In a nutshell, what is OIS?
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A constellation of signs and symptoms owing to chronic ocular...
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**Ocular Ischemic Syndrome**

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Blood flow in the ophthalmic artery and its branches. (A) Normal unobstructed flow.
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Ocular Ischemic Syndrome

Blood flow in the ophthalmic artery and its branches. (A) Normal unobstructed flow.
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Giant cell arteritis (GCA). Always bear GCA in mind when you evaluate an OIS pt!
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Very—at least 90%

In one word, what disease process is responsible for occluding the ICA in these pts?
Atherosclerosis

Atherosclerosis is an affliction of vasculopaths—is vasculopathy a risk factor for OIS?
Very much so
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High-grade stenosis of the internal carotid artery origin (arrow) in two pts
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With what nonocular atherosclerotic conditions is OIS associated?

How common are these conditions in OIS pts?

--CAD is present in half of OIS pts
--CVA has occurred previously in 25% of them
--PAD?
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Does it present unilaterally, or bilaterally?
Q/A

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Unilaterally (in about \% of cases)
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In a nutshell, what is OIS?
A constellation of signs and symptoms owing to chronic ocular hypoperfusion

Does it present unilaterally, or bilaterally?
Unilaterally (in about 80% of cases)

Is there a gender predilection?
Yes, men are twice as likely to have it

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What are the signs/symptoms of OIS?

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- Intraretinal hemorrhages
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- Retinal vascular changes

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**Size:** Medium to large

**Configuration (shape):** Dot/blot (DBH)

**Location:** The mid v periphery

Q/A

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*Ocular Ischemic Syndrome*
In a nutshell, what is OIS? A constellation of signs and symptoms owing to chronic ocular hypoperfusion.

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Intraretinal hemorrhages in OIS: Midperipheral, medium-large, dot-blot
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Note: Inconsistencies exist among the BCSC books regarding the location of retinal hemorrhages in OIS:
- Location per the Neuro book: “Midperipheral”
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**Location:** The mid-periphery?

No question—proceed at your own pace
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**Symptoms:**
- Decreased vision
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- Prolonged photostress recovery time

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FWIW, *EyeWiki* indicates they’re mid-peripheral. I was ‘raised’ to believe they’re mid-peripheral myself, so that’s how I roll. Caveat emptor.

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*There is another classic configuration for intraretinal hemorrhages, one not expected in OIS. What is it?*

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There is another classic configuration for intraretinal hemorrhages, one not expected in OIS. What is it?
Elongated and streaky
In a nutshell, what is OIS?
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*There is another classic configuration for intraretinal hemorrhages, one not expected in OIS. What is it? Elongated and streaky*

**What descriptive term is used to label such hemorrhages?**
They are known as ‘flame hemorrhages’ (FH).

Unilaterally (in about 80% of cases)

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Ocular Ischemic Syndrome

Flame vs DB hemorrhages (and a CWS for lulz)
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Are CWS expected in OIS?
Flame vs DB hemorrhages (and a CWS for lulz)

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Why causes some intraretinal hemorrhages to be DBHs and others to be FHs?
In a nutshell, what is OIS?
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It’s a function of the two words in which the blood is located
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*Why causes some intraretinal hemorrhages to be DBHs and others to be FHs?* It’s a function of the retinal layer(s) in which the blood is located
In a nutshell, what is OIS?
A constellation of signs and symptoms owing to chronic ocular hypoperfusion.

**Ocular Ischemic Syndrome**

What are the signs/symptoms of OIS?

**Signs:**
--Intraretinal hemorrhages
--NVI/NVA
--AC cell/flare
--Retinal vascular changes

**Symptoms:**
--Decreased vision
--Pain
--Prolonged photostress recovery time

Intraretinal hemorrhages in OIS don’t present rando, rather, there’s a classic appearance they tend to display. What is it?
The hemorrhages typically have a particular...

**Dot/blot (DBH)**

Size: Medium to large

Configuration (shape): Dot/blot (DBH)

Location: The mid-periphery

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Why causes some intraretinal hemorrhages to be DBHs and others to be FHs?
It’s a function of the retinal layer(s) in which the blood is located. FH are in the nerve fiber layer (NFL), which fibers are running parallel to the retina’s surface toward the ONH. Heme in the NFL tends to settle between fibers, giving NFL hemorrhages their elongated and streaky appearance. In contrast, the heme responsible for DBH is in the middle retinal layers, where most fibers are running perpendicular to the retinal surface. Because there is no impetus for the heme to spread with a particular orientation, it just blobs out in a circle around its source.
Ocular Ischemic Syndrome

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Does it present unilaterally, or bilaterally?

Unilaterally (in about 80% of cases)

Is there a gender predilection?

Yes, men are twice as likely to have it

Is there a relationship with age?

Yes, OIS is a disease of older individuals—average age is about 65; and it’s rare before 55

How common is it?

Estimates vary, but fair to say it’s an uncommon condition—vastly less common than diabetic retinopathy and/or CRVO, certainly

What is the long-term visual prognosis for eyes with OIS?

This is uncertain, but it is often poor. One sign in particular portends poor vision (this sign will be identified shortly).

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Indeed it does—the 5-year mortality rate of OIS is 40%!
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**Note:** NVI is the sign referred to earlier as portending a poor visual prognosis. 90% of eyes with NVI secondary to OIS will have VA of 20/200 or worse within a year of diagnosis!
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When you hear ‘NVI/NVA,’ one condition should come to mind first—what is it?

Diabetes. Diabetic retinopathy is by far the most common cause of NVI/NVA.
Other than DBR, what should come to mind before OIS when contemplating NVI/NVA? (Remember, OIS is an uncommon condition.)
CRVO is most definitely next on the list (the list after that is more difficult to order, with entities like uveitis, tumors, CRAO, sickle-cell, etc).

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Does it present unilaterally, or bilaterally?
Unilaterally (in about 80% of cases)

Is there a gender predilection?
Yes, men are twice as likely to have it

Is there a relationship with age?
Yes, OIS is a disease of older individuals—average age is about 65; and it's rare before 55

How common is it?
Estimates vary, but fair to say it's an uncommon condition—vastly less common than diabetic retinopathy and/or CRVO, certainly

What is the long-term visual prognosis for eyes with OIS?
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**NVI/NVA often leads to what dreaded ocular condition?**
Neovascular glaucoma (NVG)

**How does NVA lead to NVG?**
The NVA vessels don't ride solo; rather, they are accompanied by contractile elements (e.g., fibroblasts). Along with the neo vessels, these elements cross from the peripheral iris to the peripheral cornea. Once established, contractile elements gonna contract, and when they do, they pull the iris up against the angle, closing it.

**What one word describes the underlying cause of most cases of NVG?**
'Ischemia'

**How does ischemia lead to NVI and NVA?**
In a desperate attempt to acquire the oxygen they're lacking, ischemic cells release the signaling molecule VEGF, a potent inducer of new blood vessel formation. VEGF induces the NVI/NVA process.
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Closed angle in NVG
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Yes, men are twice as likely to have it

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Yes, OIS is a disease of older individuals—average age is about 65; and it's rare before 55

How common is it?
Estimates vary, but fair to say it's an uncommon condition—vastly less common than diabetic retinopathy and/or CRVO, certainly

What is the long-term visual prognosis for eyes with OIS?
This is uncertain, but it is often poor. One sign in particular portends poor vision (this sign will be identified shortly).

Does OIS carry implications for the general health of the afflicted individual?
Indeed it does—the 5-year mortality rate of OIS is 40%!

**Signs:**
- Intraretinal hemorrhages
- NVI/NVA
- AC cell/flare
- Retinal vascular changes

**Symptoms:**
- Decreased vision
- Pain
- Prolonged photostress recovery time

What are the signs/symptoms of OIS?

VNI/VNA often leads to what dreaded ocular condition?
Neovascular glaucoma (NVG)

How does NVI lead to NVG?
The NVA vessels don't ride solo; rather, they are accompanied by contractile elements (eg, fibroblasts). Along with the neo vessels, these elements cross from the peripheral iris to the peripheral cornea. Once established, contractile elements gonna contract, and when they do, they pull the iris up against the angle, closing it.

What one word describes the underlying cause of most cases of NVG?
'Ischemia'

How does ischemia lead to NVI and NVA?
In a desperate attempt to acquire the oxygen they're lacking, ischemic cells release the signaling molecule VEGF, a potent inducer of new blood vessel formation. VEGF induces the NVI/NVA process.

Given this description, into what general class of glaucoma does NVG fall?
It is a form of secondary angle-closure glaucoma (and an important one at that).
In a nutshell, what is OIS?
A constellation of signs and symptoms owing to chronic ocular hypoperfusion

Does it present unilaterally, or bilaterally?
Unilaterally (in about 80% of cases)

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**What do NVI and NVA stand for in this context?**
Neovascularization of the iris (NVI) and the angle (NVA)

**Are NVI/NVA common in OIS?**
Yes—roughly 2/3 of pts will manifest one or both

**When you hear 'NVI/NVA,' one condition should come to mind first—what is it?**
Diabetes. Diabetic retinopathy is by far the most common cause of NVI/NVA

**Other than DBR, what should come to mind before OIS when contemplating NVI/NVA? (Remember, OIS is an uncommon condition.)**
CRVO is mos def next on the list. The list after that is more difficult to order, with entities like uveitis, tumors, CRAO, sickle-cell, etc.

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We divide the 2ndry angle-closure glaucomas into two groups—what are they?

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With pupillary block

Without pupillary block

We divide the secondary angle-closure glaucomas into two groups—what are they?

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We divide the 2ndry angle-closure glaucomas into two groups—what are they? 

With pupillary block 

--- NVG? 

Without pupillary block 

--- NVG?

To which group does NVG belong?

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**Ocular Ischemic Syndrome**

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**Symptoms:**

- Decreased vision
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**NVI/NVA often leads to what dreaded ocular condition?**

Neovascular glaucoma (NVG)

**How does NVA lead to NVG?**

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**With pupillary block**

**Without pupillary block**

**NVG!**

We divide the 2ndry angle-closure glaucomas into two groups—what are they?

To which group does NVG belong? Without pupillary block

**Ischemia**

How does ischemia lead to NVI and NVA?

In a desperate attempt to acquire the oxygen they’re lacking, ischemic cells release the signaling molecule VEGF, a potent inducer of new blood vessel formation. VEGF induces the NVI/NVA process.

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**For (lots) more on secondary angle-closure glaucoma, see slide-set G16**
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**Symptoms:**
--Decreased vision--Pain--Prolonged photostress recovery time

What do NVI and NVA stand for in this context?
Neovascularization of the iris (NVI) and the angle (NVA)

Are NVI/NVA common in OIS?
Yes—roughly 2/3 of pts will manifest one or both

When you hear ‘NVI/NVA,’ one condition should come to mind first—what is it?
Diabetes. Diabetic retinopathy is by far the most common cause of NVI/NVA

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Yes, OIS is a disease of older individuals—average age is about 65; and it’s rare before 55

How common is it?
Estimates vary, but fair to say it’s an uncommon condition—vastly less common than diabetic retinopathy and/or CRVO, certainly

What is the long-term visual prognosis for eyes with OIS?
This is uncertain, but it is often poor. One sign in particular portends poor vision (this sign will be identified shortly).

Does OIS carry implications for the general health of the afflicted individual?
Indeed it does—the 5-year mortality rate of OIS is 40%!

**Ocular Ischemic Syndrome**

**Signs:**
-- Intra-raretinal hemorrhages
-- NVI/NVA
-- AC cell/flare
-- Retinal vascular changes

**Symptoms:**
-- Decreased vision
-- Pain
-- Prolonged photostress recovery time

What do NVI and NVA stand for in this context?
Neovascularization of the iris (NVI) and the angle (NVA)

Are NVI/NVA common in OIS?
Yes—roughly 2/3 of pts will manifest one or both

When you hear ‘NVI/NVA,’ one condition should come to mind first—what is it?
Diabetes. Diabetic retinopathy is by far the most common cause of NVI/NVA

Other than DBR, what should come to mind before OIS when contemplating NVI/NVA? (Remember, OIS is an uncommon condition.)
CRVO is mos def next on the list. The list after that is more difficult to order, with entities like uveitis, tumors, CRAO, sickle-cell, etc.

What is NVI/NVA often leads to what dreaded ocular condition?
Neovascular glaucoma (NVG)

How does NVA lead to NVG?
The NVA vessels don’t ride solo; rather, they are accompanied by contractile elements (eg, fibroblasts). Along with the neo vessels, these elements cross from the peripheral iris to the peripheral cornea. Once established, contractile elements gonna contract, and when they do, they pull the iris up against the angle, closing it.

What one word describes the underlying cause of most cases of NVG?
‘Ischemia’

How does ischemia lead to NVI and NVA?
In a desperate attempt to acquire the oxygen they’re lacking, ischemic cells release the signaling molecule VEGF, a potent inducer of new blood vessel formation. VEGF induces the NVI/NVA process.

Does OIS carry implications for the general health of the afflicted individual?
Indeed it does—the 5-year mortality rate of OIS is 40%!
In a nutshell, what is OIS?
A constellation of signs and symptoms owing to chronic ocular hypoperfusion.

Does it present unilaterally, or bilaterally?
Unilaterally (in about 80% of cases).

Is there a gender predilection?
Yes, men are twice as likely to have it.

Is there a relationship with age?
Yes, OIS is a disease of older individuals—average age is about 65; and it’s rare before 55.

How common is it?
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How does ischemia lead to NVI and NVA?
In a desperate attempt to acquire the oxygen they’re lacking, ischemic cells release the signaling molecule—VEGF, a potent inducer of new blood vessel formation. VEGF induces the NVI/NVA process.

Other than DBR, what disease induces the NVI/NVA process.
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Why doesn’t NVI/NVA in OIS consistently lead to NVG?
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Q

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VEGF induces the NVI/NVA process.

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**Ocular Ischemic Syndrome**

**Q**

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What one word describes the underlying cause of most cases of NVG?
'Ischemia'

How does ischemia lead to NVI and NVA?
In a desperate attempt to acquire the oxygen they’re lacking, ischemic cells release the signaling molecule VEGF, a potent inducer of new blood vessel formation. VEGF induces the NVI/NVA process.

So, if most OIS pts get NVI/NVA, and NVA leads to NVG, it follows that most OIS pts must get NVG, yes? You’d think so, but no. While a few OIS pts will have elevated IOP, most will not, instead presenting with normal or even low IOP.

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What specific component of the ciliary body creates aqueous?
The nonpigmented epithelial layer of the pars plicata

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What specific component of the ciliary body creates aqueous?
The distinguishing attribute is the nonpigmented epithelial layer of the pars plicata.

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**Signs:**
- Intraretinal hemorrhages
- NVI/NVA
- AC cell/flare
- Retinal vascular changes

**Symptoms:**
- Decreased vision
- Pain
- Prolonged photostress recovery time

**Neovascular glaucoma (NVG)**

If an eye has a zipped-up angle secondary to NVA from OIS, what can happen to IOP if/when blood flow to the ciliary body is re-established—say, by successful CEA for an occluded ICA?

So, if most OIS pts get NVI/NVA, and NVA leads to NVG, it follows that most OIS pts must get NVG, yes?

You'd think so, but no. While a few OIS pts will have elevated IOP, most will not, instead presenting with normal or even low IOP.

**Low IOP??!! How is that possible?**
In a word—hypoperfusion. The same lack of blood flow that resulted in ocular ischemia leads to shutdown of the ciliary body. This result in a dramatic reduction in the rate of aqueous-humor formation, which in turn precludes the development of high IOP.

**What specific component of the ciliary body creates aqueous?**
The nonpigmented epithelial layer of the pars plicata

CRVO is mos def next on the list. The list after that is more difficult to order, with entities like uveitis, tumors, CRAO, sickle-cell, etc.

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In a nutshell, what is OIS?
A constellation of signs and symptoms owing to chronic ocular hypoperfusion

Does it present unilaterally, or bilaterally?
Unilaterally (in about 80% of cases)

Is there a gender predilection?
Yes, men are twice as likely to have it

Is there a relationship with age?
Yes, OIS is a dz of older individuals—average age is about 65; and it's rare before 55

How common is it?
Estimates vary, but fair to say it's an uncommon condition—vastly less common than diabetic retinopathy and/or CRVO, certainly

What is the long-term visual prognosis for eyes with OIS?
This is uncertain, but it is often poor. One sign in particular portends poor vision (this sign will be identified shortly).

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What do NVI and NVA stand for in this context?
Neovascularization of the iris (NVI) and the angle (NVA)

Are NVI/NVA common in OIS?
Yes—roughly 2/3 of pts will manifest one or both

When you hear 'NVI/NVA,' one condition should come to mind first—what is it?
Diabetes. Diabetic retinopathy is by far the most common cause of NVI/NVA

Other than DBR, what should come to mind before OIS when contemplating NVI/NVA? (Remember, OIS is an uncommon condition.)
CRVO is mos def next on the list. The list after that is more difficult to order, with entities like uveitis, tumors, CRAO, sickle-cell, etc.

NVI/NVA often leads to what dreaded ocular condition?
Neovascular glaucoma (NVG)

If an eye has a zipped-up angle secondary to NVA from OIS, what can happen to IOP if/when blood flow to the ciliary body is re-established—say, by successful CEA for an occluded ICA?
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In such cases, IOP can spike precipitously when the CB ‘wakes up’ and resumes producing aqueous at a normal rate. If this occurs, the pt may be thrown into NVG severe enough to threaten vision or even the eye. Because of this possibility, it is vital that you 1) are looped in on plans to operate on your OIS pt, and 2) have a plan in place to intervene acutely if the above scenario comes to pass!

Low IOP??!! How is that possible? Just as IOP was low before NVA presentation due to ocular hypoperfusion, it will remain low in the setting of neovascularization of the angle (NVA) due to shutdown of the ciliary body. This results in a dramatic reduction in the rate of aqueous-humor formation, which in turn precludes the development of high IOP.

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Neovascular glaucoma (NVG)

NVI/NVA often leads to what dreaded ocular condition?

How does NVI/NVA lead to NVG?

The NVA vessels don't ride solo; rather, they are accompanied by contractile elements (eg, fibroblasts). Along with the neo vessels, these elements cross from the peripheral iris to the peripheral cornea. Once established, contractile elements gonna contract, and when they do, they pull the iris up against the angle, closing it.

What one word describes the underlying cause of most cases of NVG?

'Ischemia'

How does ischemia lead to NVI and NVA?

In a desperate attempt to acquire the oxygen they're lacking, ischemic cells release the signaling molecule VEGF, a potent inducer of new blood vessel formation. VEGF induces the NVI/NVA process.

So, if most OIS pts get NVI/NVA, and NVA leads to NVG, it follows that most OIS pts must get NVG, yes?

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How should NVI/NVA in OIS be managed?

PRP successfully induces regression of anterior-segment neo in a majority of cases. Intravitreal anti-VEGF tx can be considered as well.

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Not terrible—certainly nowhere near the 'hypopyon' range, say. Of note, the classic presentation will have flare that is out of proportion to the cell.

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‘Dilated, not tortuous’

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Ocular Ischemic Syndrome

OIS: Arteriolar narrowing; venous dilation without tortuosity
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Here we encounter another inconsistency in the BCSC books, regarding the appearance of the retinal venules in OIS:
--Appearance per the Neuro book: “dilated (nontortuous)”
--Per the Retina book: “dilated but not very tortuous”
--Per the Uveitis book: “dilated [and] tortuous”

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No question—proceed
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(Other than being able to answer OKAP/WQE/Board questions correctly, that is.)
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Because the DDx for an OIS-like fundus includes mild CRVO, and in CRVO the venules are always tortuous (as well as dilated). Thus, if non-dilated venules are a feature of OIS, their presence on DFE would point strongly toward OIS and away from CRVO.

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Ocular Ischemic Syndrome
In a nutshell, what is OIS? A constellation of signs and symptoms owing to chronic ocular hypoperfusion. Does it present unilaterally, or bilaterally? Unilaterally (in about 80% of cases). Is there a gender predilection? Yes, men are twice as likely to have it. Is there a relationship with age? Yes, OIS is a disease of older individuals—average age is about 65; and it's rare before 55. How common is it? Estimates vary, but fair to say it's an uncommon condition—vastly less common than diabetic retinopathy and/or CRVO, certainly. What is the long-term visual prognosis for eyes with OIS? This is uncertain, but it is often poor. One sign in particular portends poor vision (this sign will be identified shortly). Does OIS carry implications for the general health of the afflicted individual? Indeed it does—the 5-year mortality rate of OIS is 40%!

**Ocular Ischemic Syndrome**

**What are the signs/symptoms of OIS?**

**Signs:**
- Intraretinal hemorrhages
- NVI/NVA
- AC cell/flare
- Retinal vascular changes

**Symptoms:**
- Decreased vision
- Pain
- Prolonged photostress recovery time

**What is the classic description of the retinal arterioles in OIS?** ‘Narrowed’

**How about the venules?** ‘Dilated, not tortuous’

**Why does the appearance of the venules—specifically, whether they are tortuous—matter? (Other than being able to answer OKAP/WQE/Board questions correctly, that is.)** Because the DDx for an OIS-like fundus includes mild CRVO, and in CRVO the venules are always tortuous (as well as dilated). Thus, if non-dilated venules are a feature of OIS, their presence on DFE would point strongly toward OIS and away from CRVO. FWIW, *EyeWiki* states the venules are “dilated but not tortuous.” This is my understanding as well. Caveat emptor.
In a nutshell, what is OIS?
A constellation of signs and symptoms owing to chronic ocular hypoperfusion

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**Signs:**

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**What is the classic description of the retinal arterioles in OIS?**
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**How about the venules?**
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Here we encounter another inconsistency among the BCSC books, regarding the appearance of the retinal venules in OIS:

--Appearance per the Neuro book: “dilated (nontortuous)”
--Per the Retina book: “dilated but not very tortuous”
--Per the Uveitis book: “dilated [and] tortuous”

Why does the appearance of the venules—specifically, whether they are tortuous—matter?
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Head’s up: We will have much more to say about differentiating between OIS and CRVO later in the slide-set

Ocular Ischemic Syndrome
In a nutshell, what is OIS?
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Hints forthcoming

What are the signs/symptoms of OIS?

Three more signs come up enough that we should at least mention them here:

---Decreased vision
---Pain
---Prolonged photostress recovery time

How common is it?
Estimates vary, but fair to say it’s an uncommon condition. It’s vastly less common than diabetic retinopathy and also CRVO.

What is the long-term visual prognosis for eyes with OIS?
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One media issue dwarfs the others in terms of how frequently it’s implicated in TMVL—what is it?

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The Neuro book emphasizes three optic-nerve causes of TMVL—which ones?

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**Vascular issues**
- ?
- ?
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**What is Uhthoff’s phenomenon?**
Uhthoff’s phenomenon is TMVL in optic neuritis secondary to increased body temp.

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TMVL in condition 2ndry to three words

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- Disc edema
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Vascular issues
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- GCA
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How do OIS pts describe their pain; eg, is it a foreign-body sensation?
No, it is a dull, aching pain that locates to the eye or orbit. Pts often report that the pain eases when they do something specific—what?
When they lie down. This is a classic finding in OIS—take note of it!

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Does OIS carry implications for the general health of the afflicted individual?
Indeed it does—the 5-year mortality rate of OIS is 40%!
In a nutshell, what is OIS?
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What are the signs/symptoms of OIS?

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--Intraretinal hemorrhages
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--AC cell/flare
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**Symptoms:**
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Is there a gender predilection?
Yes, men are twice as likely to have it.

Is there a relationship with age?
Yes, OIS is a disease of older individuals—average age is about 65; and it’s rare before 55.

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In acute ACG, the entire angle becomes occluded over a short period of time, producing a precipitous rise in IOP. The extremely high IOP causes severe eye pain and HA, N/V, and blurry vision. The event will not resolve without intervention.

In subacute ACG, some portion of the angle occludes episodically, resulting in periods of moderate (not extreme) IOP elevation. This IOP causes moderate eye pain and HA, and blurry vision. The episodes resolve spontaneously, often after sleep. IOP is normal between episodes, which can make diagnosis challenging.
In a nutshell, what is OIS?
A constellation of signs and symptoms owing to chronic ocular hypoperfusion.

Does it present unilaterally, or bilaterally?
Unilaterally (in about 80% of cases).

Is there a gender predilection?
Yes, men are twice as likely to have it.

Is there a relationship with age?
Yes, OIS is a disease of older individuals—average age is about 65; and it’s rare before 55.

How common is it?
Estimates vary, but fair to say it’s an uncommon condition—vastly less common than diabetic retinopathy and/or CRVO, certainly.

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**Signs:**
--Intraretinal hemorrhages
--NV/NVA--AC cell/flare
--Retinal vascular changes

**Symptoms:**
--Decreased vision
--Pain
--Prolonged photostress recovery time

How do OIS pts describe their pain; eg, is it a foreign-body sensation?
No, it is a dull, aching pain that locates to the eye or orbit.

Pts often report that the pain eases when they do something specific—what?
When they lie down. This is a classic finding in OIS—take note of it!

**Ocular Ischemic Syndrome**
When you hear 'periocular pain that improves with lying down/sleep,' three conditions should come to mind. What are the other two?
--OIS--
--Subacute angle-closure glaucoma
--Migraine

In two words what is the pathologic mechanism underlying subacute angle-closure glaucoma?
Pupillary block

Circling back to the original point: What happens during sleep that provides pain relief in subacute angle-closure glaucoma?
Sleep-induced miosis breaks the pupillary block, thus allowing aqueous outflow to resume (and IOP to drop).

Are you sure about this? I thought pupillary block was the mechanism underlying acute angle-closure glaucoma.
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What is 'photostress recovery time'?
It refers to the length of time it takes for vision to recover after the retina has been subjected to a very bright light (OIS pts will complain of being 'blind for a long time' in the affected eye after exposure to bright light).

Why is recovery time prolonged in OIS?
Because the ischemic retinal circulation is unable to meet the high metabolic demand created by the photostress in a timely manner. Think of it as retinal claudication, with the prolonged visual recovery time being analogous to the calf pain after walking in PAD, or the jaw claudication induced by chewing in GCA.

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Because the ischemic retinal circulation is unable to meet the high metabolic demand created by the photostress in a timely manner. Think of it as retinal claudication, with prolonged visual recovery time being analogous to the calf pain PAD pts get when they walk, and the jaw pain GCA pts get when they chew.

Can photostress recovery time be formally assessed in the clinic?
It can indeed, via the photostress recovery test. The test is performed unilaterally. The BCVA for the eye is determined (reliable results require that VA be 20/80 or better). An extremely bright light is shone directly into the eye from a distance of about 1 inch for 10 seconds. The pt is then asked to read a Snellen line one row worse than their BCVA, and the amount of time it takes for them to be able to do this is recorded. A normal photostress time would be 30-s or less; pts with OIS will take significantly longer, usually at least 90-s.
In a nutshell, what is OIS? A constellation of signs and symptoms owing to chronic ocular hypoperfusion.

What are the signs/symptoms of OIS?

**Signs:**
- Intraretinal hemorrhages
- NVI/NVA
- AC cell/flare
- Retinal vascular changes

**Symptoms:**
- Decreased vision
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- Prolonged photostress recovery time

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**Does OIS carry implications for the general health of the afflicted individual?** Indeed it does—the 5-year mortality rate of OIS is 40%!
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How would a pt with VA loss 2ndry to an optic neuropathy perform on the test?
Her result would be normal—which makes this test very useful in determining whether a pt with VA loss has macular/vascular dz vs an optic neuropathy.

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Does it present unilaterally, or bilaterally?
Unilaterally (in about 80% of cases)

Is there a gender predilection?
Yes, men are twice as likely to have it

Is there a relationship with age?
Yes, OIS is a disease of older individuals—average age is about 65; and it's rare before 55

How common is it?
Estimates vary, but fair to say it's an uncommon condition—vastly less common than diabetic retinopathy and/or CRVO, certainly

What is the long-term visual prognosis for eyes with OIS?
This is uncertain, but it is often poor. One sign in particular portends poor vision (this sign will be identified shortly).

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DDx for an OIS-like fundus

Q: Ocular Ischemic Syndrome

?  OIS  ?

?
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Ocular Ischemic Syndrome

DDx for an OIS-like fundus

- Hyperviscosity syndrome
- OIS
- CRVO
The Retina book mentions three causes of hyperviscosity syndrome—what are they?

DDx for an OIS-like fundus

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Ocular Ischemic Syndrome
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DDx for an OIS-like fundus

Hyperviscosity syndrome
- Waldenström macroglobulinemia
- Multiple myeloma
- Polycythemia vera

OIS
CRVO
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**DDx for an OIS-like fundus**

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If hyperviscosity syndrome is suspected, what tests should be ordered?
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If hyperviscosity syndrome is suspected, what tests should be ordered?
--CBC
--Serum electrophoresis
--Measurement of whole-blood viscosity
What is the mechanism underlying CRVO?

CRVO

DDx for an OIS-like fundus

Ocular Ischemic Syndrome

What is the mechanism underlying CRVO?

Hyperviscosity syndrome OIS-like fundus OIS

Where does thrombosis typically occur?

At the lamina cribrosa, or just posterior to it

Do CRVO pts tend to be vasculopaths?

Yes—HTN is second only to age as a risk factor for CRVO

What role does vasculopathy play in the genesis of a CRVO?

Vasculopathy contributes to the development of atherosclerotic dz, and it's atherosclerotic changes to retinal arterial vessels that cause them to impinge upon and compress adjacent venous vessels. Impingement impedes blood flow through the venous vessel, as well as damages its endothelial cells. The combination of endothelial damage and impeded blood flow initiates the clotting cascade, with the result being formation of a thrombus.

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DDx for an OIS-like fundus

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CRVO: Dilated and tortuous veins; RNFL hemorrhages
Ocular Ischemic Syndrome

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What does ophthalmodynamometry measure?
Perfusion pressure of the retinal arterial tree

How does ophthalmodynamometry differentiate between OIS and CRVO?
Perfusion pressure will be low in OIS but normal in CRVO

My ophthalmodynamometer is in the shop. Is there a way to check perfusion pressure without it?
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Ocular Ischemic Syndrome

OIS and a mild CRVO can be difficult to differentiate from one another.
Ocular Ischemic Syndrome

OIS and a mild CRVO can be difficult to differentiate from one another. For each statement, indicate whether it best applies to OIS, CRVO, or Both.
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- Retinal hemorrhages present:
For each statement, indicate whether it best applies to **OIS, CRVO, or Both**.

- Retinal hemorrhages present: **Both**
Ocular Ischemic Syndrome

For each statement, indicate whether it best applies to OIS, CRVO, or Both.

- Retinal hemorrhages present: Both
- c/o periorbital ache:
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Ocular Ischemic Syndrome

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- Retinal hemorrhages present: Both
- c/o periorbital ache: OIS
Ocular Ischemic Syndrome

For each statement, indicate whether it best applies to OIS, CRVO, or Both.

- Retinal hemorrhages present: Both
- c/o periorbital ache: OIS
- Retinal veins dilated:
For each statement, indicate whether it best applies to *OIS*, *CRVO*, or *Both*.

- Retinal hemorrhages present: *Both*
- c/o periorbital ache: *OIS*
- Retinal veins dilated: *Both*
For each statement, indicate whether it best applies to **OIS**, **CRVO**, or **Both**.

- Retinal hemorrhages present: **Both**
- c/o periorbital ache: **OIS**
- Retinal veins dilated: **Both**
- Hemorrhages confined to mid-periphery:
For each statement, indicate whether it best applies to OIS, CRVO, or Both.

- Retinal hemorrhages present: Both
- c/o periorbital ache: OIS
- Retinal veins dilated: Both
- Hemorrhages confined to mid-periphery: OIS
For each statement, indicate whether it best applies to OIS, CRVO, or Both.

- Retinal hemorrhages present: Both
- c/o periorbital ache: OIS
- Retinal veins dilated: Both
- Hemorrhages confined to mid-periphery: OIS
- Ophthalmodynamometry normal:
Ocular Ischemic Syndrome

For each statement, indicate whether it best applies to *OIS*, *CRVO*, or *Both*.

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- Retinal veins dilated: *Both*
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- Ophthalmodynamometry normal: *CRVO*
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- Slow vision loss: OIS
For each statement, indicate whether it best applies to \textit{OIS}, \textit{CRVO}, or \textit{Both}.

- Retinal hemorrhages present: \textit{Both}
- c/o periorbital ache: \textit{OIS}
- Retinal veins dilated: \textit{Both}
- Hemorrhages confined to mid-periphery: \textit{OIS}
- Ophthalmodynamometry normal: \textit{CRVO}
- Slow vision loss: \textit{OIS}
- Retinal veins tortuous:

\textbf{Ocular Ischemic Syndrome}
For each statement, indicate whether it best applies to **OIS**, **CRVO**, or **Both**.

- Retinal hemorrhages present: **Both**
- c/o periorbital ache: **OIS**
- Retinal veins dilated: **Both**
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- Cell and flare present:
For each statement, indicate whether it best applies to **OIS**, **CRVO**, or **Both**.

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- Slow vision loss: OIS
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- Cell and flare present: OIS
- At risk for rubeosis iridis:
For each statement, indicate whether it best applies to **OIS**, **CRVO**, or **Both**.

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- Retinal veins dilated: **Both**
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- Ophthalmodynamometry normal: **CRVO**
- Slow vision loss: **OIS**
- Retinal veins tortuous: **CRVO**
- Cell and flare present: **OIS**
- At risk for rubeosis iridis: **Both**
What is the landmark clinical trial dictating management of carotid occlusive disease as is so often associated with OIS?

The Carotid Endarterectomy (CEA) Study.

What forms of management were compared in the CEA Study?

It looked at CEA vs antiplatelet therapy for carotid occlusive disease in symptomatic patients.

How was symptomatic defined?

Patients had a history of TIA, amaurosis fugax, or nondisabling CVA.

What was the major finding of the CEA Study?

The major finding was that treatment risk/benefit ratio was a function of the extent of carotid blockage. Specific recommendations were as follows:

- If blockage was 70-99%: risk of CVA 9% in CEA group, 26% in antiplatelet group; the benefit outweighed the risk, and these patients should be offered CEA.
- If blockage was 50-69%: risk of CVA is 16% in CEA, 22% in antiplatelet group; the risk outweighed the benefit, and these patients should not be offered CEA.

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It is not uncommon for pts with OIS to have 100% blockage of their ipsilateral carotid artery. Note that 100% blockage of the carotids is a contraindication to CEA, as it is ineffective in these cases.
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