CRVO

*Is central retinal vein occlusion (CRVO) an embolic condition?*
Is central retinal vein occlusion (CRVO) an embolic condition?
No! (How on earth would an embolism reach the CRV?)
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OK then, what is the mechanism underlying CRVO?
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Thrombosis of the CRV, usually at the level of the lamina cribrosoa or just posterior to it
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Do CRVO pts tend to be vasculopaths?
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Regarding CRVO risk factors--may I introduce ‘the Hs.’
You know two already; how about the other three?
--Hypertension
--Hyperglycemia (ie, DM)
--H
--H
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Regarding CRVO risk factors--may I introduce ‘the Hs.’ You know two already; how about the other three?

---Hypertension
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---Hyperlipidemia
---High IOP (ie, OAG)
---Hypercoaguability
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--Hypertension  
--Hyperglycemia (ie, DM)  
--Hyperlipidemia  
--High IOP (ie, OAG)  
--Hypercoaguability

What implication does this have for evaluating a CRVO pt?  
It implies that, in addition to determining the glaucoma status of the CRVO eye, you need to consider whether the fellow eye has glaucoma
Is central retinal vein occlusion (CRVO) an embolic condition?
No! (How on earth would an embolism reach the CRV?)

OK then, what is the mechanism underlying CRVO?
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What is the strongest risk factor for CRVO? (It’s not listed on this page.)
Is central retinal vein occlusion (CRVO) an embolic condition?
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Thrombosis of the CRV, usually at the level of the lamina cribrosa or just posterior to it

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Regarding CRVO risk factors--may I introduce ‘the Hs.’
You know two already; how about the other three?
--Hyperglycemia (ie, DM)
--Hyperlipidemia
--Hypercoagulability

What is the strongest risk factor for CRVO? (It’s not listed on this page.)
Age. Over 90% of CRVO pts are older than
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-- Hyperglycemia (ie, DM)
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What is the strongest risk factor for CRVO? (It’s not listed on this page.)
Age. Over 90% of CRVO pts are older than 50.
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What role does vasculopathy play in the genesis of a CRVO?
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Do CRVO pts tend to be vasculopathes?
Yes. DM and HTN are both risk factors for CRVO.

What role does vasculopathy play in the genesis of a CRVO?
It’s believed that atherosclerotic disease of the central retinal artery causes it (the CRA) to impinge upon/partially compress the adjacent CRV. This compression disrupts blood flow through the CRV as well as damages its endothelial cells, thereby increasing the possibility of stasis and clot formation, with the resulting development of a thrombus.
CRVO

(Not ischemic vs nonischemic—we’ll get to that shortly)
CRVO

Primary/idiopathic

Secondary
CRVO

Primary/idiopathic

Secondary

- Syndrome with multiple causes
- Specific group of diseases
- Broad category
- Specific disease
CRVO

- Primary/idiopathic
- Secondary
  - Hyperviscosity syndrome
  - Vasculitis
  - Drugs
  - Carotid occlusive dz
CRVO

Primary/idiopathic

Secondary

Hyperviscosity syndrome

Vasculitis

Drugs

Carotid occlusive dz

(There are many others)
CRVO

Primary/idiopathic

Secondary

Hyperviscosity syndrome

- Waldenström macroglobulinemia
- Multiple myeloma
- Polycythemia vera
- Hypercoagulable states

Vasculitis

Drugs

Carotid occlusive dz
What specific conditions may contribute to hypercoaguability?

---

---
What specific conditions may contribute to hypercoaguability?
- Hyperhomocystinemia (yet another ‘H’)
- Protein S deficiency
- Protein C deficiency
Hypercoagulable states

CRVO

Primary/idiopathic

CRVO

Secondary

Hyperviscosity syndrome

Vasculitis

Drugs

Carotid occlusive dz

Pearl: If a pt presents with bilateral CRVOs, consider hyperviscosity first. (Check electrophoresis, viscosity studies)
CRVO

- Primary/idiopathic

CRVO

Secondary

- Hyperviscosity syndrome
  - Waldenström macroglobulinemia
  - Multiple myeloma
  - Polycythemia vera
  - Hypercoagulable states

- Vasculitis
- Drugs
- Carotid occlusive dz

Pearl: If a pt presents with bilateral CRVOs, consider hyperviscosity first. (Check electrophoresis, viscosity studies)
Note: The Retina book indicates that hyperviscosity retinopathy is an entity clinically similar to but distinct from CRVO, and that apparent CRVOs stemming from Waldenström’s, multiple myeloma and polycythemia vera should be considered hyperviscosity retinopathy, not CRVO.
CRVO

Primary/idiopathic

Secondary

Hyperviscosity syndrome
- Waldenström macroglobulinemia
- Multiple myeloma
- Polycythemia vera
- Hypercoagulable states

Vasculitis

Drugs

Carotid occlusive dz
CRVO

Primary/idiopathic

Secondary

Hyperviscosity syndrome
- Waldenström macroglobulinemia
- Multiple myeloma
- Polycythemia vera
- Hypercoagulable states

Vasculitis

Drugs
- Sarcoid
- SLE (*systemic lupus erythematosus*)
- (ditto)

Carotid occlusive dz
CRVO

CRVO

Primary/idiopathic

Secondary

Hyperviscosity syndrome
- Waldenström macroglobulinemia
- Multiple myeloma
- Polycythemia vera
- Hypercoagulable states

Vasculitis
- Sarcoid
- SLE

Drugs
- OCP (oral contraceptive pills)
- Diuretics
  (ditto)

Carotid occlusive dz
CRVO

Primary/idiopathic

Secondary

Hyperviscosity syndrome
  - Waldenström macroglobulinemia

Vasculitis
  - Sarcoid

Drugs
  - OCP

Carotid occlusive dz

Hypercoagulable states

Polycythemia vera

What two things does this imply regarding CRVO in young females?

--

--

Hypercoagulable states
What two things does this imply regarding CRVO in young females?
--Always inquire about OCP use in any young female presenting with CRVO
--Likewise, advise female CRVO pts to avoid OCP use
CRVO

Primary/idiopathic

CRVO

Secondary

Hyperviscosity syndrome
- Waldenström macroglobulinemia
- Multiple myeloma
- Polycythemia
- Hypercoagulable states

Vasculitis
- Sarcoid
- SLE

Drugs
- OCP
- Diuretics

Carotid occlusive dz

The **history** will be important in picking up on these. If suggestive of vasculitis, order the appropriate labs.
CRVO

Primary/idiopathic

Secondary

Hyperviscosity syndrome
- Waldenström macroglobulinemia
- Multiple myeloma
- Polycythemia vera
- Hypercoagulable states

Vasculitis
- Sarcoid
- SLE

Drugs
- OCP

Carotid occlusive dz

Ocular ischemic syndrome (OIS) can closely mimic CRVO
What aspects of a CRVO case should make you concerned that it is secondary in origin?

- If the pt is not a vasculopath, and the other risk factors (ie, cholesterol, smoking, hyperhomocystinemia) are absent;
- Or if the pt is less than 50 years of age.
What aspects of a CRVO case should make you concerned that it is secondary in origin? --If the pt is not a general med condition, and the other risk factors (ie, cholesterol, smoking, hyperhomocystinemia) are absent
What aspects of a CRVO case should make you concerned that it is secondary in origin?
--If the pt is not a vasculopath, and the other risk factors (ie, cholesterol, smoking, hyperhomocystinemia) are absent; or
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--If CRVO presents bilaterally
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--If the pt is less than 50 years of age; or
--If CRVO presents bilaterally
The more traditional way to divvy them up
The more traditional way to divvy them up

Ischemic  Nonischemic

(We’ll define ischemic and nonischemic shortly)
What if, for whatever reason, a CRVO’s ischemia-status cannot be determined?
What if, for whatever reason, a CRVO’s ischemia-status cannot be determined? Such a CRVO is classified as indeterminate.
What if, for whatever reason, a CRVO’s ischemia-status cannot be determined? Such a CRVO is classified as *indeterminate*.

What is the natural history of indeterminate CRVOs?
What if, for whatever reason, a CRVO’s ischemia-status cannot be determined? Such a CRVO is classified as indeterminate.

What is the natural history of indeterminate CRVOs? A big percentage of them turn out to be ischemic. You got a 50:50 shot...
What if, for whatever reason, a CRVO’s ischemia-status cannot be determined? Such a CRVO is classified as indeterminate.

What is the natural history of indeterminate CRVOs? ~80% of them turn out to be ischemic.
What if, for whatever reason, a CRVO’s ischemia-status cannot be determined? Such a CRVO is classified as **indeterminate**.

**What is the natural history of indeterminate CRVOs?**

~80% of them turn out to be ischemic.

As an (important) aside: A number of CRVOs initially classified as nonischemic will ‘convert’ to ischemic. What depressingly-high percentage will do so by 36 months post-event?
What if, for whatever reason, a CRVO's ischemia-status cannot be determined? Such a CRVO is classified as **indeterminate**.

What is the natural history of indeterminate CRVOs? About 80% of them turn out to be ischemic.

As an (important) aside: A number of CRVOs initially classified as nonischemic will ‘convert’ to ischemic. What depressingly-high percentage will do so by 36 months post-event? About a third.
**More re CRVO: Complete the tables and fill in the blanks**

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<thead>
<tr>
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<td><strong>No</strong></td>
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<td><strong>Good</strong></td>
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<tr>
<td><strong>Bad</strong></td>
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<td><strong>10+ disc diameters of capillary nonperfusion</strong></td>
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**What test must be run to determine whether a CRVO is ischemic or nonischemic?**

Fluorescein angiography
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What test must be run to determine whether a CRVO is ischemic or nonischemic? Fluorescein angiography
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What FA finding is common to both ischemic and nonischemic subtypes? Prolonged retinal circulation time

What FA finding defines an ischemic CRVO? 10+ disc diameters of capillary nonperfusion

What is the classic description of the fundus in CRVO? Blood and thunder

Heme and cotton-wool spots (CWS) may obscure FA hyperfluorescence, rendering the FA results uninterpretable.
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What test must be run to determine whether a CRVO is ischemic or nonischemic? Fluorescein angiography
CRVO: Blood and thunder
What test must be run to determine whether a CRVO is ischemic or nonischemic? Fluorescein angiography

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<td></td>
<td></td>
<td>Prolonged retinal circulation time with capillary nonperfusion</td>
<td>Prolonged retinal circulation time, but NO capillary nonperfusion</td>
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**What test must be run to determine whether a CRVO is ischemic or nonischemic?**

Fluorescein angiography

**What FA finding is common to both ischemic and nonischemic subtypes?**

Prolonged retinal circulation time

**What FA finding defines an ischemic CRVO?**

10+ disc diameters of capillary nonperfusion

**What is the classic description of the fundus in CRVO?**

Blood and thunder

**How are such CRVOs classified?**

As indeterminate, as mentioned previously

**Heme and cotton-wool spots (CWS) may obscure FA hyperfluorescence, rendering FA interpretation problematic**

**What impact does this frequently have on attempts to determine whether a CRVO is ischemic or not?**

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**What test must be run to determine whether a CRVO is ischemic or nonischemic?**
Fluorescein angiography

**What FA finding is common to both ischemic and nonischemic subtypes?**

10+ disc diameters of capillary nonperfusion
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What test must be run to determine whether a CRVO is ischemic or nonischemic?
Fluorescein angiography

What FA finding is common to both ischemic and nonischemic subtypes?
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CRVO: Prolonged circ time (note the timer)
What test must be run to determine whether a CRVO is ischemic or nonischemic? Fluorescein angiography

What FA finding is common to both ischemic and nonischemic subtypes? Prolonged retinal circulation time

What FA finding differentiates ischemic from nonischemic CRVO? 10+ disc diameters of capillary nonperfusion

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What test must be run to determine whether a CRVO is ischemic or nonischemic? Fluorescein angiography

What FA finding is common to both ischemic and nonischemic subtypes? Prolonged retinal circulation time

What FA finding differentiates ischemic from nonischemic CRVO? The extent of capillary nonperfusion.
What test must be run to determine whether a CRVO is ischemic or nonischemic? Fluorescein angiography

What FA finding is common to both ischemic and nonischemic subtypes? Prolonged retinal circulation time

What FA finding differentiates ischemic from nonischemic CRVO? The extent of capillary nonperfusion. In ischemic CRVO, at least 10 disc diameters of capillary nonperfusion are present, whereas in nonischemic, only a minimal amount (if any) is present.

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CRVO: Nonischemic

(A) Fundus photograph of a central retinal vein occlusion demonstrating typical features of venous tortuosity, macular thickening, and intraretinal hemorrhage in all four quadrants of the fundus. (B) Early-phase angiogram of the fundus depicted in A, demonstrating an intact parafoveal capillary network in this perfused central retinal vein occlusion
(A) Fundus photograph of an eye with central retinal vein occlusion demonstrating scattered retinal hemorrhages, venous engorgement, and cotton-wool spots. (B) Midphase fluorescein angiogram of the eye shown in A, demonstrating capillary nonperfusion involving the foveal center. This eye also had extensive peripheral nonperfusion and is an example of the nonperfused form of central retinal vein occlusion.

CRVO: Ischemic
More re CRVO: Complete the tables and fill in the blanks

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Next Q
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- ≥20/40

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**tl;dr for Final VA after CRVO:** Good vision stays good…

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**tl;dr for Final VA after CRVO:**  
*Good vision stays good...Bad vision stays bad*

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Re NVI after CRVO: According to the CVOS…

What does CVOS stand for?

(neovascularization of the iris)
Re NVI after CRVO: According to the CVOS...

What does CVOS stand for? Central Vein Occlusion Study
Re NVI after CRVO: According to the CVOS...

- What is the #1 predictor for neo?
Re NVI after CRVO: According to the CVOS...

What is the #1 predictor for neo? *Poor VA*
Re NVI after CRVO: According to the CVOS...

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Why is this important?

- When is the follow-up visit after PRP? One week; check IOP and assess response; re-treat if needed.
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For many reasons, not least of which is the fact that many CRVO pts have glaucoma.

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- What are you checking for via gonioscopy?

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- What are the main sequelae you’re looking to catch on these visits?
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- How long after the event does NVG typically appear? Somewhere in the 3-4 month range.

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When is the follow-up visit after PRP?
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Why is this important?
For many reasons, not least of which is the fact that many CRVO pts have glaucoma.

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Gonioscopy, on more than one visit

What are you checking for via gonioscopy?
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Assuming no PRP or other treatment (a subject we’ll get to shortly), how frequently should a CRVO pt be re-evaluated, and for how long?
Monthly for at least 6 months

What are the main sequelae you’re looking to catch on these visits?
The development of neovascularization (NVI/NVA)—or worse, neovascular glaucoma

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3-4 months later…This explains the name by which post-CRVO NVG is known. What is that name?
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*Trick question! The CVOS demonstrated that GML improved macular edema angiographically, but did **not** improve vision.*
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A third new treatment modality, the dexamethasone intravitreal implant (the Ozurdex), has recently received FDA approval for the treatment of macular edema after C/BRVO. So that would have been an OK answer too.
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