uveitis is **primary vitreoretinal lymphoma** (PVRL).
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Virtually all PVRLs are non-Hodgkin B-cell lymphomas. The typical PVRL pt is an adult in their 50s-60s. They usually present with complaints of decreased vision and/or floaters.
The most common entity to masquerade as intraocular uveitis is **primary vitreoretinal lymphoma** (PVRL).

Virtually all PVRLs are non-Hodgkin B-cell lymphomas. The typical PVRL pt is an adult in their 50s-60s. They usually present with complaints of decreased vision and/or floaters. Importantly, many will also manifest evidence of CNS involvement, the most common being changes in behavior or personality. Other, more obvious S/S include seizures, cerebellar signs, hemiparesis and cranial nerve palsies. Confusion, weakness, and memory loss may also occur.
The most common entity to masquerade as intraocular uveitis is primary vitreoretinal lymphoma (PVRL).
Uveitis

PVRL: Typical white-yellow subretinal infiltrates
The most common entity to masquerade as intraocular uveitis is **primary vitreoretinal lymphoma (PVRL)**.

Virtually all PVRLs are non-Hodgkin B-cell lymphomas. The typical PVRL pt is an adult in their 50s-60s. They usually present with complaints of decreased vision and/or floaters. Importantly, many will also manifest evidence of CNS involvement, the most common being changes in behavior or personality. Other, more obvious S/S include seizures, cerebellar signs, hemiparesis and cranial nerve palsies. Confusion, weakness, and memory loss may also occur. DFE in PVRL typically reveals subretinal infiltrates described as “**creamy yellow**” in color. The infiltrates can mimic the findings of other, more common conditions (eg, toxoplasmosis). **PVRL is diagnosed by finding ‘big blue cells’ on vitreous biopsy.**
Uveitis

Typical cytology of PVRL cells from the vitreous showing several atypical lymphoid cells with basophilic cytoplasm and large prominent irregular nuclei.
Other hematologic neoplasias can masquerade as well, but are far less common than PVRL.
Uveitis

Masquerade Syndrome

Neoplastic

Nonneoplastic

Solid

Hematologic

Leukemic

Leukemia

Lymphoid

Primary

Uveal melanoma

Rb

Mets

Lung

Breast

Primary vitreoretinal lymphoma

Secondary to systemic lymphoma

Lymphoproliferative dz

Solid tumors can masquerade as well.
Lung and breast are the solid malignancies most likely to be implicated in a masquerade syndrome. Commonly, they will present with bilateral multifocal choroidal lesions mimicking choroiditis, often with an overlying vitritis.
Uveitis

Bilateral metastatic lung cancer
Uveitis

Bilateral metastatic breast cancer
These are the nonneoplastic masquerade entities discussed in the *Uveitis* book.
Ocular ischemic syndrome (OIS) is a constellation of ocular abnormalities stemming from chronic hypoperfusion of the globe. The classic cause is carotid stenosis ipsilateral to the eye in question. The typical pt is an elderly vasculopathic male.
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Four findings, common in OIS, can (mis)lead one to conclude the pt has uveitis:

--AC cell and flare
--Low IOP
--Neovascularization of the iris and/or angle
--Cataract more advanced on that side
Uveitis

**Masquerade Syndrome**

**Neoplastic**

**Lymphoid**
- Primary vitreoretinal lymphoma
- Secondary to systemic lymphoma
- Lymphoproliferative dz

**Leukemic**
- Leukemia
- Uveal melanoma
- Rb

**Nonneoplastic**
- RP
- OIS
- Chronic rhegmatogenous RD
- IOFB
- Pigment dispersion syndrome
- Juvenile xanthogranuloma

The hallmark of *PDS* is the liberation of pigment from the posterior aspect of the iris. This pigment subsequently migrates into the anterior chamber, where the pigment granules can be mistaken for inflammatory cells. Typically, retroillumination of the iris will reveal transillumination defects with a radial orientation.
Uveitis

Masquerade Syndrome

Nonneoplastic

Neoplastic

Lymphoid
- Leukemia
  - Primary vitreoretinal lymphoma
  - Secondary to systemic lymphoma
  - Lymphoproliferative dz

Leukemic

Primary vitreoretinal lymphoma
Secondary to systemic lymphoma
Lymphoproliferative dz

Pigment dispersion syndrome

RP
OIS
Chronic rhegmatogenous RD
IOFB
Juvenile xanthogranuloma

The hallmark of PDS is the liberation of pigment from the posterior aspect of the iris. This pigment subsequently migrates into the anterior chamber, where the pigment granules can be mistaken for inflammatory cells. Typically, retroillumination of the iris will reveal transillumination defects with a radial orientation.
Uveitis

PDS: Radial transillumination defects