What pathologic process results in cotton-wool spots (CWS)?
What pathologic process results in cotton-wool spots (CWS)?
Interruption of the retinal Blood supply, ie, ischemia
How many blood supplies does the retina receive?
How many blood supplies does the retina receive? Two
What are the sources of the retina’s two blood supplies?
Blood supply: 
Central retinal artery

What are the sources of the retina’s two blood supplies?

Blood supply: 
Choriocapillaris
**Retinal Layers**
- Internal limiting membrane
- Nerve fiber layer
- Ganglion cell layer
- Inner plexiform layer
- Inner nuclear layer
- Outer plexiform layer (Henle’s layer)
- Outer nuclear layer
- External limiting membrane
- Rod/cone inner and outer segments

**RPE**

**Bruch’s membrane**
A

- Retinal Layers
  - Internal limiting membrane
  - Nerve fiber layer
  - Ganglion cell layer
  - Inner plexiform layer
  - Inner nuclear layer
  - Outer plexiform layer (Henle’s layer)
  - Outer nuclear layer
  - External limiting membrane
  - Rod/cone inner and outer segments

- RPE
- Bruch’s membrane

Blood supply: *Central retinal artery*

Which layers are supplied by each blood supply?

Blood supply: *Choriocapillaris*
Q

- Retinal Layers
  - Internal limiting membrane?
  - Nerve fiber layer?
  - Ganglion cell layer?
  - Inner plexiform layer?
  - Inner nuclear layer?
  - Outer plexiform layer (Henle’s layer)?
  - Outer nuclear layer?
  - External limiting membrane?
  - Rod/cone inner and outer segments?

- RPE
- Bruch’s membrane

Which retinal layer is involved in CWS?

Blood supply: Central retinal artery

Which layers are supplied by each blood supply?

Blood supply: Choriocapillaris
A

Retinal Layers
- Internal limiting membrane
- Nerve fiber layer
- Ganglion cell layer
- Inner plexiform layer
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- Outer nuclear layer
- External limiting membrane
- Rod/cone inner and outer segments

RPE

Bruch’s membrane

Which retinal layer is involved in CWS?
The nerve fiber layer (NFL)

Blood supply:
- Central retinal artery:
  Which layers are supplied by each blood supply?
  Inner 2/3 of INL on in
  Outer 1/3 of INL on out

Blood supply: Choriocapillaris
Retinal Layers
- Internal limiting membrane
- Nerve fiber layer
- Ganglion cell layer
- Inner plexiform layer
- Inner nuclear layer
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- Outer nuclear layer
- External limiting membrane
- Rod/cone inner and outer segments
- RPE
- Bruch’s membrane

What does this imply about the etiology of CWS?

Blood supply:
- Central retinal artery

Which layers are supplied by each blood supply?
- Choriocapillaris

Which retinal layer is involved in CWS?
The nerve fiber layer (NFL)
- **Retinal Layers**
  - Internal limiting membrane
  - **Nerve fiber layer**
  - Ganglion cell layer
  - Inner plexiform layer
  - Inner nuclear layer
  - Outer plexiform layer (Henle’s layer)
  - Outer nuclear layer
  - External limiting membrane
  - Rod/cone inner and outer segments

- **RPE**
- **Bruch’s membrane**

**Blood supply:**
- Central retinal artery

**What does this imply about the etiology of CWS?**
It implies the infarction occurs within branches of the CRA.

**Which retinal layer is involved in CWS?**
The nerve fiber layer (NFL).
In one word, what sort of structure are the NFL ‘fibers’?
Nerve fiber layer

*In one word, what sort of structure are the NFL ‘fibers’?*

The fibers are **axons**
In one word, what sort of structure are the NFL ‘fibers’?
The fibers are axons.

Speaking of axons—to what does the term axoplasmic flow refer?

Axoplasmic stasis leads to swelling and opacification of the axon at the site of accumulation. If enough axons in an area are similarly affected, the collective appearance of all those swollen/opacified axons results in the ophthalmoscopic finding known as a CWS.
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\textit{What is the relationship between CWS and axoplasmic stasis?}
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*Speaking of ophthalmoscopy—what do CWS look like?*
--Size: 
--Color:
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Speaking of ophthalmoscopy—what do CWS look like? --Size: Usually ≤ ¼ DD --Color:
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**Speaking of ophthalmoscopy—what do CWS look like?**
--Size: Usually ≤ ¼ DD
--Color: White (is why they are called **cotton wool spots**
In one word, what sort of structure are the NFL ‘fibers’? The fibers are axons.

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Do CWS resolve spontaneously?  Speaking of ophthalmoscopy—what do CWS look like?  --Size: Usually ≤ ¼ DD  --Color: White (is why they are called cotton wool spots)
Nerve fiber layer

In one word, what sort of structure are the NFL ‘fibers’? The fibers are **axons**.

Speaking of axons—to what does the term *axoplasmic flow* refer? It refers to the movement of organelles, proteins, lipids, etc, along the length of an axon (i.e., to and from the cell body).

To what does the term *axoplasmic stasis* refer? If axoplasmic flow is interrupted, the material being transported comes to rest and accumulates at the site of the interruption. This is the state of ‘axoplasmic stasis.’

What is the relationship between CWS and axoplasmic stasis? Axoplasmic stasis leads to swelling and opacification of the axon at the site of accumulation. If enough axons in an area are similarly affected, the collective appearance of all those swollen/opacified axons results in the ophthalmoscopic finding known as a CWS.

Do CWS resolve spontaneously? Yes

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Do CWS resolve spontaneously? Yes

How long does it take?

Speaking of ophthalmoscopy—what do CWS look like? --Size: Usually ≤ ¼ DD --Color: White (is why they are called **cotton wool spots**).
In one word, what sort of structure are the NFL ‘fibers’?
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**Do CWS resolve spontaneously?**
Yes

**How long does it take?**
A month or two

**Speaking of ophthalmoscopy—what do CWS look like?**
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If axoplasmic flow is interrupted, the material being transported comes to rest and accumulates at the site of the interruption. This is the state of 'axoplasmic stasis.'

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Axoplasmic stasis leads to swelling and opacification of the axon at the site of accumulation. If enough axons in an area are similarly affected, the collective appearance of all those swollen/opacified axons results in the ophthalmoscopic finding known as a **CWS**.

Do they resolve without sequelae, or with?  
Resolves  

- **How long does it take?**  
  - A month or two

Do CWS resolve spontaneously?  
Yes

What do CWS look like?  
- Size: Usually ≤ ¼ DD  
- Color: White (is why they are called **cotton wool spots**)

Do CWS resolve spontaneously?  
Yes

**How long does it take?**  
A month or two
In one word, what sort of structure are the NFL ‘fibers’? The fibers are axons.

Speaking of axons—to what does the term axoplasmic flow refer? It refers to the movement of organelles, proteins, lipids, etc, along the length of an axon (ie, to and from the cell body).

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What is the relationship between CWS and axoplasmic stasis? Axoplasmic stasis leads to swelling and opacification of the axon at the site of accumulation. If enough axons are similarly affected, this collective appearance of all those swollen/opacified axons results in the ophthalmoscopic finding known as a CWS.

Do they resolve without sequelae, or with? With

Do CWS resolve spontaneously? Yes

How long does it take? A month or two

Do they resolve without sequelae? Yes

What are the sequelae? The RNFL at the site is a little atrophic/thinned.
In one word, what sort of structure are the NFL ‘fibers’?
The fibers are axons.

Speaking of axons—to what does the term axoplasmic flow refer?
It refers to the movement of organelles, proteins, lipids, etc, along the length of an axon (ie, to and from the cell body).

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What is the relationship between CWS and axoplasmic stasis?
Axoplasmic stasis leads to swelling and opacification of the axon at the site of accumulation. If enough axons are similarly affected, the collective appearance of all those swollen/opacified axons can result in the ophthalmoscopic finding known as a CWS.

Do CWS resolve without sequelae, or with?
With

What is the sequelae?
The RNFL at the site is a little atrophic/thinned.

Do CWS resolve spontaneously?
Yes

How long does it take?
A month or two
Nerve fiber layer

In one word, what sort of structure are the NFL ‘fibers’?
The fibers are axons.

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To what does the term axoplasmic stasis refer?
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What is the relationship between CWS and axoplasmic stasis?
Axoplasmic stasis leads to swelling and opacification of the axon at the site of accumulation. If enough axons become similarly affected, the collective appearance of all those swollen/opacified axons will produce the ophthalmoscopic finding known as a CWS.

Do CWS resolve without sequelae, or with?
Yes

With

What is the sequelae?
The RNFL at the site is a little atrophic/thinned.

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Yes

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What is the relationship between CWS and axoplasmic stasis?
Axoplasmic stasis leads to swelling and opacification of the axon at the site of accumulation. If enough axons in an area are similarly affected, the collective appearance of all those swollen/opacified axons results in the ophthalmoscopic finding known as a CWS.

If axoplasmic flow is interrupted at the ONH, what condition results?
In one word, what sort of structure are the NFL ‘fibers’?

The fibers are **axons**

*Speaking of axons—to what does the term axoplasmic flow refer?*

It refers to the movement of organelles, proteins, lipids, etc, along the length of an axon (ie, to and from the cell body)

*To what does the term axoplasmic stasis refer?*

If axoplasmic flow is interrupted, the material being transported comes to rest and accumulates at the site of the interruption. This is the state of ‘axoplasmic stasis.’

*What is the relationship between CWS and axoplasmic stasis?*

Axoplasmic stasis leads to swelling and opacification of the axon at the site of accumulation. If enough axons in an area are similarly affected, the collective appearance of all those swollen/opacified axons results in the ophthalmoscopic finding known as a CWS.

*If axoplasmic flow is interrupted at the ONH, what condition results?*

Disc edema
In one word, what sort of structure are the NFL ‘fibers’?
The fibers are **axons**

What cells are they the axons of?
Nerve fiber layer

*In one word, what sort of structure are the NFL ‘fibers’?*
The fibers are **axons**

*What cells are they the axons of?*
Retinal ganglion cells
In one word, what sort of structure are the NFL ‘fibers’?
The fibers are axons

What cells are they the axons of?
Retinal ganglion cells

Where are the cell bodies of the ganglion cells located?
Retinal Layers

- Internal limiting membrane
- Nerve fiber layer
- Ganglion cell layer
- Inner plexiform layer
- Inner nuclear layer
- Outer plexiform layer
- Outer nuclear layer
- External limiting membrane
- Rod/cone inner and outer segments

RPE

Bruch’s membrane

In one word, what sort of structure are the NFL ‘fibers’?
The fibers are axons

What cells are they the axons of?
Retinal ganglion cells

Where are the cell bodies of the ganglion cells located?
In the ganglion cell layer
Retinal Layers

- Internal limiting membrane
- Nerve fiber layer
- Ganglion cell layer
- Inner plexiform layer
- Inner nuclear layer

In one word, what sort of structure are the NFL ‘fibers’?
The fibers are axons.

What cells are they the axons of?
Retinal ganglion cells.

These ganglion-cell axons--where are they headed?

RPE
Bruch’s membrane
Retinal Layers
- Internal limiting membrane
- Nerve fiber layer
- Ganglion cell layer
- Inner plexiform layer
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In one word, what sort of structure are the NFL ‘fibers’? The fibers are axons

What cells are they the axons of? Retinal ganglion cells

These ganglion-cell axons--where are they headed? To the optic nerve head (ONH)

RPE
- Bruch’s membrane
Q

Retinal Layers
- Internal limiting membrane
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In one word, what sort of structure are the NFL ‘fibers’? The fibers are axons.

What cells are they the axons of? Retinal ganglion cells.

These ganglion-cell axons--where are they headed? To the optic nerve head (ONH).

Will they synapse at the ONH?

RPE
- Bruch’s membrane
Q/A

- Retinal Layers
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In one word, what sort of structure are the NFL ‘fibers’?
The fibers are axons

What cells are they the axons of?
Retinal ganglion cells

These ganglion-cell axons--where are they headed?
To the optic nerve head (ONH)

Will they synapse at the ONH?
No. The ONH contains no synapses; it is simply the aggregate of nerve fibers as they leave the globe via a hole in the sclera called the lamina cribrosa.

- RPE
- Bruch’s membrane
Retinal Layers

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The fibers are **axons**

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- RPE
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● **Retinal Layers**
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  - Ganglion cell layer
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  These ganglion-cell axons--where are they headed?
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  **Will they synapse at the ONH?**
  **No.** The ONH contains no synapses, it is simply the aggregate of nerve fibers as they leave the globe via a hole in the sclera called the lamina cribrosa.

  **OK, then where will they synapse?**
  Most will synapse in the lateral geniculate nucleus (LGN)

● **RPE**

● **Bruch’s membrane**
Retinal Layers

- Internal limiting membrane
- Nerve fiber layer
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Most will synapse in the lateral geniculate nucleus (LGN)

Most? Where will the others synapse, and what are they responsible for?
Q/A

- Retinal Layers
  - Internal limiting membrane
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  - Ganglion cell layer
  - Inner plexiform layer
  - Inner nuclear layer

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The fibers are axons

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OK, then where will they synapse?
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Most? Where will the others synapse, and what are they responsible for?
Most of the others are involved in the pupillary light reflex; they peel off just prior to reaching the LGN, heading instead to the pretectum of the dorsal midbrain to synapse in the pretectal nuclei.

- RPE
- Bruch’s membrane
Retinal Layers

- Internal limiting membrane
- Nerve fiber layer
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A

Cotton Wool Spots
Retinal Layers

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- Inner nuclear layer
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- Bruch's membrane
- Retinal Layers

In one word, what sort of structure are the NFL ‘fibers’?

The fibers are axons

What cells are they the axons of?

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These ganglion-cell axons--where are they headed?

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There’s that word again. Where will the others synapse, and what are they responsible for?

Most of the others are involved in the pupillary light reflex; they peel off just prior to reaching the LGN, heading instead to the pretectum of the dorsal midbrain to synapse in the pretectal nuclei.
Q/A

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Most of the others are involved in the pupillary light reflex; they peel off just prior to reaching the LGN, heading instead to the pretectum of the dorsal midbrain to synapse in the pretectal nuclei

There’s that word again. Where will the others synapse, and what are they responsible for?
The hypothalamus, where they are involved in modulating activity
Retinal Layers

- Internal limiting membrane
- Nerve fiber layer
- Ganglion cell layer
- Inner plexiform layer
- Inner nuclear layer
- Outer plexiform layer
- Outer nuclear layer
- External limiting membrane
- Rod/cone inner and outer segments
- RPE
- Bruch's membrane

In one word, what sort of structure are the NFL ‘fibers’?
The fibers are axons

What cells are they the axons of?
Retinal ganglion cells

These ganglion-cell axons--where are they headed?
To the optic nerve head (ONH)

Will they synapse at the ONH?
No. The ONH contains no synapses, it is simply the aggregate of nerve fibers as they leave the globe via a hole in the sclera called the lamina cribrosa

OK, then where will they synapse?
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What is its eponymous name?
Parinaud syndrome

The hypothalamus, where they are involved in modulating circadian activity

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What cells are the axons of? Retinal ganglion cells.

Where are the cell bodies of the ganglion cells located? In the ganglion cell layer.

Where will the NFL fibers synapse? To the optic nerve head.

Will they synapse at the optic nerve head (ONH)? No. The ONH contains no synapses; it is the aggregate of nerve fibers as they leave the globe via a hole in the sclera called the lamina cribrosa.

OK, then where will they synapse? Most will synapse in the lateral geniculate nucleus (LGN). The others are involved in the pupillary light reflex; they peel off just prior to reaching the LGN, heading instead to the pretectal nuclei of the dorsal midbrain. Where will these others synapse? Most of the others are involved in modulating circadian activity, so they will synapse in the hypothalamus.

There is an important clinical entity caused by damage to the pretectal nuclei of the dorsal midbrain. What is its eponymous name? Parinaud syndrome.

The two noneponymous names for Parinaud syndrome are: 1) Pretectal syndrome, 2) Dorsal midbrain syndrome.
Q

- Retinal Layers
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Parinaud syndrome has four classic clinical features. What are they?
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Parinaud syndrome has four classic clinical features. What are they?

- Light-near dissociation
- Impaired upgaze
- Lid retraction
- Convergence-retraction nystagmus

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What are the two noneponymous names for Parinaud syndrome?

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There is an important clinical entity caused by damage to the pretectal nuclei of the dorsal midbrain. What is its eponymous name?
Parinaud syndrome

The hypothalamus, where they are involved in modulating circadian activity
What is the clinical status of most pts with CWS?
What is the clinical status of most pts with CWS?

diabetic
What should you do if a nondiabetic pt has CWS?
What should you do if a **nondiabetic** pt has CWS?
Work it up!
The rule:
Work up even 1 cotton-wool spot in a nondiabetic patient!

(No question--proceed when ready)
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. 

- Sphygnamometry
- Echocardiogram
- Carotid dopplers
- Hgb electrophoresis
- History for head/neck CA
- ESR, CRP, ANCA
- ANA, RF
- CBC with diff
- ELISA

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- CBC with diff
- ELISA
- Hj

Note: By *reasonable battery* I don’t mean that all nondiabetic pts with CWS should undergo all of the tests. As always, the history, ROS and exam should be used to winnow and motivate any tests pursued.

(No question--proceed when ready)
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient.

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- Echocardiogram
- Carotid dopplers
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- ELISA
- Hj, Chj

First things first: Check ‘em for DM. (Maybe they just don’t realize they have it, or are in denial)

(No question--proceed when ready)
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. **nondiabetic**

- Sphygnamometry
- Echocardiogram
- Carotid dopplers
- Hgb electrophoresis
- History for head/neck CA
- ESR, CRP, ANCA
- ANA, RF
- CBC with diff
- ELIS

**First things first:** Check ‘em for DM. (Maybe they just don’t realize they have it, or are in denial)

**What percent of ‘nondiabetics’ with CWS will be found to have elevated blood sugar?**
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient.

- Sphygnamometry
- Echocardiogram
- Carotid dopplers
- Hgb electrophoresis
- History for head/neck CA
- ESR, CRP, ANCA
- ANA, RF
- CBC with diff
- ELIS
- Vhj, chjkA

First things first: Check 'em for DM. (Maybe they just don’t realize they have it, or are in denial)

What percent of ‘nondiabetics’ with CWS will be found to have elevated blood sugar? One study (quoted by an Academy publication) pegged it at 20%!
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

- Sphygnamometry:
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. *What disease is being ruled out with each?*

- **Sphygnamometry**: HTN
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. *What disease is being ruled out with each?*

- **Sphygmanometry:** HTN

*What percent of ‘nonhypertensives’ with CWS will be found to have elevated BP?*
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

- **Sphygnamometry**: HTN

What percent of ‘nonhypertensives’ with CWS will be found to have elevated BP?

50% (per the same study mentioned previously)
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

- Sphygnamometry: HTN
- Echocardiogram:
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. *What disease is being ruled out with each?*

- **Sphygnamometry:** HTN
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- ELISA
- Vκ, γδκA

Broadly speaking, what sort of disease is sickle-cell anemia?
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- **ESR, CRP, ANCA**
- **ANA, RF**
- **CBC with diff**
- **ELIS**
- **Vk, gjkA**

Broadly speaking, what sort of disease is sickle-cell anemia? A hemoglobinopathy

What are the four common genotypes?

- SS
- SC
- S-Thal
- SA

In America, people of which two ethnic heritages are at greatest risk?

African-American; Hispanic-American
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

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**Sickle-cell disease**
The following tests constitute a reasonable battery in working up a cotton-wool spot in a non-diabetic patient. What disease is being ruled out with each?

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- **Echocardiogram:** Cardiac embolic source
- **Carotid dopplers:** Carotid embolic source
- **Hgb electrophoresis:** Sickle-cell disease
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What percent of African-Americans test positive for sickle trait?
8% (1 in 12)

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What is the incidence of significant retinopathy in: S-Thal vs SA disease? The first three manifest as clinically apparent dz, whereas SA is an asymptomatic (usually) carrier state—aka 'sickle trait'

What percent of African-Americans test positive for sickle trait? 8% (1 in 12)

In America, people of which two ethnic heritages are at greatest risk? African-American; Hispanic-American

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- **Carotid dopplers:**
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- **Hgb electrophoresis:**
  - Sickle-cell disease

- **History for head/neck CA**

- **ESR, CRP, ANCA**

- **ANA, RF**

- **CBC with diff**

- **ELIS**

- **VkJ, gkJA**

Broadly speaking, what sort of disease is sickle-cell anemia? A hemoglobinopathy

What are the four common genotypes?

- **SS**
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- **SA**

What is the incidence of significant retinopathy in:

- **SS dz? 3%**
- **SC dz? 33%**

What percent of African-Americans test positive for sickle trait?

- **8% (1 in 12)**

In America, people of which two ethnic heritages are at greatest risk?

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- **SS dz?** 3%
- **SC dz?** 33%

Sickle-cell retinopathy comes in two basic forms—what are they?

- **Nonproliferative (NPSR), and proliferative (PSR)**

Nonproliferative and proliferative—just like diabetic retinopathy?

- **Indeed**

Like DBR, does sickle retinopathy tend to occur in the posterior pole?

- **No—unlike DBR, sickle retinopathy tends to occur well into the periphery (usually anterior to the equator)**
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient.

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Nonproliferative and proliferative—just like diabetic retinopathy? Indeed

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In a very few words, what is the pathogenesis of NPSR?

Capillary and/or arteriolar occlusion

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NPSR manifests as three lesions. What are they? What is their appearance?

- **Salmon patches:** Retinal hemorrhage trapped under the internal limiting membrane
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What is the pathogenesis of PSR?
As with NPSR, it's vascular occlusion, but severe enough to produce significant ischemia

By what appearance-based name are sickle-cell neovascular lesions known?
'Sea fans'

Do sea fans always require treatment?
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Broadly speaking, what sort of disease is sickle-cell anemia? Also, what is it?

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**Cotton Wool Spots**

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In a word, what common form of retinopathy does radiation retinopathy resemble clinically?
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Of the following manifestations of DBR, which can present in radiation retinopathy?
- CWS? Yes (duh, that’s what the slide-set is about)
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- Macular edema?
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What specific form of radiation is implicated in radiation retinopathy? Ionizing radiation

What two mechanisms/techniques for delivering ionizing radiation to the eye are employed in clinical practice?

- External-beam
- Plaque therapy

Is one modality more likely than the other to result in radiation retinopathy?

No—delivery modality is not a risk factor for the development of retinopathy

If delivery modality isn't a risk factor, what is?

The total radiation dose, area of retina treated, and fractionation schedule.
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If delivery modality isn't a risk factor, what is?
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In a word, what common form of retinopathy does radiation retinopathy resemble clinically? Diabetic retinopathy (DRB)

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All can be manifestations of radiation retinopathy!

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PMHx for head/neck CA: **Radiation retinopathy**

How quickly does retinopathy develop after radiation therapy?

Not very—typically months to years

What factors influence the time-to-onset of retinopathy?

Here is where modality is a risk factor. The average latency for external-beam therapy is 18 months, whereas retinopathy after plaque therapy tends to develop more rapidly.
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- ANA, RF: **Collagen-vascular disease**
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. *What disease is being ruled out with each?*

- **Sphygnamometry:** HTN
- **Echocardiogram:** Cardiac embolic source
- **Carotid dopplers:** Carotid embolic source
- **Hgb electrophoresis:** Sickle-cell disease
- **PMHx for head/neck CA:** Radiation retinopathy
- **ESR, CRP, ANCA:** Vasculitis
- **ANA, RF:** **Collagen-vascular disease**

*To what does the term collagen-vascular disease refer?*
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

- Sphygmonamometry: HTN
- Echocardiogram: Cardiac embolic source
- Carotid dopplers: Carotid embolic source
- Hgb electrophoresis: Sickle-cell disease
- PMHx for head/neck CA: Radiation retinopathy
- ESR, CRP, ANCA: Vasculitis
- ANA, RF: Collagen-vascular disease

To what does the term collagen-vascular disease refer?
It is a catch-all (and outdated—more shortly) term for systemic rheumatologic conditions that present with arthralgias, vascular s/s, and skin changes (many other manifestations can occur as well)
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

- **Sphyngamometry:** HTN
- **Echocardiogram:** Cardiac embolic source
- **Carotid dopplers:** Carotid embolic source
- **Hgb electrophoresis:** Sickle-cell disease
- **PMHx for head/neck CA:** Radiation retinopathy
- **ESR, CRP, ANCA:** Vasculitis
- **ANA, RF:** Collagen-vascular disease
- **CBC with diff**
- **ELISA**
- **Bjerk:**
- **VHA**

**To what does the term collagen-vascular disease refer?**

It is a catch-all (and outdated—more shortly) term for systemic rheumatologic conditions that present with arthralgias, vascular s/s, and skin changes (many other manifestations can occur as well).
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

- **Sphygmonometry:** HTN

**Why is the term collagen-vascular dz outdated?**

It was once thought that the causal nexus of these conditions was found in the tissue that manifests them; i.e., that these conditions were primary disorders of collagen (‘connective’) and vascular tissues. However, we now know that it is derangements of the immune system that are responsible for these conditions—connective and vascular tissue are simply the locations at which the immune derangements declare themselves. Because the term **collagen-vascular dz** (and the related **connective-tissue dz**) reflect a (mis)understanding of the pathology involved, they are no longer preferred.

- **ANA, RF:** Collagen-vascular disease

**To what does the term collagen-vascular disease refer?**

It is a catch-all (and even—more shortly) term for systemic rheumatologic conditions that present with arthralgias, vascular s/s, and skin changes (many other manifestations can occur as well).
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

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- **ESR, CRP, ANCA:** Vasculitis
- **ANA, RF:** Collagen-vascular disease
- **CBC with diff:**
- **ELIS:**

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**Q**

**Collagen-vascular disease**

To what does the term collagen-vascular disease refer?

It is a catch-all (and more shortly) term for systemic rheumatologic conditions that present with arthralgias, vascular s/s, and skin changes (many other manifestations can occur as well)

Why is the term collagen-vascular dz outdated?

It was once thought that the causal nexus of these conditions was found in the tissue that manifests them—ie, that these conditions were primary disorders of collagen (‘connective’) and vascular tissue. However, we now know that it is derangements of the immune system that are responsible for these conditions—connective and vascular tissue are simply the locations at which the immune derangements declare themselves. Because the term collagen-vascular dz (and the related connective-tissue dz) reflect a (mis)understanding of the pathology involved, they are no longer preferred.

OK then, if ‘collagen-vascular disease’ and ‘connective-tissue disease’ are outdated, what is the preferred term?

**Systemic rheumatic disease**
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. *What disease is being ruled out with each?*

- **Sphygnamometry**: HTN
- **Echocardiogram**: Cardiac embolic source
- **Carotid dopplers**: Carotid embolic source
- **Hgb electrophoresis**: Sickle-cell disease
- **PMHx for head/neck CA**: Radiation retinopathy
- **ESR, CRP, ANCA**: Vasculitis
- **ANA, RF**: Collagen-vascular disease
- **CBC with diff**
- **ELIS**

Why is the term collagen-vascular dz outdated?
It was once thought that the causal nexus of these conditions was found in the tissue that manifested them. Therefore disorders of collagen (‘connective’) and vascular tissue are simply the locations at which the immune system is deranged. Because the term *collagen-vascular dz* (and the related connective-tissue dz) reflect a (mis)understanding of the pathology involved, they are no longer preferred.

OK then, if ‘collagen-vascular disease’ and ‘connective-tissue disease’ are outdated, *what is the preferred term?* ‘Systemic rheumatic disease’

To *what does the term* collagen-vascular disease *refer?*
It is a catch-all (and, more shortly) term for systemic rheumatologic conditions that present with arthralgias, vascular s/s, and skin changes (many other manifestations can occur as well)
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

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- **ESR, CRP, ANCA:** Vasculitis
- **ANA, RF:** Collagen-vascular disease
- **CBC with diff:**

Why is the term collagen-vascular dz outdated?
It was once thought that the causal nexus of these conditions was found in the tissue that manifests vascular response, which disorders of collagen (‘connective’) and vascular tissue are simply the locations at which the immune derangements of the immune system that are responsible for these conditions—connective and vascular tissue are simply the locations at which the immune derangements of the immune system that are responsible for these conditions—are manifest. Because the term collagen-vascular dz (and the related connective-tissue dz) reflect a (mis)understanding of the pathology involved, they are no longer preferred.

OK then, if ‘collagen-vascular disease’ and ‘connective-tissue disease’ are outdated, what is the preferred term?
‘Systemic rheumatic disease’

When you hear the terms ‘collagen-vascular disease’ and ‘cotton-wool spots’ in the same sentence, one disease should immediately come to mind. What is it?
Systemic lupus erythematosus
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

- **Sphygnamometry:** HTN
- **Echocardiogram:** Cardiac embolic source
- **Carotid dopplers:** Carotid embolic source
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- **PMHx for head/neck CA:** Radiation retinopathy
- **ESR, CRP, ANCA:** Vasculitis
- **ANA, RF:** Collagen-vascular disease
- **CBC with diff:**

**Why is the term collagen-vascular dz outdated?**

It was once thought that the causal nexus of these conditions was found in the tissue that manifested vascular responses, which are disorders of collagen (‘connective’) and vascular tissue are simply the locations at which immune system derangements of the **immune system** that are responsible for these conditions—connective and vascular tissue are simply the locations at which the immune derangements declare themselves. Because the term *collagen-vascular dz* (and the related connective-tissue dz) reflect a (mis)understanding of the pathology involved, they are no longer preferred.

**OK then, if ‘collagen-vascular disease’ and ‘connective-tissue disease’ are outdated, what is the preferred term?**

‘Systemic rheumatic disease’

When you hear the terms ‘collagen-vascular disease’ and ‘cotton-wool spots’ in the same sentence, one disease should immediately come to mind. What is it? **Systemic lupus erythematosus**

and skin changes (many other manifestations can occur as well)
Who is the classic lupus pt?

- A woman of childbearing age
- Yes, black and Hispanic women are at greater risk than are white women
- About 3-30%
- It heralds CNS and/or renal involvement with the disease, and carries a high risk of mortality
- With the big dogs: Plasmapheresis + IV cyclophosphamide acutely

Q: When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it?

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- **ESR, CRP, ANCA:** Vasculitis
- **ANA, RF:** Collagen-vascular disease
- **CBC with diff:**
- **ELISA:**

When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it? **Systemic lupus erythematosus**

Who is the classic lupus pt?
- A. M v F

Is there a racial predilection?
- Yes, black and Hispanic women are at greater risk than are white women

What proportion of lupus pts manifest retinal findings?
- About 3-30%

Why is lupus-associated retinal arteritis an ominous finding?
- It heralds CNS and/or renal involvement with the disease, and carries a high risk of mortality

Given its dire systemic significance, how should lupus-associated retinal arteritis be managed?
- With the big dogs: Plasmapheresis + IV cyclophosphamide acutely

M v F
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

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- **PMHx for head/neck CA:** Radiation retinopathy
- **ESR, CRP, ANCA:** Vasculitis
- **ANA, RF:** Collagen-vascular disease
- **CBC with diff:**

When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it?

**Systemic lupus erythematosus**

Who is the classic lupus pt?

A woman of childbearing age

Is there a racial predilection?

Yes, black and Hispanic women are at greater risk than are white women

What proportion of lupus pts manifest retinal findings?

About 3-30%

Why is lupus-associated retinal arteritis an ominous finding?

It heralds CNS and/or renal involvement with the disease, and carries a high risk of mortality.

Given its dire systemic significance, how should lupus-associated retinal arteritis be managed?

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When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it?

**Systemic lupus erythematosus**

Who is the classic lupus pt?
A woman of childbearing age

Is there a racial predilection?
Yes, black and Hispanic women are at greater risk than are white women.

What proportion of lupus pts manifest retinal findings?
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Why is lupus-associated retinal arteritis an ominous finding?
It heralds CNS and/or renal involvement with the disease, and carries a high risk of mortality.

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- **CBC with diff:**
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When you hear the terms ‘collagen-vascular disease’ and ‘cotton-wool spots’ in the same sentence, one disease should immediately come to mind. What is it?

**Systemic lupus erythematosus**

Who is the classic lupus pt?
A woman of childbearing age

Is there a racial predilection?
Yes, black and Hispanic women are at greater risk than are white women.

What proportion of lupus pts manifest retinal findings?
About 3-30%

Why is lupus-associated retinal arteritis an ominous finding?
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- **ESR, CRP, ANCA:** Vasculitis
- **ANA, RF:** Collagen-vascular disease
- **CBC with diff**: 
- **ELIS**: 

When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it? **Systemic lupus erythematosus**

Who is the classic lupus pt?
A woman of childbearing age

Is there a racial predilection?
Yes, black and Hispanic women are at greater risk than are white women

(3 different ethnicities)

When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it? **Systemic lupus erythematosus**
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

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- **PMHx for head/neck CA**: Radiation retinopathy
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When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it? **Systemic lupus erythematosus**

*Who is the classic lupus pt?*
A woman of childbearing age

*Is there a racial predilection?*
Yes, black and Hispanic women are at greater risk than are white women
Who is the classic lupus pt?
A woman of childbearing age

Is there a racial predilection?
Yes, black and Hispanic women are at greater risk than are white women

What proportion of lupus pts manifest retinal findings?

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- **PMHx for head/neck CA:** Radiation retinopathy
- **ESR, CRP, ANCA:** Vasculitis
- **ANA, RF:** Collagen-vascular disease
- **CBC with diff**

When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it? **Systemic lupus erythematosus**

**Who is the classic lupus pt?**
- A woman of childbearing age

**Is there a racial predilection?**
- Yes, black and Hispanic women are at greater risk than are white women

**What proportion of lupus pts manifest retinal findings?**
- About 3-30%

**Why is lupus-associated retinal arteritis an ominous finding?**
- It heralds CNS and/or renal involvement with the disease, and carries a high risk of mortality

**Given its dire systemic significance, how should lupus-associated retinal arteritis be managed?**
- With the big dogs: Plasmapheresis + IV cyclophosphamide acutely
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

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- **PMHx for head/neck CA:** Radiation retinopathy
- **ESR, CRP, ANCA:** Vasculitis
- **ANA, RF:** Collagen-vascular disease
- **CBC with diff:**
- **ELIS Bjk. Vhj, A**

When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it?

**Systemic lupus erythematosus**

**Who is the classic lupus pt?**
A woman of childbearing age

**Is there a racial predilection?**
Yes, black and Hispanic women are at greater risk than are white women

**What proportion of lupus pts manifest retinal findings?**
About 3-30%

**What retinal findings may occur?**

- Occlusive events: Asymptomatic cotton wool spots
- If 'lucky,' an infarcted macula if not
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

- Sphygmamometry: HTN
- Echocardiogram: Cardiac embolic source
- Carotid dopplers: Carotid embolic source
- Hgb electrophoresis: Sickle-cell disease
- PMHx for head/neck CA: Radiation retinopathy
- ESR, CRP, ANCA: Vasculitis
- ANA, RF: Collagen-vascular disease
- CBC with diff

When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it?

Systemic lupus erythematosus
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

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- **Hgb electrophoresis:** Sickle-cell disease
- **PMHx for head/neck CA:** Radiation retinopathy
- **ESR, CRP, ANCA:** Vasculitis
- **ANA, RF:** Collagen-vascular disease
- **CBC with diff:** ESR, CRP, ANCA
- **ELIS Bjk:** ANA, RF

When you hear the terms ‘collagen-vascular disease’ and ‘cotton-wool spots’ in the same sentence, one disease should immediately come to mind. What is it?

**Systemic lupus erythematosus**

Who is the classic lupus pt?
A woman of childbearing age

Is there a racial predilection?
Yes, black and Hispanic women are at greater risk than are white women

What proportion of lupus pts manifest retinal findings?
About 3-30%

What retinal findings may occur?
Occlusive events: Asymptomatic cotton wool spots
if ‘lucky,’ an infarcted macula if not

*CWS are the classic manifestation of lupus retinopathy!*

(No question—proceed when ready)
Who is the classic lupus pt?
A woman of childbearing age

Is there a racial predilection?
Yes, black and Hispanic women are at greater risk than are white women

What proportion of lupus pts manifest retinal findings?
About 3-30%

Note: This range represents a compromise between inconsistencies in the BCSC series:
--Rate of lupus retinopathy per the Retina book: 3-10%
--Rate per the Uveitis book: 3-29%
(I rounded to 30 to make it easier to remember)

(No question—proceed when ready)

When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it?
Systemic lupus erythematosus
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

- **Sphygnamometry:** HTN
- **Echocardiogram:** Cardiac embolic source
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- **Hgb electrophoresis:** Sickle-cell disease
- **PMHx for head/neck CA:** Radiation retinopathy
- **ESR, CRP, ANCA:** Vasculitis
- **ANA, RF:** Collagen-vascular disease
- **CBC with diff:**

When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it?

**Systemic lupus erythematosus**
The following tests constitute a reasonable battery in working up a cotton-wool spot in a non-diabetic patient. What disease is being ruled out with each?

- **Sphygnamometry**: HTN
- **Echocardiogram**: Cardiac embolic source
- **Carotid dopplers**: Carotid embolic source
- **Hgb electrophoresis**: Sickle-cell disease
- **PMHx for head/neck CA**: Radiation retinopathy
- **ESR, CRP, ANCA**: Vasculitis
- **ANA, RF**: Collagen-vascular disease
- **CBC with diff**: Erythrocytosis
- **ELISA**: IgM, IgG, IgA

When you hear the terms ‘collagen-vascular disease’ and ‘cotton-wool spots’ in the same sentence, one disease should immediately come to mind. What is it?

**Systemic lupus erythematosus**

Who is the classic lupus pt?
A woman of childbearing age

Is there a racial predilection?
Yes, black and Hispanic women are at greater risk than are white women

What proportion of lupus pts manifest retinal findings?
About 3-30%

Why is lupus-associated retinal arteritis an ominous finding?
It heralds CNS and/or renal involvement with the disease, and carries a high risk of mortality.

Given its dire systemic significance, how should lupus-associated retinal arteritis be managed?
With the big dogs: Plasmapheresis + IV cyclophosphamide acutely.
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

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- **Hgb electrophoresis:** Sickle-cell disease
- **PMHx for head/neck CA:** Radiation retinopathy
- **ESR, CRP, ANCA:** Vasculitis
- **ANA, RF:** Collagen-vascular disease
- **CBC with diff:**
- **ELISA:**

**Question:**

Q

When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it?

**Systemic lupus erythematosus**

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**Who is the classic lupus pt?**
A woman of childbearing age

**Is there a racial predilection?**
Yes, black and Hispanic women are at greater risk than are white women

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About 3-30%

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When you hear the terms collagen-vascular disease and cotton-wool spots’ in the same sentence, one disease should immediately come to mind. What is it?
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A woman of childbearing age

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- **ESR, CRP, ANCA:** Vasculitis
- **ANA, RF:** Collagen-vascular disease
- **CBC with diff ELIS Bjkn A:**

When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it?

**Systemic lupus erythematosus**

Who is the classic lupus pt?
A woman of childbearing age

Is there a racial predilection?
Yes, black and Hispanic women are at greater risk than are white women

What proportion of lupus pts manifest retinal findings?
About 3-30%

Why is lupus-associated retinal arteritis an ominous finding?
It heralds CNS and/or renal involvement with the disease, and carries a high risk of mortality

Given its dire significance, how should lupus-associated retinal arteritis be managed?
With the big dogs: Plasmapheresis + IV cyclophosphamide

What med is notorious for causing drug-induced SLE?
Procainamide

Generally speaking, what sort of drug is it?
An anti-arrhythmic

Can procainamide-induced SLE cause cotton-wool spots?
You betcha. It also can cause macular ischemia resulting in severe vision loss
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

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- **Echocardiogram:** Cardiac embolic source
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- **ESR, CRP, ANCA:** Vasculitis
- **ANA, RF:** Collagen-vascular disease
- **CBC with diff:**
- **ELIS**

When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it?

**Systemic lupus erythematosus** secondary to...**procainamide**

Who is the classic lupus pt?
A woman of childbearing age

Is there a racial predilection?
Yes, black and Hispanic women are at greater risk than are white women

What proportion of lupus pts manifest retinal findings?
About 3-30%

Why is lupus-associated retinal arteritis an ominous finding?
It heralds CNS and/or renal involvement with the disease, and carries a high risk of mortality

Given its dire systemic significance, how should lupus-associated retinal arteritis be managed?
With the big dogs: Plasmapheresis + IV cyclophosphamide

What med is notorious for causing drug-induced SLE?
**Procainamide**

Generally speaking, what sort of drug is it?
An anti-arrhythmic

Can procainamide-induced SLE cause CWS?
You betcha. It also can cause macular ischemia resulting in severe vision loss
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

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- **CBC with diff**
- **ELISA**

When you hear the terms ‘collagen-vascular disease’ and ‘cotton-wool spots’ in the same sentence, one disease should immediately come to mind. What is it?

**Systemic lupus erythematosus**

Who is the classic lupus pt?
A woman of childbearing age

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What med is notorious for causing drug-induced SLE?
**Procainamide**

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You betcha. It also can cause macular ischemia resulting in severe vision loss
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- **ELIS Bjk. Vhj,

When you hear the terms 'collagen-vascular disease' and 'cotton-wool spots' in the same sentence, one disease should immediately come to mind. What is it? **Systemic lupus erythematosus**

**Who is the classic lupus pt?**
A woman of childbearing age

**Is there a racial predilection?**
Yes, black and Hispanic women are at greater risk than are white women

**What proportion of lupus pts manifest retinal findings?**
About 3-30%

**Why is lupus-associated retinal arteritis an ominous finding?**
It heralds CNS and/or renal involvement with the disease, and carries a high risk of mortality.

**Given its dire significance, how should lupus-associated retinal arteritis be managed?**
With the big dogs: Plasmapheresis + IV cyclophosphamide acutely secondary to procainamide.

**What med is notorious for causing drug-induced SLE?**
Procainamide

**Generally speaking, what sort of drug is it?**
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**Q**

Cotton Wool Spots
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- **Hx of compressive chest trauma:** Purtscher’s retinopathy

*Is HIV retinopathy common?* Yes—it occurs in up to 70% of HIV pts.

*What is the appearance of HIV retinopathy?* Cotton-wool spots in the posterior pole +/- MAs and DBH

*What is the pathophysiology?* Arteriolar occlusion → focal ischemia → disruption of axoplasmic flow → CWS
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What is the classic DFE appearance in Purtscher’s retinopathy?

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**What is the classic clinical scenario?**
An acute hx of compressive trauma to the chest (it can occur in the context of head trauma as well)
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. 

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What is the classic DFE appearance in Purtscher’s retinopathy?

Multiple peripapillary CWS surrounding a relatively normal-appearing ONH (disc edema may be present). Additional areas of retinal whitening are usually present, as are small intraretinal hemorrhages.

There are other ‘classic clinical scenarios’ that get mentioned in this context. What are they?

- Pancreatitis, amniotic-fluid embolization, long-bone fracture (there are others). But to be clear, these do not cause Purtscher’s retinopathy.
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**What is the classic clinical scenario?**
An acute hx of compressive trauma to the chest trauma as well.

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**Wadda ya mean, ‘these don’t cause Purtscher’s’? Everyone knows they do. What’s the deal?**

**What is the classic clinical scenario?**

An acute hx of compressive trauma to head trauma as well.

**What are other scenarios?**

Pancreatitis, amniotic-fluid embolization, long-bone fracture (there are others). But to be clear, *these do not cause Purtscher’s retinopathy*.

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**Wadda ya mean, ‘these don’t cause Purtscher’s’? Everyone knows they do. What’s the deal?**

It’s true that these conditions can cause a retinopathy identical in appearance to Purtscher’s. That said, Dr Purtscher’s original description was in the context of thoracic or head trauma. Thus, technically speaking, the term Purtscher retinopathy is reserved for only situations in which the retinopathy results from thoracic/head trauma.

What is the classic clinical scenario? An acute hx of compressive trauma to the chest, or head trauma as well.

Other scenarios: Pancreatitis, amniotic-fluid embolization, long-bone fracture (there are others). But to be clear, these do not cause Purtscher’s retinopathy.

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What is the classic DFE appearance in Purtscher's retinopathy?
Multiple peripapillary CWS surrounding a relatively normal-appearing ONH (disc edema may be present). Additional areas of non-CWS retinal whitening are usually present, as are small intraretinal hemorrhages.

What is the classic clinical scenario?
An acute hx of compressive trauma to the chest (it can occur in the context of head trauma as well).

Are other clinical scenarios mentioned in this context? What are they? Pancreatitis, amniotic-fluid embolization, long-bone fracture, etc.

OK then, what is the name for the Purtscher's-like retinopathy due to pancreatitis, amniotic-fluid embolization, long-bone fracture, etc?
Purtscher's retinopathy
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

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What is the classic clinical scenario?

An acute hx of compressive trauma to the chest (it can occur in the context of head trauma as well). There are other scenarios that are usually mentioned in this context. What are they? Pancreatitis, amniotic-fluid embolization, long-bone fracture, etc?

OK then, what is the name for the Purtscher's-like retinopathy due to pancreatitis, amniotic-fluid embolization, long-bone fracture, etc?

It's called 'Purtscher's-like retinopathy'

Wadda ya mean, 'these don't cause Purtscher's'? Everyone knows they do. What's the deal?

It's true that these conditions can cause a retinopathy identical in appearance to Purtscher's. That said, Dr Purtscher's original description was in the context of thoracic or head trauma. Thus, technically speaking, the term *Purtscher retinopathy* is reserved for only situations in which the retinopathy results from thoracic/head trauma.

OK then, what is the name for the Purtscher's-like retinopathy due to pancreatitis, amniotic-fluid embolization, long-bone fracture, etc?

It's called 'Purtscher's-like retinopathy'

Are other scenarios get mentioned in this context. What are they? Pancreatitis, amniotic-fluid embolization, long-bone fracture (there are others). But to be clear, these do not cause Purtscher's retinopathy.
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What is the pathologic process underlying Purtscher’s?

- Complement activation
- Granulocyte aggregation
- Leukoembolization
- Occlusion of small retinal arterioles

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There are other scenarios. What is the classic clinical scenario? An acute hx of compressive trauma to head trauma as well.

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**Let's tackle this question in reverse. What is the direct, proximal cause of retinal hemorrhages in Purtscher’s?**

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It’s called ‘Purtscher’s-like retinopathy’

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That said, Dr Purtscher’s original description was in the context of thoracic or head trauma. Thus, technically speaking, these are other ‘classic clinical scenarios’ that get mentioned in this context. What are they? Pancreatitis, amniotic-fluid embolization, long-bone fracture (there are others). But to be clear, these do not cause Purtscher’s retinopathy.

What is the classic DFE appearance in Purtscher’s retinopathy?

Multiple peripapillary CWS surrounding a relatively normal-appearing ONH (disc edema may be present). Additional areas of non-CWS retinal whitening are usually present, as are small intraretinal hemorrhages.

What is the classic clinical scenario?

An acute hx of compressive trauma to the chest (it can occur in the context of head trauma as well)

What is the pathologic process underlying Purtscher’s?

Complement activation → granulocyte aggregation → occlusion of small retinal arterioles
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

- **Sphygmametry:** HTN
- **Echocardiogram:** Cardiac embolic source
- **Carotid dopplers:** Carotid embolic source
- **Hgb electrophoresis:** Sickle-cell disease
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There are other scenarios that get mentioned in this context. What are they? Pancreatitis, amniotic-fluid embolization, long-bone fracture, etc. But to be clear, these do not cause Purtscher’s retinopathy.

What is the pathologic process underlying Purtscher’s?

- Complement activation → granulocyte aggregation → occlusion of small retinal arterioles

Let’s tackle this question in reverse. What is the direct, proximal cause of retinal hemorrhages in Purtscher’s?

**Occlusion of small retinal arterioles**
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

- Sphygmamometry: HTN
- Echocardiogram: Cardiac embolic source
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What is the classic clinical scenario?

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Let’s tackle this question in reverse. What is the direct, proximal cause of retinal hemorrhages in Purtscher’s?

Occlusion of small retinal arterioles

What is the cause of the occlusion?

Leukoembolization

What is the pathologic process underlying Purtscher’s?

Complement activation → granulocyte aggregation → occlusion of small retinal arterioles

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It’s true that these conditions can cause a retinopathy identical in appearance to Purtscher’s. That said, Dr Purtscher’s original description was in the context of thoracic or head trauma. Thus, technically speaking, the term ‘Purtscher’s’ is reserved for only situations in which the retinopathy results from thoracic/head trauma.
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  - HTN

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- **Hgb Electrophoresis:**
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- **PMHx for Head/Neck CA:**
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- **ESR, CRP, ANCA:**
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- **ANA, RF:**
  - Collagen-vascular disease

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  - Leukemia; severe anemia

- **ELISA:**
  - HIV

- **Hx of Compressive Chest Trauma:**
  - Purtscher’s retinopathy

What is the classic DFE appearance in Purtscher’s retinopathy?

Multiple peripapillary CWS surrounding a relatively normal-appearing ONH (disc edema may be present). Additional areas of non-CWS retinal whitening are usually present, as are small intraretinal hemorrhages.

What is the classic clinical scenario?

An acute hx of compressive trauma to the chest (it can occur in the context of head trauma as well).

There are other 'classic clinical scenarios' that get mentioned in this context. What are they?

Pancreatitis, amniotic-fluid embolization, long-bone fracture (there are others). But to be clear, these do not cause Purtscher’s retinopathy.

What is the cause of the occlusion? **Leukoembolization**

What is the pathologic process underlying Purtscher’s?

- Complement activation
- Granulocyte aggregation
- Leukoembolization
- Occlusion of small retinal arterioles
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

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What is the classic clinical scenario?

An acute hx of compressive trauma to the chest (it can occur in the context of head trauma as well) are other scenarios that get mentioned in this context. What are they?

- Pancreatitis
- Amniotic-fluid embolization
- Long-bone fracture (there are others)

But to be clear, these do not cause Purtscher’s retinopathy.

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It’s true that these conditions can cause a retinopathy identical in appearance to Purtscher’s. That said, Dr Purtscher’s original description was in the context of thoracic or head trauma. Thus, technically speaking, the term Purtscher retinopathy is reserved for only situations in which the retinopathy results from thoracic/head trauma.

OK then, what is the name for the Purtscher’s-like retinopathy due to pancreatitis, amniotic-fluid embolization, long-bone fracture, etc?

It’s called ‘Purtscher’s-like retinopathy’.

What is the pathologic process underlying Purtscher’s?

Complement activation \(\rightarrow\) granulocyte aggregation \(\rightarrow\) leukoembolization \(\rightarrow\) occlusion of small retinal arterioles

What is the cause of the occlusion?

Leukoembolization

Aggregates of what sort of immune cells for the emboli?

Granulocytes

Let’s tackle this question in reverse. What is the direct, proximal cause of retinal hemorrhages in Purtscher’s?

Occlusion of small retinal arterioles
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The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

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- **Hx of compressive chest trauma:** Purtscher's retinopathy

What is the classic DFE appearance in Purtscher's retinopathy?

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What is the classic clinical scenario?

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What is the pathologic process underlying Purtscher’s?

[Diagram: Complement activation -> granulocyte aggregation -> leukoembolization -> occlusion of small retinal arterioles]

What is the cause of the occlusion?

Leukoembolization

Let’s tackle this question in reverse. What is the direct, proximal cause of retinal hemorrhages in Purtscher’s?

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Aggregates of what sort of immune cells for the emboli?

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What is the classic DFE appearance in Purtscher’s retinopathy?
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What is the classic DFE appearance in Purtscher's retinopathy?

- Multiple peripapillary CWS surrounding a relatively normal-appearing ONH (disc edema may be present). Additional areas of non-CWS retinal whitening are usually present, as are small intraretinal hemorrhages.

What is the classic clinical scenario?

- An acute hx of compressive trauma to the chest (it can occur in the context of head trauma as well). There are other 'classic clinical scenarios' that get mentioned in this context. What are they?
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OK then, what is the name for the Purtscher’s-like retinopathy due to pancreatitis, amniotic-fluid embolization, etc?

It’s called ‘Purtscher’s-like retinopathy’.

What is the pathologic process underlying Purtscher’s?

- Activation of the complement system
  - Granulocyte aggregation
  - Leukoembolization
  - Occlusion of small retinal arterioles

What is the cause of the occlusion?

- Leukoembolization

Aggregates of what sort of immune cells for the emboli?

- Granulocytes

And lastly: Activation of which aspect of the immune system begins the cascade?
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

- **Sphygmametry:** HTN
- **Echocardiogram:** Cardiac embolic source
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What is the classic DFE appearance in Purtscher's retinopathy? Multiple peripapillary CWS surrounding a relatively normal-appearing ONH (disc edema may be present). Additional areas of non-CWS retinal whitening are usually present, as are small intraretinal hemorrhages.

What is the classic clinical scenario? An acute hx of compressive trauma to the chest (it can occur in the context of head trauma as well). There are other 'classic clinical scenarios' that get mentioned in this context. What are they? Pancreatitis, amniotic-fluid embolization, long-bone fracture (there are others). But to be clear, these do not cause Purtscher's retinopathy.

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OK then, what is the name for the Purtscher’s-like retinopathy due to pancreatitis, amniotic-fluid embolization, etc? It’s called ‘Purtscher-like retinopathy.’

What is the pathologic process underlying Purtscher’s? Complement activation $\rightarrow$ granulocyte aggregation $\rightarrow$ leukoembolization $\rightarrow$ occlusion of small retinal arterioles.

Let’s tackle this question in reverse. What is the direct, proximal cause of retinal hemorrhages in Purtscher’s? Occlusion of small retinal arterioles.

What is the cause of the occlusion? Leukoembolization.

Aggregates of what sort of immune cells for the emboli? Granulocytes.

What is the classic clinical scenario? An acute hx of compressive trauma to the chest (it can occur in the context of head trauma as well).

What is the pathologic process underlying Purtscher’s? Complement activation $\rightarrow$ granulocyte aggregation $\rightarrow$ leukoembolization $\rightarrow$ occlusion of small retinal arterioles.

And lastly: Activation of which aspect of the immune system begins the cascade? The complement system.
The following tests constitute a reasonable battery in working up a cotton-wool spot in a nondiabetic patient. What disease is being ruled out with each?

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- **Hx of compressive chest trauma:** Purtscher’s retinopathy

Purtscher’s retinopathy is characterized by multiple peripapillary CWS surrounding a relatively normal-appearing ONH (disc edema may be present). Additional areas of retinal whitening are usually present, as are small intraretinal hemorrhages.

What is the classic clinical scenario? An acute hx of compressive trauma to the chest (it can occur in the context of head trauma as well)

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- Hx of compressive chest trauma: **Purtscher’s retinopathy**
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Circling back to ‘additional areas of retinal whitening.’ Isn’t this the same thing as CWS? No. This is referring to Purtscher flecken, polygonal-shaped areas of retinal whitening found in the peripapillary area and macula.

What is the classic DFE appearance in Purtscher’s retinopathy? Multiple peripapillary CWS surrounding a relatively normal-appearing ONH (disc edema may be present) are usually present, as are small intraretinal hemorrhages.

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**Additional areas of retinal whitening**

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**What is the mechanism by which Purtscher flecken form?**

**Additional areas of retinal whitening**

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What is the classic DFE appearance in Purtscher’s retinopathy?

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What is the mechanism by which Purtscher flecken form?
Vascular occlusion

Additional areas of retinal whitening

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Occlusion…that’s what causes CWS. Do Purtscher flecken and CWS differ in any meaningful way? Indeed they do.

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Occlusion…that’s what causes CWS. Do Purtscher flecken and CWS differ in any meaningful way?

Indeed they do. Cotton-wool spots occur when branches of the pre-capillary arteriolar network are occluded. These vessels are located in the superficial (ie, inner) portion of the retina; thus, the layer of the retina most affected by their occlusion is the nerve fiber layer. Obstruction of the RNFL causes axoplasmic stasis in the nerve fibers served by the obstructed vessel. Axoplasmic stasis renders the affected nerve fibers white—ie, a CWS. In contrast, Purtscher flecken develop when occlusion occurs at the capillary level of retinal circulation. These vessels are located deeper in the retina, and thus their occlusion doesn’t affect the retina nerve fiber layer—so no CWS.

What is the classic clinical scenario?

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**Additional areas of retinal whitening**

Multiple peripapillary CWS surrounding a relatively normal-appearing ONH (disc edema may be present) are usually present, as are small intraretinal hemorrhages.

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### What is the classic DFE appearance in Purtscher’s retinopathy?

A classic appearance is the multiple peripapillary CWS surrounding a relatively normal-appearing ONH (disc edema may be present). Additional areas of retinal whitening are usually present, as are small intraretinal hemorrhages.

### What is the classic clinical scenario?

An acute hx of compressive trauma to the chest (it can occur in the context of head trauma as well)

- ELISA: HIV
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### Circling back to ‘additional areas of retinal whitening.’ Isn’t this the same thing as CWS?

No. This is referring to **Purtscher flecken**, polygonal-shaped areas of retinal whitening found in the peripapillary area and macula.

### What is the mechanism by which Purtscher flecken form?

**Vascular occlusion**

**Occlusion…that’s what causes CWS. Do Purtscher flecken and CWS differ in any meaningful way?** Indeed they do. **Cotton-wool spots** occur when branches of the pre-capillary arteriolar network are occluded. These vessels are located in the superficial (ie, inner) portion of the retina; thus, the layer of the retina most affected by their occlusion is the nerve fiber layer. Obstruction of the RNFL causes axoplasmic stasis in the nerve fibers served by the obstructed vessel. Axoplasmic stasis renders the affected nerve fibers white—ie, a CWS. In contrast, **Purtscher flecken** develop when occlusion occurs at the capillary level of retinal circulation. These vessels are located deeper in the retina, and thus their occlusion doesn’t affect the retina nerve fiber layer--so no CWS.

### Additional areas of retinal whitening

Multiple peripapillary CWS surrounding a relatively normal-appearing ONH (disc edema may be present). Additional areas of retinal whitening develop in Purtscher’s retinopathy.
Circling back to ‘additional areas of retinal whitening.’ Isn’t this the same thing as CWS?
No. This is referring to Purtscher flecken, polygonal-shaped areas of retinal whitening found in the peripapillary area and macula.

What is the mechanism by which Purtscher flecken form?
Vascular occlusion

Cotton-wool spots

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How can Purtscher flecken and CWS be differentiated at DFE?
CWS have indistinct borders (like puffs of cotton wool--get it?), and obscure vessels running through them. In contrast, Purtscher flecken are more sharply demarcated and do not obscure adjacent vessels--in fact, a ‘clear zone’ appears between vessels and the surrounding flecken.

What is the mechanism by which Purtscher flecken form?
Vascular occlusion.

Occlusion...that’s what causes CWS. Indeed they do get occluded. These vessels are located in the deeper portion of the retina most affected by their occlusion is the nerve fiber layer. Occlusion of the RNFL causes axoplasmic stasis in the nerve fibers served by the obstructed vessel. Axoplasmic stasis renders the affected nerve fibers white—i.e., a CWS. In contrast, Purtscher flecken develop when occlusion occurs at the capillary level of retinal circulation. These vessels are located deeper in the retina, and thus their occlusion doesn’t affect the retina nerve fiber layer—so no CWS.

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**Which interferon formulation are we talking about here?**

Interferon alfa-2a

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**Do CWS present in isolation?**

No, they are often accompanied by retinal hemorrhages

**Is interferon retinopathy a common finding in interferon pts?**

Yes—estimates vary widely, but run no lower than about 20%

**How is it managed?**

If mild, by lowering the dose; if severe, by stopping the drug
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**Remember: Work-up even one CWS in a non-DM patient!**

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