Uveitis

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Uveitis

Uveal tissue. Note its deep purple hue
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**Identifying a uveitis is, in essence, a pattern-recognition task.** The uveitides do not present in random fashion; rather, they ‘select’ their victims based on pt demographics. Likewise, the nonocular manifestations of those 2ndry to a systemic condition tend to follow specific patterns as well. Each tends to affect the eye in a characteristic manner.
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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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Because nonocular manifestations are so important in identifying the underlying cause of a uveitis, a thorough review of systems and physical exam are absolutely essential in uveitis management. (Some uveitis experts assert that the ROS is the single most important component of a uveitis evaluation.)
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**Let’s drill down on the different ways uveitis manifests within the eye.**

**One very important manifestation issue is location, i.e., the portion or segment of the eye that’s involved.**
Uveitis

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I say ‘technically speaking’ because in clinical practice, *uveitis* refers to inflammatory processes involving most non-uveal segments of the eye as well, (limited to) the retina, optic nerve, sclera, etc. The observed inflammation is the product of an ocular disease in some cases, whereas the ocular inflammation is actually a manifestation of a *systemic* inflammatory condition. A key component of uveitis management is distinguishing between ocular-only and systemic cases, as the two require different responses on the part of the treating physician. Because of this, a significant component of uveitis education involves learning how to discern which cases are which.

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**The location of the uveitis**

--Anterior
--Intermediate
--Posterior
--Panuveitis

Let’s drill down on the different ways uveitis manifests within the eye. One very important manifestation issue is location, ie, the portion or segment of the eye that’s involved. Regarding intraocular inflammation, four locations are recognized. Let’s look at each for a moment.
Technically speaking, the term "uveitis" refers to inflammation of one or more components of the uvea. The uvea includes the choroid, ciliary body, and iris. As is always the case with inflammatory disease, the etiology of ocular inflammation can be infectious or noninfectious. I say "technically speaking" because in clinical practice, "uveitis" refers to inflammatory processes involving most non-uveal segments of the eye as well, including (but not limited to) the cornea, retina, optic nerve, sclera, etc.

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

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

**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**

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**Intermediate uveitis**
- **If condition is…**
  - Idiopathic
  - Not idiopathic
- **The location of the uveitis**
  - Anterior
  - Intermediate
  - Posterior
  - Panuveitis

**Intermediate uveitis**
- **It is called:**
  - Pars planitis
  - Intermediate uveitis

**In intermediate uveitis**, the primary location of inflammation is the main vitreous cavity, +/- the peripheral retina.
Technically speaking, the term **uveitis** refers to inflammation of one or more components of the *uvea*. The three structures that comprise the uvea are the choroid, ciliary body, and iris. As is always the case with inflammatory disease, the etiology of ocular inflammation can be infectious or noninfectious.

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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**

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**The location of the uveitis**

- Anterior
- Intermediate
- Posterior
- Panuveitis

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

**In panuveitis, all three locations are equally involved**
Many experts endorse a *profiling and meshing* approach to diagnosing uveitis.
Uveitis

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*Profiling* refers to identifying germane aspects of the pt’s personal history (age, ethnicity, occupation, etc); nonocular signs and symptoms associated with the uveitis (eg, skin findings; CNS involvement); and key features of the inflammation itself (ie, location, duration, etc).
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**Meshing** refers to matching the profile of the pt with the known proclivities of specific uveitic entities. In this way, a DDx is generated:
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‘*VKH* affects adults of ME descent, produces a bilateral granulomatous panuveitis, and is associated with CNS manifestations, especially tinnitus’
‘*Lyme uveitis* is granulomatous, and CNS manifestations are very common.’
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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 (e.g., 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 overlaps the inflamed ciliary body—hence the term *ciliary flush* for this 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). 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
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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, i.e., they typically do not present as an isolated anterior uveitis.)
Granulomatous

The rest of the anterior-uveitis classification tree concerns non-granulomatous dz.
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**Acute uveitis** comes on suddenly and resolves fairly quickly.
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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**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*.
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*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.)
Acute Chronic
Unilateral Bilateral
Granulomatous Nongranulomatous
Anterior Uveitis

HLA-B27 dz Posner-Schlossman HSV/VZV
Sarcoid Syphilis TB

Acute
Unilateral
HLA-B27 dz
Posner-Schlossman
HSV/VZV
Sarcoid Syphilis TB
Bilateral
TINU Behçet
Drug rxn Leptospirosis
IBD/PA
Sarcoid Syphilis TB

Chronic
JIA FHI IBD/PA
Sarcoid Syphilis TB

Note that syphilis, sarcoid and TB show up everywhere on the tree. This is because all three can manifest in so many different ways.
Rule of thumb:
Syphilis, sarcoid and TB are on the DDx for every pt with any form of uveitis!
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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**.
Snowbanking in intermediate uveitis
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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.
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

Posterior

Intermediate uveitis

Pars planitis

Intermediate

The entities most likely to produce IU:

- MS
- Lyme
- Toxocariasis
- Sarcoid
- Syphilis
- TB

Anterior

Panuveitis
Note the appearance of these three on the IU DDx as well.
Let’s take a closer look at posterior uveitis
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
  - *It is called:* Retinitis

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

Posterior uveitis

*If inflammation is located...*

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

- Exclusively in the retina and ONH
  - *It is called:* Retinitis
  - *It is called:* 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.
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About 80% of anterior uveitis cases are noninfectious 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
Uveitis

Posterior uveitis

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  - Neuroretinitis

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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**.’
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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The panuveitides are divvied into **Noninfectious** and **Infectious** causes.
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- Sarcoid
- Sympathetic ophthalmia
- Vogt-Koyanagi-Harada
- Behçet syndrome
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- Sarcoid
- Sympathetic ophthalmia
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- Syphilis
- TB
- Lyme
- Leptospirosis
- Whipple dz
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Note that these three appear yet again:

- Sarcoid
- Sympathetic ophthalmia
- Syphilis
- TB
- Vogt-Koyanagi-Harada
- Lyme
- Behçet syndrome
- Leptospirosis
- Whipple dz
Endophthalmitis

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!
Endogenous Endophthalmitis

- Bacterial
- Fungal

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

- **Endocarditis:** Strep
- **Skin infections:** Staph
- **IVDU:** Bacillus
- **Liver abscess:** Klebsiella
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. **Yeast**s (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.
Endogenous Endophthalmitis

Bacterial

Fungal

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 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

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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.
There are three classic signs of anterior scleritis:
--Scleral edema
--
Uveitis

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
--
Uveitis

Anterior scleritis: Violaceous hue
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*
Diffuse anterior scleritis
Nodular anterior scleritis
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
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:

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**Proptosis**

**Disc edema**

---

w/ inflammation  
w/o inflammation
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
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

**Scleritis**

**Anterior**

**Posterior**

**Diffuse**

**Nodular**

w/ inflammation  w/o inflammation

**Uveitis**
Uveitis

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:

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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**
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
**Scleritis**

**Scleritis**

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

- **Posterior**

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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.
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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.
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The most common entity to masquerade as intraocular uveitis is **primary vitreoretinal lymphoma** (PVRL)

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**Lymphoid**

**Primary vitreoretinal lymphoma**
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.
Uveitis

Masquerade Syndrome

Nonneoplastic

Neoplastic

Hematologic

Leukemic

Lymphoid

Leukemia

Primary vitreoretinal lymphoma

Secondary to systemic lymphoma

Lymphoproliferative dz

Other hematologic neoplasias can masquerade as well, but are far less common than PVRL
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**
  - **Nonneoplastic**
    - **RP**
    - **OIS**
    - **Chronic rhegmatogenous RD**
    - **IOFB**
    - **Pigment dispersion syndrome**
    - **Juvenile xanthogranuloma**

**Lymphoid**

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

**Leukemic**

- **Uveal melanoma**
- **Rb**

**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.**
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