Before you begin: This is a big topic, and big topics beget big slide-sets. There’s a couple of natural breaks (around slides 207 and 389); *break time!* slides have been placed at those spots.
What does TVL stand for in this context?
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Transient visual loss
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**Transient visual loss**

How is the ‘transient’ in TVL defined?
What does TVL stand for in this context?

**Transient visual loss**

*How is the ‘transient’ in TVL defined?*
Less than 24 hrs
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How brief can the VL be?
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**Transient** visual loss

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*How brief can the VL be?*
As little as a few seconds*

*We will expand on this assertion shortly*
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It can be complete, or partial
**What does TVL stand for in this context?**

**Transient visual loss aka...**

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**What is the older term by which TVL is often known?**
**What does TVL stand for in this context?**

**Transient visual loss aka...amaurosis fugax**

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Time to expand... **The statement is true; however, when TVL lasts just a few seconds, by what term is it usually known?**
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**Q/A**

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*The statement is true; however, when TVL lasts just a few seconds, by what term is it usually known? Episodes of unilateral or bilateral vision loss that last but a few seconds are called transient visual obscurations (TVO)*

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There are lots of ways to categorize TVL, but one is fundamental—what is it?
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Monocular

Usually referred to as transient monocular visual loss, TMVL

Binocular

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*And maybe even do formal VF testing
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The Neuro book divvies the causes of TMVL into three broad categories—what are they?
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(To be clear: By *ocular* the book is referring to *nonvascular* causes that localize to the eye)
Which is most commonly implicated in TMVL?
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Ocular causes, by a mile
Six specific ocular etiologies are addressed—
we will pick them up one or a few at a time.
Ocular causes of TVML are most common.

- Dry eyes

We already touched on this. Note that it’s the reason
The odds of this one go up considerably if your pt has an AC IOL or malpositioned PC IOL in the affected eye: Uveitis-glaucoma-hyphema (UGH) syndrome.
Ocular

--Dry eyes
--Recurrent hyphema
--?

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If your recurrent-hyphema-with-TMVL has an AC IOL in that eye, what diagnosis tops the DDx?
--Dry eyes
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Uveitis-glaucoma-hyphema (UGH) syndrome (see slide-set L2 for a discussion thereof)
In these two, the pt will likely c/o halos around lights in the affected eye:

Dry eyes
Recurrent hyphema

What ocular (but nonvisual) c/o do both conditions present with?

Ocular pain

What causes the TMVL and light-haloes?

Corneal edema

What causes the corneal edema in:

- Angle-closure glaucoma?
  - A sharp increase in IOP
- PDS?
  - Also a sharp increase in IOP

What is the classic precipitating event for TMVL/halos/eye pain in each?

- Angle closure:
  - An emotionally-charged occurrence
- PDS:
  - Exercise
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What are glaukomflecken?
Opacities of the sub-anterior lens capsule

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**Ocular Vascular**

**Orbital**

--- Dry eyes
--- Recurrent hyphema
--- Pigment dispersion syndrome
--- Angle-closure glaucoma
--- Optic nerve abnormalities

--- ?

**TMVL Binocular**

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For more info on…

**PDS**: see slide-set G4

**Angle-closure glaucoma**: Sets G16, 17, and 18

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--angle-closure glaucoma? A sharp increase in IOP
--PDS? Also a sharp increase in IOP

What is the classic precipitating event for TMVL/halos/eye pain in each?
--Angle closure: An event that promotes pupil dilation
--PDS: Exercise

---
When this structure is implicated in TVL, the pt often reports that a change in posture precipitates the vision loss:

- Dry eyes
- Recurrent hyphema
- Pigment dispersion syndrome
- Angle-closure glaucoma
- ?
- ?

When you hear 'TVL associated with postural change,' two ONH conditions should come to mind. What are they?

- Papilledema
- Optic nerve drusen

How long do TVLs of this sort typically last?

A few seconds (hence they are more commonly labeled TVOs)
Ocular
--Dry eyes
--Recurrent hyphema
--Pigment dispersion syndrome
--Angle-closure glaucoma
--Optic nerve abnormalities

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--Angle-closure glaucoma
--Optic nerve abnormalities

Orbital

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A few seconds (hence they are more commonly labeled TVOs)
Ocular

- Dry eyes
- Recurrent hyphema
- Pigment dispersion syndrome
- Angle-closure glaucoma
- Optic nerve abnormalities

Orbital

When this structure is implicated in TVL, the pt often reports that a change in posture precipitates the vision loss.

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- Papilledema
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How long do TVLs of this sort typically last?

A few seconds (hence they are more commonly labeled TVOs)

Head’s up—Don’t lock in ‘posture change in TVL = optic nerve issue.’ In a few slides we will encounter other causes of TVL that are influenced by changes in posture!
Ocular

- Dry eyes
- Recurrent hyphema
- Pigment dispersion syndrome
- Angle-closure glaucoma
- Optic nerve abnormalities
- **Macular disorder** *(couldn't come up with a top-line question for this one)*

Vascular

Orbital

TMVL

TVL

Binocular
Ocular

--Dry eyes
--Recurrent hyphema
--Pigment dispersion syndrome
--Angle-closure glaucoma
--Optic nerve abnormalities

--Macular disorder

‘Macular disorder’ is rather vague. What three conditions does the Neuro book mention in this regard?

--?
--?
--?
Ocular Vascular Orbital

TMVL

Binocular

TVL

Ocular

--Dry eyes
--Recurrent hyphema
--Pigment dispersion syndrome
--Angle-closure glaucoma
--Optic nerve abnormalities
--Macular disorder

Orbital

'Macular disorder' is rather vague. What three conditions does the Neuro book mention in this regard?

--Serous RD
--ARMD
--Ocular ischemia

These pts may c/o a specific trigger for their TVL—what is it?

Exposure to a very bright light. In ophthospeak, they c/o a prolonged photostress recovery time.

In addition to TVL, these pts may c/o that bright lights produce prolonged afterimages.
‘Macular disorder’ is rather vague. What three conditions does the Neuro book mention in this regard?

--Serous RD
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These pts may c/o a specific trigger for their TVL—what is it?
Q/A

TVL

Ocular

--Dry eyes
--Recurrent hyphema
--Pigment dispersion syndrome
--Angle-closure glaucoma
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--Macular disorder

Orbital

'Macular disorder' is rather vague. *What three conditions does the Neuro book mention in this regard?*

--Serous RD
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*These pts may c/o a specific trigger for their TVL—what is it?*

Exposure to a very

two words
Ocular

--Dry eyes
--Recurrent hyphema
--Pigment dispersion syndrome
--Angle-closure glaucoma
--Optic nerve abnormalities

`Macular disorder` is rather vague. What three conditions does the Neuro book mention in this regard?
--Serous RD
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These pts may c/o a specific trigger for their TVL—what is it?
Exposure to a very bright light

Orbital
Ocular
--Dry eyes
--Recurrent hyphema
--Pigment dispersion syndrome
--Angle-closure glaucoma
--Optic nerve abnormalities
--Macular disorder

Orbital

'Macular disorder' is rather vague. What three conditions does the Neuro book mention in this regard?
--Serous RD
--ARMD
--Ocular ischemia

These pts may c/o a specific trigger for their TVL—what is it? Exposure to a very bright light. In ophthospeak, they c/o a prolonged three words.
Ocular

- Dry eyes
- Recurrent hyphema
- Pigment dispersion syndrome
- Angle-closure glaucoma
- Optic nerve abnormalities
- Macular disorder

Orbital

'Macular disorder' is rather vague. What three conditions does the Neuro book mention in this regard?

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- Recurrent hyphema
- Pigment dispersion syndrome
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In addition to TVL, these pts may c/o that bright lights produce prolonged [underline].
Ocular
--- Dry eyes
--- Recurrent hyphema
--- Pigment dispersion syndrome
--- Angle-closure glaucoma
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In addition to TVL, these pts may c/o that bright lights produce prolonged afterimages.
Ocular Vascular Orbital

TVL

TMVL Binocular

Q

Dry eyes
- Recurrent hyphema
- Pigment dispersion syndrome
- Angle-closure glaucoma
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- Macular disorder

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In addition to TVL, these pts may c/o that bright lights produce prolonged afterimages.

Can photostress recovery time be formally assessed in the clinic?

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.
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.
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-- Recurrent hyphema
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Exposure to a very bright light—in ophthalmospeak, they c/o a prolonged photostress recovery time.

In addition to TVL, these pts may c/o that bright lights produce prolonged afterimages.
Can photostress recovery time be formally assessed in the clinic?
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Exposure to a very bright light (ophthospeak, they c/o a prolonged)

photostress recovery time

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**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.

Exposure to a very bright light (ophthalmospeak, they c/o a prolonged *photostress recovery time*).

In addition to TVL, these pts may c/o that bright lights produce prolonged afterimages.
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Exposure to a very bright light - in ophthospeak, they c/o a prolonged 'photostress recovery time'.

In addition to TVL, these pts may c/o that bright lights produce prolonged afterimages.
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; it is pathologic if much longer than this.

In addition to TVL, these pts may c/o that bright lights produce prolonged afterimages.
What complaint/finding should alert you to the possibility that a pt’s TMVL is orbital in origin?
What complaint/finding should alert you to the possibility that a pt’s TMVL is orbital in origin?

That the vision loss is precipitated by two words
What complaint/finding should alert you to the possibility that a pt’s TMVL is orbital in origin?
That the vision loss is precipitated by eye movement
What complaint/finding should alert you to the possibility that a pt’s TMVL is orbital in origin?

That the vision loss is precipitated by eye movement, aka two-words TVL.
What complaint/finding should alert you to the possibility that a pt’s TMVL is orbital in origin?
That the vision loss is precipitated by eye movement, aka *gaze-evoked TVL*
What complaint/finding should alert you to the possibility that a pt’s TMVL is orbital in origin? That the vision loss is precipitated by eye movement, aka *gaze-evoked TVL*.

What other findings might be present that would clue you in to the possibility of an orbital process?
--?
--?
What complaint/finding should alert you to the possibility that a pt’s TMVL is orbital in origin?

That the vision loss is precipitated by eye movement, aka *gaze-evoked* TVL

What other findings might be present that would clue you in to the possibility of an orbital process?

-- Proptosis
-- EOM limitations
What complaint/finding should alert you to the possibility that a pt’s TMVL is orbital in origin?
That the vision loss is precipitated by eye movement, aka *gaze-evoked TVL*

What other findings might be present that would clue you in to the possibility of an orbital process?
--Proptosis
--EOM limitations

*In broad/general terms, what is the likely cause of orbit-related TMVL?*
What complaint/finding should alert you to the possibility that a pt’s TMVL is orbital in origin?
That the vision loss is precipitated by eye movement, aka gaze-evoked TVL

What other findings might be present that would clue you in to the possibility of an orbital process?
--Proptosis
--EOM limitations

In broad/general terms, what is the likely cause of orbit-related TMVL?
An orbital mass
What complaint/finding should alert you to the possibility that a pt’s TMVL is orbital in origin?
That the vision loss is precipitated by eye movement, aka gaze-evoked TVL

What other findings might be present that would clue you in to the possibility of an orbital process?
--Proptosis
--EOM limitations

In broad/general terms, what is the likely cause of orbit-related TMVL?
An orbital mass

The Neuro book mentions three examples—what are they?
What complaint/finding should alert you to the possibility that a pt’s TMVL is orbital in origin?
That the vision loss is precipitated by eye movement, aka gaze-evoked TVL

What other findings might be present that would clue you in to the possibility of an orbital process?
--Proptosis
--EOM limitations

In broad/general terms, what is the likely cause of orbit-related TMVL?
An orbital mass

The Neuro book mentions three examples—what are they?
- Hemangioma
- Meningioma
- Foreign body
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)
Hypoperfusion*

*Ischemia is also a reasonable answer here (in fact, we’ll use it shortly)
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve.

Hypoperfusion of what two structures are implicated?
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of...the retina and/or optic nerve

Hypoperfusion of what two structures are implicated?
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of...the retina and/or optic nerve?

In the TMVL section, the book discusses at length only one cause of optic nerve hypoperfusion. What is it?
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)
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In the TMVL section, the book discusses at length only one cause of optic nerve hypoperfusion. What is it?
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.) Hypoperfusion of...the retina and/or optic nerve

In general terms, what is GCA?
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.) Hypoperfusion of...the retina and/or optic nerve

In general terms, what is GCA? An inflammatory disease that targets arteries.

GCA

small? mid-sized? large?
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)
Hypoperfusion of...the retina and/or optic nerve

In general terms, what is GCA?
An inflammatory disease that targets mid-sized arteries
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

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In general terms, what is GCA?
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Who is the classic GCA patient?
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In general terms, what is GCA?
An inflammatory disease that targets mid-sized arteries

Who is the classic GCA patient?
An old white lady
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Who is the classic GCA patient? An old white lady.

How old is old?
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.) Hypoperfusion of...the retina and/or optic nerve.

In general terms, what is GCA? An inflammatory disease that targets mid-sized arteries.

Who is the classic GCA patient? An old white lady.

How old is old? Usually 70+.
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.) Hypoperfusion of...the retina and/or optic nerve.

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Who is the classic GCA patient? An old white lady.

How old is old? Usually 70+.

At what age should GCA enter one’s DDx for TMVL?
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Who is the classic GCA patient?
An old white lady

How old is old?
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At what age should GCA enter one’s DDx for TMVL?
The Neuro book says to consider it in anyone 50+
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.) Hypoperfusion of...the retina and/or optic nerve.

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An old white lady.

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At what age should GCA enter one's DDx for TMVL?

The Neuro book says to consider it in anyone 50+.

Other than TMVL, what symptoms might a GCA pt report?

--?
--?
--?
--?
--?
--?
--?
--?

--(There are other legit answers as well)
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

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At what age should GCA enter one's DDx for TMVL?

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Other than TMVL, what symptoms might a GCA pt report?

--Headache
--Jaw claudication
--Fever
--Malaise
--PMR symptoms
--Diplopia

--(There are other legit answers as well)
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

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At what age should GCA enter one's DDx for TMVL?

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Other than TMVL, what else can a patient with GCA present with?

--Headache
--Jaw claudication
--Fever
--Malaise
--PMR symptoms
--Diplopia
--(There are other legit answers as well)

What is jaw claudication?

Pain in the jaw brought on by chewing

What is the etiology?

Same as the leg claudication that PAD pts get when walking—poor muscle perfusion \(\rightarrow\) muscle ischemia with use \(\rightarrow\) pain

Does the pain localize to the TMJ?

No! If it does, it's not claudication
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.) Hypoperfusion of…the retina and/or optic nerve.

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At what age should GCA enter one's DDx for TMVL? The Neuro book says to consider it in anyone 50+.

Other than TMVL, what are other clinical GCA clues?--Headache
--Jaw claudication
--Fever
--Malaise
--PMR symptoms
--Diplopía
--(There are other legit answers as well)

What is jaw claudication? Pain in the jaw brought on by use.

Does the pain localize to the TMJ? No! If it does, it's not claudication.
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Other than TMVL, what are other signs signaling GCA when considering TMVL?

--Headache
--Jaw claudication
--Fever
--Malaise
--PMR symptoms
--Diplopia
--(There are other legitimate answers as well)

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The etiology?

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Other than TMVL, when should one consider GCA when evaluating:

--Headache
--Jaw claudication
--Fever
--Malaise
--PMR symptoms
--Diplopia
--(There are other legit)

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At what age should GCA enter one’s DDx for TMVL? The Neuro book says to consider it in anyone 50+.

Other than TMVL, what are other conditions that CCA targets?—Headache,
--Jaw claudication
--Fever
--Malaise
--PMR symptoms
--Diplopia
--(There are other legitimate answers)

What is jaw claudication? Pain in the jaw brought on by chewing.

What is the etiology? Same as the leg claudication that PAD pts get when walking—poor muscle perfusion → muscle ischemia with use → pain.

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Usually 70+

At what age should GCA enter one's DDx for TMVL?
The Neuro book says to consider it in anyone 50+

Other than TMVL, what are other symptoms of GCA that could help with the diagnosis?
- Headache
- Jaw claudication
- Fever
- Malaise
- PMR symptoms
- Diplopia
- (There are other legitimate answers)

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What does PMR stand for in this context?
Polymyalgia rheumatica

Which is...?
A syndrome consisting of pain and stiffness in the proximal muscles/joints (ie, shoulder and hip joints)
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.) Hypoperfusion of...the retina and/or the optic nerve.

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An old white lady.

How old is old?
Usually 70+.

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--H&H

What procedure is the gold standard for diagnosing GCA?
Temporal artery biopsy (TAB).

Why biopsy the temporal artery?
It’s readily accessible, and (usually) nonvital.

What is the treatment for GCA?
High-dose steroids started immediately.

How high is high?
Pts with TMVL should receive IV pred 1 g/d x 3-5 days, then switch over to PO.
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)
Hypoperfusion of...the retina and/or optic nerve

What lab studies might be useful in diagnosing GCA?
--- ESR
--- CRP
--- Platelet count
--- H&H

What procedure is the gold standard for diagnosing GCA?
Temporal artery biopsy (TAB)

What is jaw claudication?
Pain in the jaw brought on by chewing

What is the etiology?
Same as the leg claudication that PAD pts get when walking—poor muscle perfusion→muscle ischemia with use→pain

Does the pain localize to the TMJ?
No! If it does, it's not claudication

What does PMR stand for in this context?
Polymyalgia rheumatica

Which is...
A syndrome consisting of pain and stiffness in the proximal muscles/joints (ie, shoulder and hip joints/muscles)

If a TMVL pt is over 50—and especially if she’s over 70, and AFAB—establishing an index of suspicion for GCA (and acting on it, if high) is the first order of the day. (Fortunately, this is easily accomplished via a few questions posed while taking an initial history or performing the anterior-segment slit lamp exam.)
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)
Hypoperfusion of...the retina and/or optic nerve

In general terms, what is GCA?
An inflammatory disease that targets mid-sized arteries

Who is the classic GCA patient?
An old white lady

How old is old?
Usually 70+

At what age should GCA enter one's DDx for TMVL?
The Neuro book says to consider it in anyone 50+

Other than TMVL, what symptoms might a GCA pt report?
--Headache
--Jaw claudication
--Fever
--Malaise
--PMR symptoms
--Diplopia

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What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)
- Hypoperfusion of...the retina and/or optic nerve
- GCA

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- Malaise
- PMR symptoms
- Diplopia

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What does PMR stand for in this context?
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- A syndrome consisting of pain and stiffness in the proximal muscles/joints (ie, shoulder and hip joints/muscles)

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For more on GCA, see slide-set N17
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.) Hypoperfusion of… the retina and/or optic nerve?

The book discusses three processes by which retinal hypoperfusion may come about. What are they?
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.) Hypoperfusion of...the retina and/or optic nerve.

The book discusses three processes by which retinal hypoperfusion may come about. What are they?

- Embolization of retinal arterial tree
- Occlusion in the retinal venous system
- Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of…the retina and/or optic nerve

The book discusses three processes by which retinal hypoperfusion may come about. What are they?

- Embolization of retinal arterial tree
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We’ll come back to and drill down on these shortly
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of…the retina and/or optic nerve

The ‘and’ here implies that the hypoperfusion in question is global or diffuse.
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.) Hypoperfusion of...the retina and/or optic nerve.

The ‘and’ here implies that the hypoperfusion in question is global or diffuse. In this regard, two specific conditions are mentioned—what are they?
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of...the retina and/or optic nerve

The ‘and’ here implies that the hypoperfusion in question is global or diffuse. In this regard, two specific conditions are mentioned—what are they?

Diffuse ocular hypoperfusion
- OIS
- Stenosis of the great vessels

- Embolization of retinal arterial tree
- Occlusion in the retinal venous system
- Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)
Hypoperfusion of...the retina and/or optic nerve

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What does OIS stand for in this context?

--OIS
--Stenosis of the great vessels
Embolization of retinal arterial tree
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What does OIS stand for in this context? Ocular ischemic syndrome

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---Stenosis of the great vessels
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--OIS
--Stenosis of the great vessels

To what does 'great vessels' refer?

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Vasospasm of the CRA
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What does OIS stand for in this context? Ocular ischemic syndrome

To what does ‘great vessels’ refer? To the vessels that directly enter or exit the heart
In a nutshell, what is OIS?

Ocular ischemic syndrome

---Stenosis of the great vessels

To what does ‘great vessels’ refer?
To the vessels that directly enter or exit the heart

Enlarged retinal arteriolar caliber
Occlusion in the retinal venous system
Vasospasm of the CRA

Hypoperfusion of the retina and/or optic nerve
Diffuse ocular hypoperfusion—OIS
Stenosis of the great vessels
Embolization of retinal arterial tree
Occlusion in the retinal venous system
Vasospasm of the CRA

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Ocular hypoperfusion—OIS
Stenosis of the great vessels
Embolization of retinal arterial tree
Occlusion in the retinal venous system
Vasospasm of the CRA

What does OIS stand for in this context?

Ocular ischemic syndrome

Does OIS 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

Does OIS carry implications for the general health of the afflicted individual?

Indeed it does—the 5-year mortality rate associated with OIS is 40%!
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

Diffuse ocular hypoperfusion -- OIS -- Stenosis of the great vessels

Embolization of retinal arterial tree

Occlusion in the retinal venous system

Vasospasm of the CRA

What does OIS stand for in this context?

Ocular ischemic syndrome

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To what does ‘great vessels’ refer?

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In a nutshell, what is OIS?

A constellation of signs and symptoms owing to chronic ocular ischemia/hypoperfusion

Where’s the choke point, ie, what vessel is being occluded that produces the chronic hypoperfusion?

The vessel most commonly implicated is the ipsilateral internal carotid artery

How occluded does the internal carotid artery (ICA) have to be for OIS to occur?

Very—at least 50%, and probably closer to 90%

Where's the choke point, ie, what vessel is being occluded that produces the chronic hypoperfusion?
In a nutshell, what is OIS?
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Vasospasm of the CRA
Occlusion in the retinal venous system
Stenosis of the great vessels

En

TVL

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Diffuse ocular hypoperfusion — OIS

Stenosis of the great vessels

Embolization of retinal arterial tree

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**OIS**

— Stenosis of the great vessels

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Ocular ischemic syndrome

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A constellation of signs and symptoms owing to chronic ocular ischemia/hypoperfusion

Where’s the choke point, ie, what vessel is being occluded that produces the chronic hypoperfusion?

The vessel most commonly implicated is the ipsilateral internal carotid artery

How occluded does the internal carotid artery (ICA) have to be for OIS to occur?

Very—at least 50%, and probably closer to 90%
High-grade stenosis of the internal carotid artery origin (arrow) in two pts
What are the signs/symptoms of OIS?

Signs:

- ?
- ?
- ?
- ?

Symptoms:

- ?
- ?
- ?
- ?

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

What does OIS stand for in this context?
Ocular ischemic syndrome.

To what does ‘great vessels’ refer?
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Diffuse ocular hypoperfusion -- OIS -- Stenosis of the great vessels

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Vasospasm of the CRA

What does OIS stand for in this context?

Ocular ischemic syndrome

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In a nutshell, what is OIS?

A constellation of **signs and symptoms** owing to chronic ocular ischemia/hypoperfusion

What are the signs/symptoms of OIS?

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

**Symptoms**

-- Retinal vascular changes

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**Signs:**

-- Intraretinal hemorrhages

-- NVI/NVA

-- AC cell/flare

-- Retinal vascular changes

**Symptoms**
Classic presentation of intraretinal hemorrhages in OIS:

- Location
- Size
- Shape/Type
Classic presentation of intraretinal hemorrhages in OIS:

Midperipheral, medium-large, dot-blot
Classic vascular changes in OIS:

--Arteriolar
Classic vascular changes in OIS:
--Arteriolar narrowing
Classic vascular changes in OIS:
--Arteriolar **narrowing**
--Venules **but not**
Classic vascular changes in OIS:
--Arteriolar **narrowing**
--Venules **dilated** but not **tortuous**
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

- Hypoperfusion of the retina and/or optic nerve
- Diffuse ocular hypoperfusion -- OIS
- Stenosis of the great vessels
- Embolization of retinal arterial tree
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- Vasospasm of the CRA

In a nutshell, what is OIS?

A constellation of **signs and symptoms** owing to chronic ocular ischemia/hypoperfusion.

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- Retinal vascular changes

**Symptoms:**
- ?
- ?
- ?

What does OIS stand for in this context?

**Ocular ischemic syndrome**

To what does ‘great vessels’ refer?

- To the vessels that directly enter or exit the heart

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- Retinal vascular changes

**Symptoms:**
- ?
- ?
- ?
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

Diffuse ocular hypoperfusion -- OIS -- Stenosis of the great vessels

Embolization of retinal arterial tree

Occlusion in the retinal venous system

Vasospasm of the CRA

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A constellation of signs and symptoms owing to chronic ocular ischemia/hypoperfusion

What are the signs/symptoms of OIS?

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

**Symptoms:**
- Decreased vision
- Pain (described as ‘dull’)
- Prolonged photostress recovery time

Does it present unilaterally, or bilaterally?

Unilaterally (in about 80% of cases)

Is there a gender predilection?

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OIS
--Stenosis of the great vessels

En
Occlusion in the retinal venous system
Vasospasm of the CRA
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A constellation of signs and symptoms owing to chronic ocular ischemia/hypoperfusion

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Unilaterally (in about % of cases)

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Hypoperfusion of the retina and/or optic nerve

Diffuse ocular hypoperfusion—OIS—Stenosis of the great vessels

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**OIS**

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Unilaterally (in about 80% of cases)

Is there a gender predilection?
Yes, M vs F are twice as likely to have it

In what aspect of OIS is stenosis of the great vessels mentioned?

To what does ‘great vessels’ refer?
To the vessels that directly enter or exit the heart

Occlusion in the retinal venous system
Vasospasm of the CRA

What is the ultimate cause of TMVL related to the vasculature?
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What is OIS?
A constellation of signs and symptoms owing to chronic ocular ischemia/hypoperfusion

OIS stands for...
Ocular Ischemic Syndrome

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A constellation of signs and symptoms owing to chronic ocular ischemia/hypoperfusion

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OIS

Stenosis of the great vessels

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Ocular Ischemic Syndrome

--Stenosis of the great vessels

To what does ‘great vessels’ refer?
To the vessels that directly enter or exit the heart

Occlusion in the retinal venous system

Vasospasm of the CRA
In a nutshell, what is OIS?
A constellation of signs and symptoms owing to chronic ocular ischemia/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

Does OIS carry implications for the general health of the afflicted individual?
Indeed it does—the 5-year mortality rate associated with OIS is yikes

To what does ‘great vessels’ refer?
To the vessels that directly enter or exit the heart

---Stenosis of the great vessels

Occlusion in the retinal venous system

Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

Diffuse ocular hypoperfusion -- OIS -- Stenosis of the great vessels

Embolization of retinal arterial tree

Occlusion in the retinal venous system

Vasospasm of the CRA

What does OIS stand for in this context?

Ocular ischemic syndrome

To what does ‘great vessels’ refer?

To the vessels that directly enter or exit the heart

In a nutshell, what is OIS?

A constellation of signs and symptoms owing to chronic ocular ischemia/hypoperfusion

Does it present unilaterally, or bilaterally?

Unilaterally (in about 80% of cases)

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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

Does OIS carry implications for the general health of the afflicted individual?

Indeed it does — the 5-year mortality rate associated with OIS is 40%! 
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

Diffuse ocular hypoperfusion

--OIS

--Stenosis of the great vessels

Embolization of retinal arterial tree

Occlusion in the retinal venous system

Vasospasm of the CRA

The ‘and’ here implies that the hypoperfusion in question is global or diffuse. In this regard, two specific conditions are mentioned—what are they?

Remember when we remarked a while back that we would encounter other TVL conditions for which ‘posture change’ played a role? We are now encountering them now.

They report experiencing TVL when they go from a sitting to a standing position

The OIS-associated ocular ache gets better when they lie down
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of...and/or optic nerve

Diffuse ocular hypoperfusion --OIS --Stenosis of the great vessels

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What is the classic posture-related finding pts report with regard to stenosis of the great vessels?
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of…

Diffuse ocular hypoperfusion—OIS—Stenosis of the great vessels

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What is the classic posture-related finding pts report with regard to OIS?

The OIS-associated ocular ache gets better when they lie down.

Embolization of retinal arterial tree
Occlusion in the retinal venous system
Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

- Hypoperfusion of the retina and/or optic nerve
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The OIS-associated ocular symptom gets better when they lie down vs stand up.

The ‘and’ here implies that the hypoperfusion in question is global or diffuse. In this regard, two specific conditions are mentioned—what are they?
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

- Hypoperfusion of the retina and/or optic nerve

**Diffuse ocular hypoperfusion**
- OIS
- Stenosis of the great vessels

The 'and' here implies that the hypoperfusion in question is global or diffuse. In this regard, two specific conditions are mentioned—what are they?

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They report experiencing TVL when they go from a sitting to a standing position.

What is the classic posture-related finding pts report with regard to **OIS**?
The OIS-associated ocular ache gets better when they lie down.
(This is a good point in the set to take a break)
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.) Hypoperfusion of...the retina and/or optic nerve.

Next we will delve into TMVL secondary to embolization. Take note: This topic receives more attention than any other in the chapter.
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.) Hypoperfusion of...the retina and/or optic nerve.

Next we will delve into TMVL 2ndry to embolization. Take note: This topic receives more attention than any other in the chapter. The point being: As it is obviously of high importance to the people responsible for the content of the BCSC—and by extension, the content of the OKAPs and Boards—so too should it be of high importance to you!
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

- Hypoperfusion of the retina and/or optic nerve
- GCA
- Diffuse ocular hypoperfusion
  --OIS
  --Stenosis of the great vessels
- Embolization of retinal arterial tree
- Occlusion in the retinal venous system
- Vasospasm of the CRA

What is the classic description of TMVL secondary to retinal embolus?

A curtain moving fairly quickly across a portion (or all) of the VF

Is it associated with pain?

No, it is painless

How long until the vision loss resolves?

Usually a matter of minutes
What is the classic description of TMVL 2ndry to retinal embolus? A moving fairly quickly across a portion (or all) of the VF
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How long until the vision loss resolves?
Usually a matter of minutes? seconds? hours? days?

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In terms of how we should think about/approach them, what are the key differences between a retinal TIA and a cerebral one?

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In general terms, where do emboli usually lodge?

At a branch point in the arterial tree. That is, an emboli will continue to float along in the bloodstream until it reaches a bifurcation for which it is too large to travel down either fork, and becomes lodged. (There is one caveat to this—we’ll get to it a few slides down the road.)

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Hypoperfusion of the retina and/or optic nerve

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Hypoperfusion of the retina and/or optic nerve

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4. Diffuse ocular hypoperfusion--OIS
   --Stenosis of the great vessels

3. Embolization of retinal arterial tree
   Occlusion in the retinal venous system
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---

What are the three types of embolus?

- Cholesterol
- Calcium
- Platelet-fibrin

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Emboli composed of cholesterol are known by what eponymous name?
Hollenhorst plaque

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Hollenhorst plaque

What do Hollenhorst plaques look like on DFE?

Yellowish globules described as ‘refractile’
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

- Hypoperfusion of the retina and/or optic nerve
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Hollenhorst plaque

What do Hollenhorst plaques look like on DFE?
Yellowish globules described as color(ish) appearance
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

- Diffuse ocular hypoperfusion --OIS
- Stenosis of the great vessels

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Hollenhorst plaque

What do Hollenhorst plaques look like on DFE?
Yellowish globules described as ‘refractile’
Hollenhorst plaque at the bifurcation of a retinal arteriole
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

Diffuse ocular hypoperfusion --OIS --Stenosis of the great vessels

Embolization of retinal arterial tree

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In general terms, where do emboli usually lodge? At a branch point in the arterial tree. That is, an emboli will continue to float along in the bloodstream until it reaches a bifurcation for which it is too large to travel down either fork, and becomes lodged. (There is one caveat to this—we’ll get to it a few slides down the road.)

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What do Hollenhorst plaques look like on DFE? Yellowish globules described as ‘refractile’

In two words, what pathologic entity is the source of cholesterol emboli?

Atheromatous plaques

Embolization of retinal arterial tree

Occlusion in the retinal venous system

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Within which vascular structure are TVL-implicated plaques most likely to be located?
The carotid artery

Where specifically in the carotid system are such plaques most likely to form?
The bifurcation

How stenosed does the carotid lumen have to be for emboli to occur?
Emboli can occur at any degree of stenosis (in contrast to global hypoperfusion conditions, eg, OIS)

What two characteristics of an atheromatous plaque increase the likelihood it will flick off emboli?
If it is ulcerated and/or unstable
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

GCA

Diffuse ocular hypoperfusion -- OIS -- Stenosis of the great vessels

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At a branch point in the arterial tree. That is, an emboli will continue to float along in the bloodstream until it reaches a bifurcation for which it is too large to travel down either fork, and becomes lodged. (There is one caveat to this – we’ll get to it a few slides down the road.)

What are the three types of embolus?

-- Cholesterol
-- Calcium
-- Platelet-fibrin

Emboli composed of cholesterol are known by what eponymous name?

Hollenhorst plaque

What do Hollenhorst plaques look like on DFE?

Yellowish globules described as ‘refractile’

In two words, what pathologic entity is the source of cholesterol emboli?

Atheromatous plaques

Within which vascular structure are TVL-implicated plaques most likely to be located?

The carotid artery

Where specifically in the carotid system are such plaques most likely to form?

The bifurcation

How stenosed does the carotid lumen have to be for emboli to occur?

Emboli can occur at any degree of stenosis (in contrast to global hypoperfusion conditions, eg, OIS)

What two characteristics of an atheromatous plaque increase the likelihood it will flick off emboli?

If it is ulcerated and/or unstable

Embolization of retinal arterial tree

Occlusion in the retinal venous system

Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

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- Hypoperfusion of the retina and/or optic nerve
- GCA
- Diffuse ocular hypoperfusion
- --OIS
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Hypoperfusion of...the retina and/or optic nerve.

GCA

Diffuse ocular hypoperfusion -- OIS -- Stenosis of the great vessels

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What do Hollenhorst plaques look like on DFE? (two words)

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The Neuro book mentions another “common” (their word) location for atheromatous plaques that can give rise to cholesterol emboli—what is it?

The aortic arch
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

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Hypoperfusion of the retina and/or optic nerve

Diffuse ocular hypoperfusion -- OIS

-- Stenosis of the great vessels

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- Hypoperfusion of the retina and/or optic nerve

What are the three types of embolus?
- Cholesterol
- Calcium
- Platelet-fibrin

What is the classic source of calcium emboli?
- Diseased heart valves; eg, calcific aortic stenosis

What do calcium emboli look like on DFE?
- Chalk-white and round

Do they tend to be larger, or smaller than Hollenhorst plaques?
- Larger. In fact, they tend to be so large as to not be able to pass farther along the arterial tree than the first bifurcation, ie, they often lodge at the ONH itself.
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

- GCA
- Diffuse ocular hypoperfusion
  -- OIS
  -- Stenosis of the great vessels

- Embolization of retinal arterial tree
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Hypoperfusion of the retina and/or optic nerve

GCA

Diffuse ocular hypoperfusion --OIS --Stenosis of the great vessels

Embolization of retinal arterial tree

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Vasospasm of the CRA

What are the three types of embolus?

--Cholesterol

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What is the classic source of calcium emboli?

Diseased heart valves; eg, calcific aortic stenosis

Speaking of diseased heart valves: There is a particular condition/history—classic for leading to heart-valve disease—that, if mentioned, should clue you in that an embolus is calcific. What history?

Diseased heart valves; eg, calcific aortic stenosis

What do calcium emboli look like on DFE?

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What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)
Hypoperfusion of the retina and/or optic nerve

GCA
--Diffuse ocular hypoperfusion
--OIS
--Stenosis of the great vessels

What are the three types of embolus?
--Cholesterol
--Calcium
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What is the classic source of calcium emboli?
Diseased heart valves; eg, calcific aortic stenosis

Speaking of diseased heart valves: There is a particular condition/history—classic for leading to heart-valve disease—that, if mentioned, should clue you in that an embolus is calcific. What history?
Rheumatic heart disease

Embolization of retinal arterial tree
--Occlusion in the retinal venous system
--Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)
Hypoperfusion of the retina and/or optic nerve

What are the three types of embolus?
- Cholesterol
- Calcium
- Platelet-fibrin

What is the classic source of calcium emboli?
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Larger. In fact, they tend to be so large as to not be able to pass farther along the arterial tree than the first bifurcation, ie, they often lodge at the ONH itself.
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

- Hypoperfusion of the retina and/or optic nerve

What are the three types of embolus?
- Cholesterol
- Calcium
- Platelet-fibrin

What is the classic source of calcium emboli?
Diseased heart valves; eg, calcific aortic stenosis

What do calcium emboli look like on DFE?
- Color and shape

- Diffuse ocular hypoperfusion
  --OIS
  --Stenosis of the great vessels

- Embolization of retinal arterial tree
- Occlusion in the retinal venous system
- Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

GCA

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Calcific embolus with branch retinal artery occlusion
What are the three types of embolus?
--Cholesterol
--Calcium
--Platelet-fibrin

What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)
Hypoperfusion of the retina and/or optic nerve

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Embolization of retinal arterial tree
--Occlusion in the retinal venous system
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What is the classic source of platelet-fibrin emboli?
A thrombus in the atrium of a pt with A-fib

What do platelet-fibrin emboli look like on DFE?
Unlike their calcific and cholesterol cousins, platelet-fibrin emboli are not compact structures—they tend to be elongated, filling a small section of an arteriole. For this reason, they may lodge in and occlude an arteriole at a non-branch point.
What is the ultimate cause of TMVL related to the vasculature?

- Hypoperfusion of the retina and/or optic nerve

**Ocular**

What are the three types of embolus?

- Cholesterol
- Calcium
- Platelet-fibrin

**What is the classic source of platelet-fibrin emboli?**

A thrombus in the atrium of a pt with [dx]

**Embolization of retinal arterial tree**

- Diffuse ocular hypoperfusion
  --OIS
  --Stenosis of the great vessels
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Q/A
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

- GCA
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What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

- Hypoperfusion of the retina and/or optic nerve

- GCA

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*This is the caveat to the ‘bifurcation rule’ alluded to earlier.
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When you hear ‘retinal arteriole occlusion at non-branch points,’ another specific condition should come to mind. What is it?

Susac syndrome

What is the classic presentation?

Multiple bilateral BRAOs occurring at non-branch points

Is it common, or rare?

Rare

In terms of age and gender, who is the classic patient?

A young-adult (15-40 or so) female

Susac syndrome has two classic nonophthalmic manifestations—what are they?

-- Sensorineural hearing loss

-- Encephalopathy
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

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Susac syndrome. Note the area of infarct doesn’t correspond to a branch-point blockage.
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What is the classic presentation?
Multiple bilateral BRAOs occurring at non-branch points

occlude an arteriole at a non-branch point.

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Susac syndrome. Note the multiple areas of arteriolar inflammation and blockage at non-branch points.
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Is it common, or rare?

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Diffuse ocular hypoperfusion
--OIS
--Stenosis of the great vessels

Embolization of retinal arterial tree
Occlusion in the retinal venous system
Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

GCA

Diffuse ocular hypoperfusion

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What are the three types of embolus?

--Cholesterol

--Calcium

--Platelet-fibrin

What is the classic source of platelet-fibrin emboli?

A thrombus in the atrium of a pt with A-fib

What do platelet-fibrin emboli look like on DFE?

Unlike their calcific and cholesterol cousins, platelet-fibrin emboli are not compact structures— they tend to be elongated, filling a small section of an arteriole. For this reason, they may lodge in an arteriole at a non-branch point.

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What is the classic presentation?

Multiple bilateral BRAOs occurring at non-branch points

Is it common, or rare?

Rare

In terms of age and gender, who is the classic patient?

A young-adult (15-40 or so) female

Susac syndrome has two classic nonophthalmic manifestations— what are they?

--Sensorineural hearing loss

--Encephalopathy
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Embolization of retinal arterial tree
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Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

- Hypoperfusion of the retina and/or optic nerve

**Vascular**

**Orbital**

- GCA
- Diffuse ocular hypoperfusion
  - OIS
  - Stenosis of the great vessels
- Embolization of retinal arterial tree
- Occlusion in the retinal venous system
- Vasospasm of the CRA

**Ocular**

- What are the three types of embolus?
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--Hearing loss
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Susac syndrome is aka SICRET syndrome, with SICRET being an acronym. What does the acronym stand for?

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--T: Tissue (same)
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For more on Susac syndrome, see slide-set R68

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What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

- Hypoperfusion of the retina and/or optic nerve

**GCA**

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**What are the three types of embolus?**

--Cholesterol

--Calcium

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---

Let’s keep it 💯 for a minute. Clinically speaking, an embolus is an embolus, isn’t it? Other than as a topic for torturing residents, does it really matter what sort of embolus is involved?

---

Let’s keep it 💯 for a minute. Clinically speaking, an embolus is an embolus, isn’t it? Other than as a topic for torturing residents, does it really matter what sort of embolus is involved? Because medical management depends upon the sort of embolus one is dealing with. In this regard, the Neurobook focuses on cholesterol and platelet-fibrin emboli:

--Cholesterol emboli, which most commonly arise from carotid dz, are managed with antiplatelet agents. In addition, vasculopathic risk factors must be optimized, and consideration given to whether CEA is indicated.

--Platelet-fibrin emboli, which usually arise from thrombi owing to AFib, are managed with warfarin and/or the so-called direct oral anticoagulants (DOACs)
Let’s keep it 🏆 for a minute. Clinically speaking, an embolus is an embolus, isn’t it? Other than as a topic for torturing residents, does it really matter what sort of embolus is involved?

It actually does.
Let’s keep it 100 for a minute. Clinically speaking, an embolus is an embolus, isn’t it? Other than as a topic for torturing residents, does it really matter what sort of embolus is involved? Why?

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--Platelet-fibrin emboli
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

- Hypoperfusion of the retina and/or optic nerve
- GCA
- Diffuse ocular hypoperfusion
- OIS
- Stenosis of the great vessels
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- Cholesterol emboli, which most commonly arise from carotid dz, are managed with antiplatelet agents.

The book mentions several antiplatelet agents by name— which ones?

- Aspirin + dipyridamole
- ?
- ?
- ?

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--Cholesterol emboli, which most commonly arise from carotid dz, are managed with antiplatelet agents.
The book mentions several antiplatelet agents by name— which ones?
--Aspirin
--Aspirin + dipyridamole
--Clopidogrel

What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)
Hypoperfusion of...the retina and/or optic nerve
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What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

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It actually does. Because medical management depends upon the sort of embolus one is dealing with. In this regard, the *Neuro* book focuses on cholesterol and platelet-fibrin emboli:

-- Cholesterol emboli, which most commonly arise from carotid dz, are managed with antiplatelet agents. **In addition, vasculopathic risk factors must be optimized, and consideration given to whether CEA is indicated.**

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Let’s keep it 100 for a minute. Clinically speaking, an embolus is an embolus, isn’t it? Other than as a topic for torturing residents, does it really matter what sort of embolus is involved? What’s the point?

It actually does. Because medical management depends upon the sort of embolus one is dealing with. In this regard, the Neuro book focuses on cholesterol and platelet-fibrin emboli:

--Cholesterol emboli, which most commonly arise from carotid disease, are managed with antiplatelet agents. In addition, vasculopathic risk factors must be optimized, and consideration given to whether CEA is indicated.

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The book focuses on several DOACs—which ones?
--?
--?
--?

What advantages do the DOACs enjoy c/w warfarin?
--Better safety profile
--Do not require routine monitoring

What is the main disadvantage of the DOACs c/w warfarin?
Cost

Embolization of retinal arterial tree
Occlusion in the retinal venous system
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What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

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The book focuses on several DOACs—which ones?

--Apixaban

--Rivaroxaban

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Let’s keep it 100 for a minute. Clinically speaking, an embolus is an embolus, isn’t it? Other than as a topic for torturing residents, does it really matter what sort of embolus is involved? We’ll find out.

It actually does. Because medical management depends upon the sort of embolus one is dealing with. In particular, cholesterol and platelet-fibrin emboli are discussed in the book.

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are managed with antiplatelet agents. In addition, vasculopathic risk factors must be optimized, and consideration given to whether CEA is indicated.
--Platelet-fibrin emboli, which usually arise from thrombi owing to AFib
are managed with warfarin and/or the so-called direct oral anticoagulants (DOACs)

The book focuses on several DOACs—which ones?
--Apixaban
--Rivaroxaban
--Dabigatran

What advantages do the DOACs enjoy c/w warfarin?
--Better safety profile
--Do not require routine monitoring

What is the main disadvantage of the DOACs c/w warfarin?
Cost
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

- Hypoperfusion of the retina and/or optic nerve
- GCA
- Diffuse ocular hypoperfusion
- --OIS
- --Stenosis of the great vessels
- Embolization of retinal arterial tree
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Finding that you’ve ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low.

In a nutshell, what is the goal of the workup?
To identify the embolic source

Who should do the workup?
Ideally an ER-affiliated Stroke Center; otherwise, the ER

Who should not do the workup?
You, and/or the pt’s PCP via an outpt referral. Remember, a retinal TIA is a TIA.

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- Embolization of retinal arterial tree
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**Q/A**

In a pt with TMVL, what should push you to suspect an embolic cause? Finding that you’ve ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low.

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In a nutshell, what is the goal of the workup?

Identify the embolic source.

How does one go about identifying the embolic source?

Embolization of retinal arterial tree
- Occlusion in the retinal venous system
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You, and/or the pt’s PCP via an outpatient referral. Remember, a retinal TIA is a TIA. If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.
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How does one go about identifying the embolic source? Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found...

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Before we get to the w/u, let’s hit a quick review

- Embolization of retinal arterial tree
- Occlusion in the retinal venous system
- Vasospasm of the CRA
In a pt with TVL, what should push you to suspect an embolic cause? Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low.

OK, so I've concluded the acute case of TVL sitting in front of me is likely embolic in origin. What now?

Well, they need a workup.

In a nutshell, what is the goal of the workup?

To identify the embolic source.

Who should do the workup?

Ideally an ER-affiliated Stroke Center; otherwise, the ER.

Who should not do the workup?

You, and/or the pt's PCP via an outpt referral. Remember, a retinal TIA is a TIA.

If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.

How does one go about identifying the embolic source?

Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the pathology might be found.

Platelet-fibrin emboli are suggestive of thrombotic dz of the atria in AFib.

Cholesterol emboli are suggestive of atherosomatous dz of the carotids.

Calcium emboli are suggestive of calcific dz of the heart valves.

Before we get to the w/u, let's hit a quick review.

The three types of emboli are:

- Embolization of retinal arterial tree
- Occlusion in the retinal venous system
- Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

Diffuse ocular hypoperfusion -- OIS

-- Stenosis of the great vessels

-- Occlusion in the retinal venous system

-- Vasospasm of the CRA

In a pt with TMVL, what should push you to suspect an embolic cause?

Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low.

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

Well, they need a workup.

In a nutshell, what is the goal of the workup?

To identify the embolic source.

Who should do the workup?

Ideally an ER-affiliated Stroke Center; otherwise, the ER.

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You, and/or the pt's PCP via an outpt referral. Remember, a retinal TIA is a TIA.

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Embolization of retinal arterial tree

Occlusion in the retinal venous system

Vasospasm of the CRA

Before we get to the w/u, let’s hit a quick review

The three types of emboli are:

- Cholesterol emboli
- Platelet-fibrin emboli
- Calcium emboli

How does one go about identifying the embolic source?

Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…

- Platelet-fibrin emboli are suggestive of thrombotic dz of…
- Cholesterol emboli are suggestive of atheromatous dz of…
- Calcium emboli are suggestive of calcific dz of…

- Carotid auscultation
- Carotid imaging
- Check the pt’s pulse
- Cardiac auscultation
- EKG
- Holter study
- Echo
- Echo
The ultimate cause of TMVL is often related to vascular issues. Specifically, hypoperfusion of the retina and/or optic nerve is a common factor.

Findings that rule out ocular and orbital causes, along with a conclusion that GCA is unlikely, suggest an embolic origin for TMVL.

In a patient with TMVL, suspect an embolic cause if you have ruled out ocular and orbital causes and concluded that the likelihood of GCA is low.

In the acute case of TMVL, the workup is needed to identify the embolic source. Ideally, the workup is performed at an ER-affiliated Stroke Center; otherwise, it can be done in the ER.

Who should not perform the workup? You, and/or the patient's PCP via an outpatient referral. Remember, a retinal TIA is a TIA.

If a patient reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.

How does one go about identifying the embolic source? Each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found.

**Cholesterol emboli** are suggestive of:
- **dz**, not **location**

**Platelet-fibrin emboli** are suggestive of:
- **Calcium emboli** are suggestive of:
- **Cholesterol emboli** are suggestive of:
- **Thrombotic dz of...**
- **Atherosomatous dz of...**
- **Calcific dz of...**

Before we get to the w/u, let’s hit a quick review:

- Embolization of retinal arterial tree
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What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

GCA

Diffuse ocular hypoperfusion -- OIS

-- Stenosis of the great vessels

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In a pt with TMVL, what should push you to suspect an embolic cause?

Finding that you’ve ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low

OK, so I’ve concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

Well, they need a workup

In a nutshell, what is the goal of the workup?

To identify the embolic source

Who should do the workup?

Ideally an ER-affiliated Stroke Center; otherwise, the ER

Who should not do the workup?

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Embolization of retinal arterial tree

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Before we get to the w/u, let’s hit a quick review

Cholesterol emboli are suggestive of:

Platelet-fibrin emboli

Calcium emboli

identify the embolic source

How does one go about identifying the embolic source?

Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the pathology might be found…

Cholesterol emboli are suggestive of:

-- atheromatous dz

Platelet-fibrin emboli

Calcium emboli

-- Carotid auscultation

-- Carotid imaging

-- Check the pt’s pulse

-- Cardiac auscultation

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are suggestive of…thrombotic dz of…

Cholesterol emboli

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Calcium emboli

are suggestive of…calcific dz of…

Platelet-fibrin emboli

--Carotid auscultation

--Carotid imaging

--Check the pt's pulse

--Cardiac auscultation

--EKG

--Holter study

--Echo

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Cholesterol emboli implicated in TMVL are most likely to have originated in atheromatous plaques of:

identify the embolic source
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

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To identify the embolic source

Who should do the workup?

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*But shout-out the aortic arch as well, remember
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

GCA

Diffuse ocular hypoperfusion -- OIS

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- Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…

- **Platelet-fibrin emboli** are suggestive of:
  - not location

**Before we get to the w/u, let's hit a quick review**

- **Cholesterol emboli** are suggestive of…
  - atheromatous dz of…
  - the carotids

- **Calcium emboli** are suggestive of…
  - calcific dz of…
  - the heart valves

- **Embolization of retinal arterial tree**
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In a pt with TMVL, what should push you to suspect an embolic cause?

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- Check the pt’s pulse
- Cardiac auscultation
- EKG
- Holter study
- Echo

Cholesterol emboli are suggestive of:

- Atheromatous dz of the carotids
- Carotid auscultation
- Carotid imaging

Calcium emboli are suggestive of:

- Calcific dz of the heart valves
- Echo

Before we get to the w/u, let’s hit a quick review:

Embolization of retinal arterial tree

- Occlusion in the retinal venous system
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What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

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Platelet-fibrin emboli implicated in TMVL are most likely to have originated in:

- Platelet-fibrin emboli
- Embolization of retinal arterial tree
- Occlusion in the retinal venous system
- Vasospasm of the CRA

Cholesterol emboli are suggestive of…atheromatous dz of… the carotids

Platelet-fibrin emboli are suggestive of…thrombotic dz of…

Calcium emboli are suggestive of…calcific dz of… the heart valves

Platelet-fibrin emboli implicated in TMVL are most likely to have originated in:

structure and dz condition
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

Vascular Orbital

In a pt with TMVL, what should push you to suspect an embolic cause?

Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low

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If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.

Embolization of retinal arterial tree

Occlusion in the retinal venous system

Vasospasm of the CRA

Platelet-fibrin emboli implicated in TMVL are most likely to have originated in:

Platelet-fibrin emboli are suggestive of…

atheromatous dz of…

the carotids

Platelet-fibrin emboli are suggestive of…

thrombotic dz of…

the atria in AFib

Calcium emboli are suggestive of…

calcific dz of…

the heart valves

Cholesterol emboli are suggestive of…

atheromatous dz of…

the carotids

Before we get to the w/u, let’s hit a quick review
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve.

In a pt with TMVL, what should push you to suspect an embolic cause?

Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low.

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

Well, they need a workup.

In a nutshell, what is the goal of the workup?

To identify the embolic source.

Who should do the workup?

Ideally an ER-affiliated Stroke Center; otherwise, the ER.

Who should not do the workup?

You, and/or the pt's PCP via an outpt referral. Remember, a retinal TIA is a TIA.

If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.

Embolization of retinal arterial tree

- Occlusion in the retinal venous system
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Cholesterol emboli are suggestive of... atheromatous dz of... the carotids

Platelet-fibrin emboli are suggestive of... thrombotic dz of... the atria in AFib

Calcium emboli are suggestive of...

Calcific emboli are suggestive of:
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.) Hypoperfusion of… the retina and/or optic nerve

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How does one go about identifying the embolic source? Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…

Cholesterol emboli are suggestive of… atheromatous dz of… the carotids

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Before we get to the w/u, let's hit a quick review.
In a nutshell, what is the goal of the workup? To identify the embolic source.

Who should do the workup? Ideally an ER-affiliated Stroke Center; otherwise, the ER. Who should not do the workup? You, and/or the pt's PCP via an outpatient referral. Remember, a retinal TIA is a TIA.

How does one go about identifying the embolic source? Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found.

- **Cholesterol emboli** are suggestive of... atheromatous dz of... the carotids
- **Platelet-fibrin emboli** are suggestive of... thrombotic dz of... the atria in AFib
- **Calcium emboli** are suggestive of... calcific dz of... the heart valves

Calcium emboli implicated in TMVL are most likely to have originated in:

**Before we get to the w/u, let’s hit a quick review**

- Embolization of retinal arterial tree
  - Occlusion in the retinal venous system
  - Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

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Before we get to the w/u, let’s hit a quick review

Embolization of retinal arterial tree

- Occlusion in the retinal venous system
- Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

OK, so now that we've ruled out ocular and orbital causes, and concluded that the likelihood of GCA is low...

Well, I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What's next?

They need a workup

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Now, the workup. What tests/exam maneuvers should be performed?

---

Embolization of retinal arterial tree

---

Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? Looking for a single word here.

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---

Now, the workup. What tests/exam maneuvers should be performed?

--Check the pt's pulse
--Carotid auscultation
--Cardiac auscultation
--Carotid imaging
--Echocardiography
--EKG
--Holter study

---

Embolization of retinal arterial tree

Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

GCA

Diffuse ocular hypoperfusion -- OIS -- Stenosis of the great vessels

Occlusion in the retinal venous system

Vasospasm of the CRA

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Calcium emboli are suggestive of… calcific dz of… the heart valves

Now, the workup. What tests/exam maneuvers should be performed?

-- Check the pt’s pulse
-- Carotid auscultation
-- Cardiac auscultation
-- Carotid imaging
-- Echocardiography
-- EKG
-- Holter study

Note: Some of you no doubt came up with a study that’s not on this list. If so, no worries—we’ll address that study shortly.

Another very important study
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**Calcium emboli**
are suggestive of... calcific dz of... the heart valves.

Now, the workup. What tests/exam maneuvers should be performed?

For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

--Check the pt’s pulse?

--Carotid auscultation

--Cardiac auscultation

--Carotid imaging

--Echocardiography

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Embolization of retinal arterial tree

Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

GCA

Diffuse ocular hypoperfusion

--OIS

--Stenosis of the great vessels

Occlusion in the retinal venous system

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**Platelet-fibrin emboli**

are suggestive of…thrombotic dz of…the atria in AFib

---Check the pt’s pulse

**Calcium emboli**

are suggestive of…calcific dz of…the heart valves

Now, the workup. What tests/exam maneuvers should be performed?

For each, indicate the type(s) of emboli for which the test has direct bearing *vis a vis* making a diagnosis:

--Check the pt’s pulse

--Carotid auscultation

--Cardiac auscultation

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--Echocardiography

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--Holter study

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the atria in AFib

Cholesterol emboli are suggestive of…atheromatous dz of…

the carotids

Calcium emboli are suggestive of…calcific dz of…

the heart valves

Now, the workup. What tests/exam maneuvers should be performed? For each, indicate the type(s) of emboli for which the test has direct bearing via a vis making a diagnosis:

--- Check the pt’s pulse

--- Carotid auscultation

--- Cardiac auscultation

--- Carotid imaging

--- Echocardiography

--- EKG

--- Holter study

--- Platelet-fibrin emboli

--- Embolization of retinal arterial tree

--- Check the pt’s pulse

What are you looking for via a pulse check?

An irregularly irregular rhythm, which would increase (although not cinch) the likelihood that AFib is present
**Cholesterol emboli** are suggestive of... atheromatous dz of... the carotids 

**Platelet-fibrin emboli** are suggestive of... thrombotic dz of... the atria in AFib

**Calcium emboli** are suggestive of... calcific dz of... the heart valves

**What are you looking for via a pulse check?**
An irregularly irregular rhythm, which would increase (although not cinch) the likelihood that AFib is present

**How does one go about identifying the embolic source?**
Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found...

**Identify the embolic source**

Now, the workup. What tests/exam maneuvers should be performed? For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

--Check the pt’s pulse

--Carotid auscultation

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**Embolization of retinal arterial tree**

**Vasospasm of the CRA**
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For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

--Check the pt's pulse
--Carotid auscultation?
--Cardiac auscultation
--Carotid imaging
--Echocardiography
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--Holter study

Embolization of retinal arterial tree
What is the ultimate cause of TVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

GCA

Diffuse ocular hypoperfusion

--OIS

--Stenosis of the great vessels

Occlusion in the retinal venous system

Vasospasm of the CRA

In a pt with TVL, what should push you to suspect an embolic cause?

Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low

OK, so I've concluded the acute case of TVL sitting in front of me is likely embolic in origin. What now?

Well, they need a workup

In a nutshell, what is the goal of the workup?

To identify the embolic source

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Ideally an ER-affiliated Stroke Center; otherwise, the ER

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**Platelet-fibrin emboli** are suggestive of…thrombotic dz of…the atria in AFib

--Check the pt’s pulse

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Now, the workup. What tests/exam maneuvers should be performed?

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Embolization of retinal arterial tree
In a pt with TMVL, what should push you to suspect an embolic cause?
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  --Check the pt's pulse

- **Calcium emboli**
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Now, the workup. What tests/exam maneuvers should be performed?
--Check the pt's pulse
--Carotid auscultation
--Cardiac auscultation
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What are you assessing for via carotid auscultation?

Emboli of retinal arterial tree
Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

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Now, the workup. What tests/exam maneuvers should be performed?

For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

--Check the pt's pulse

--Carotid auscultation

What are you assessing for via carotid auscultation?
The presence of a [bruit](#)

Where (ie, at what anatomic landmark) should auscultation be performed?
The angle of the jaw (is the location of the carotid bifurcation)

If no bruit is present, is a carotid embolic source ruled out?
No! Remember, a bruit won't be present if the artery is either minimally occluded or totally occluded.

---

**Embolization of retinal arterial tree**

--Carotid auscultation

--Cardiac auscultation

--Carotid imaging

--Echocardiography

--EKG

--Holter study
What is the ultimate cause of TMVL related to the vascular? (Looking for a single word here.)

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**Carotid auscultation**

--Cardiac auscultation
--Carotid imaging
--Echocardiography
--EKG
--Holter study

**Embolization of retinal arterial tree**

--Check the pt’s pulse

---

**Vasospasm of the CRA**
In a patient presenting with TVL, what should push you to suspect an embolic cause?

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If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.

How does one go about identifying the embolic source?

Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…

Cholesterol emboli are suggestive of…atheromatous dz of…the carotids.

Platelet-fibrin emboli are suggestive of…thrombotic dz of…the atria in AFib.

Calcium emboli are suggestive of…calcific dz of…the heart valves.

Now, the workup. What tests/exam maneuvers should be performed?

For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

- Check the pt’s pulse.
- Carotid auscultation.
- Cardiac auscultation.
- Carotid imaging.
- Echocardiography.
- EKG.
- Holter study.

Embolization of retinal arterial tree.

What are you assessing for via carotid auscultation?
The presence of a bruit.

Where (ie, at what anatomic landmark) should auscultation be performed?

The angle of the jaw (is the location of the carotid bifurcation).

If no bruit is present, is a carotid embolic source ruled out?

No! Remember, a bruit won’t be present if the artery is either minimally occluded or totally occluded.
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

In a pt with TMVL, what should push you to suspect an embolic cause?

Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

Well, they need a workup

In a nutshell, what is the goal of the workup?

To identify the embolic source

Who should do the workup?

Ideally an ER-affiliated Stroke Center; otherwise, the ER

Who should not do the workup?

You, and/or the pt's PCP via an outpt referral. Remember, a retinal TIA is a TIA.

If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.

How does one go about identifying the embolic source?

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  **Carotid auscultation**

- **Platelet-fibrin emboli** are suggestive of...thrombotic dz of...the atra in AFib

  **Check the pt's pulse**

- **Calcium emboli** are suggestive of...calcific dz of...the heart valves

What are you assessing for via carotid auscultation?

The presence of a bruit

Where (ie, at what anatomic landmark) should auscultation be performed?

The angle of the jaw (is the location of the carotid...)

---

--**Carotid auscultation**

--Cardiac auscultation
--Carotid imaging
--Echocardiography
--EKG
--Holter study

Embolization of retinal arterial tree

---

Vasospasm of the CRA
In a pt with TMVL, what should push you to suspect an embolic cause?

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OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

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  --Carotid auscultation

- **Platelet-fibrin emboli** are suggestive of... thrombotic dz of... the atria in AFib.
  --Check the pt's pulse

- **Calcium emboli** are suggestive of... calcific dz of... the heart valves.

Now, the workup. What tests/exam maneuvers should be performed?

For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

- Check the pt's pulse
  --Carotid auscultation

Where (ie, at what anatomic landmark) should auscultation be performed?

The angle of the jaw (is the location of the carotid bifurcation).

What are you assessing for via carotid auscultation?

The presence of a bruit.

If no bruit is present, is a carotid embolic source ruled out?

No! Remember, a bruit won't be present if the artery is either minimally occluded or totally occluded.
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

In a pt with TMVL, what should push you to suspect an embolic cause?

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Platelet-fibrin emboli are suggestive of…thrombotic dz of…the atra in AFib

--Check the pt’s pulse

Calcium emboli are suggestive of…calcific dz of…the heart valves

Embolization of retinal arterial tree

What are you assessing for via carotid auscultation?
The presence of a bruit

Where (ie, at what anatomic landmark) should auscultation be performed?
The angle of the jaw (is the location of the carotid bifurcation)

If no bruit is present, is a carotid embolic source ruled out?

No! Remember, a bruit won’t be present if the artery is either minimally occluded or totally occluded.

For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

--Check the pt’s pulse  Platelet-fibrin emboli
--Carotid auscultation  Cholesterol emboli
--Cardiac auscultation  Calcium emboli
--Carotid imaging  Embolization of retinal arterial tree
--Echocardiography  --EKG
--Holter study  --Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

GCA

Diffuse ocular hypoperfusion

--OIS

--Stenosis of the great vessels

Occlusion in the retinal venous system

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- **Carotid auscultation**

- **Check the pt's pulse**

---

**Platelet-fibrin emboli** are suggestive of... thrombotic dz of... the atria in AFib

- **Cardiac auscultation**

---

**Calcium emboli** are suggestive of... calcific dz of... the heart valves

---

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Now, the workup. What tests/exam maneuvers should be performed?

For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

- Check the pt's pulse
- Carotid auscultation
- Cardiac auscultation?
- Carotid imaging
- Echocardiography
- EKG
- Holter study

Embolization of retinal arterial tree

Cholesterol emboli are suggestive of atheromatous dz of the carotids

- Carotid auscultation

Platelet-fibrin emboli are suggestive of thrombotic dz of the atria in AFib

- Check the pt's pulse

Calcium emboli are suggestive of calcific dz of the heart valves

- Carotid auscultation

How does one go about identifying the embolic source?

Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…
In a pt with TMVL, what should push you to suspect an embolic cause? Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low. OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now? Well, they need a workup. In a nutshell, what is the goal of the workup? Identify the embolic source.

How does one go about identifying the embolic source? Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…

- **Cholesterol emboli** are suggestive of atheromatous dz of the carotids
  - Check the pt’s pulse
  - Carotid auscultation

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  - Check the pt’s pulse
  - Cardiac auscultation

- **Calcium emboli** are suggestive of calcific dz of... the heart valves
  - Cardiac auscultation

Now, the workup. What tests/exam maneuvers should be performed? For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

- Check the pt’s pulse
- Carotid auscultation
- Cardiac auscultation
- Carotid imaging
- Echocardiography
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- Holter study
- Embolization of retinal arterial tree
- Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

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Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…

Platelet-fibrin emboli are suggestive of…
- thrombotic dz of…
- the atria in AFib

Stenosis of the great vessels

Occlusion in the retinal venous system

Vasospasm of the CRA

Calcium emboli are suggestive of…
- calcific dz of…
- the heart valves

Cholesterol emboli are suggestive of…
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- the carotids

Now, the workup. What tests/exam maneuvers should be performed?
For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:
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What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

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GCA

Diffuse ocular hypoperfusion

--OIS

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Platelet-fibrin emboli are suggestive of...

--Thrombotic dz of...

the atria in AFib

Cholesterol emboli are suggestive of...

--Atheromatous dz of...

the carotids

Calcium emboli are suggestive of...

--Calcific dz of...

the heart valves

Now, the workup. What tests/exam maneuvers should be performed? For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

--Check the pt’s pulse

--Cardiac auscultation

--Carotid imaging

--Echocardiography

--EKG

--Holter study

--Check the pt’s pulse

--Cardiac auscultation

--Carotid imaging
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve.

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How does one go about identifying the embolic source?

Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…

- **Cholesterol emboli** are suggestive of…atheromatous dz of…the carotids.
  - --Carotid auscultation
  - --Carotid imaging

- **Platelet-fibrin emboli** are suggestive of…thrombotic dz of…the atria in AFib.
  - --Check the pt’s pulse
  - --Cardiac auscultation

- **Calcium emboli** are suggestive of…calcific dz of…the heart valves.
  - --Cardiac auscultation

How (ie, via what modality/modalities) should the carotids be imaged?

For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

- Check the pt’s pulse
- --Carotid auscultation
- --Cardiac auscultation
- --Carotid imaging

--Echocardiography
--EKG
--Holter study

How (ie, via what modality/modalities) should the carotids be imaged?

Ultrasound, MRA and CTA are all acceptable (although the Neurobook indicates MRA/CTA are preferred).

Embolization of retinal arterial tree:

- --Check the pt’s pulse
- --Carotid auscultation
- --Cardiac auscultation
- --Carotid imaging

--Echocardiography
--EKG
--Holter study
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

- Hypoperfusion of the retina and/or optic nerve
- Diffuse ocular hypoperfusion --OIS
- Stenosis of the great vessels
- Occlusion in the retinal venous system
- Vasospasm of the CRA

In a pt with TMVL, what should push you to suspect an embolic cause?

Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low.

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

Well, they need a workup.

In a nutshell, what is the goal of the workup?

To identify the embolic source.

Who should do the workup?

Ideally an ER-affiliated Stroke Center; otherwise, the ER.

Who should not do the workup?

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How does one go about identifying the embolic source?

Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found...

- **Cholesterol emboli**
  - are suggestive of... atheromatous dz of... the carotids
  - Carotid auscultation
  - Carotid imaging

- **Platelet-fibrin emboli**
  - are suggestive of... thrombotic dz of... the atria in AFib
  - Check the pt's pulse
  - Cardiac auscultation

- **Calcium emboli**
  - are suggestive of... calcific dz of... the heart valves
  - Cardiac auscultation

How (ie, via what modality/modalities) should the carotids be imaged?

Ultrasound, MRA and CTA are all acceptable (although the *Neuro* book indicates MRA/CTA are preferred).

How do you do it? What tests/exam maneuvers should be performed?

For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

- **Check the pt's pulse**
- **Carotid auscultation**
- **Cardiac auscultation**
- **Carotid imaging**
- Echocardiography
- EKG
- Holter study

Embolization of retinal arterial tree...
In a pt with TMVL, what should push you to suspect an embolic cause?

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Well, they need a workup.

In a nutshell, what is the goal of the workup?

To identify the embolic source.

Who should do the workup?

Ideally an ER-affiliated Stroke Center; otherwise, the ER.

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--Check the pt’s pulse
--Carotid auscultation
--Carotid imaging

Platelet-fibrin emboli are suggestive of… thrombotic dz of… the atria in AFib

--Check the pt’s pulse
--Cardiac auscultation

Calcium emboli are suggestive of… calcific dz of… the heart valves

Now, the workup. What tests/exam maneuvers should be performed?

For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

--Check the pt’s pulse
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Embolization of retinal arterial tree
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--Carotid auscultation
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--Check the pt’s pulse
--Cardiac auscultation
--Echo

Calcium emboli are suggestive of…calcific dz of…the heart valves

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Now, the workup. What tests/exam maneuvers should be performed?
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--Check the pt’s pulse
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--Cardiac auscultation
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--Echocardiography
--EKG
--Holter study

Embolization of retinal arterial tree

--Carotid auscultation
--Carotid imaging
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--Cardiac auscultation
--Echo
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve.

GCA

Diffuse ocular hypoperfusion --OIS --Stenosis of the great vessels

Occlusion in the retinal venous system

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**Cholesterol emboli** are suggestive of...

- atheromatous dz of...

- the carotids

**Calcium emboli** are suggestive of...

- calcific dz of...

- the heart valves

**Emboli of retinal arterial tree**

- Carotid auscultation

- Cardiac auscultation

- Check the pt's pulse

- Carotid imaging

- Echocardiography

- Holter study

- EKG

- Holter study

Now, the workup. What tests/exam maneuvers should be performed?

For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

- Check the pt's pulse

- Carotid auscultation

- Cardiac auscultation

- Carotid imaging

- Echocardiography

- EKG

- Holter study

What are you looking for on echo vis a vis working up platelet-fibrin emboli?

Well, it would certainly cinch the diagnosis if an intra-atrial clot was observed...

Other than that, the main concern is for an atrial septal wall defect that would allow an embolus originating on the venous side of the circulatory system to gain access to the arterial side...
In a pt with TMVL, what should push you to suspect an embolic cause? Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low.

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--- Carotid auscultation
--- Carotid imaging

**Platelet-fibrin emboli** are suggestive of… thrombotic dz of… the atria in AFib.

--- Check the pt's pulse
--- Cardiac auscultation
--- Echo

**Calcium emboli** are suggestive of… calcific dz of… the heart valves.

--- Cardiac auscultation
--- Echo

What are you looking for on echo vis a vis working up platelet-fibrin emboli?

Well, it would certainly cinch the diagnosis if an intra-atrial clot was observed…

How does one go about making a diagnosis?

--- Check the pt's pulse
--- Carotid auscultation
--- Cardiac auscultation
--- Carotid imaging
--- Echocardiography
--- EKG
--- Holter study

Embolization of retinal arterial tree

--- Carotid auscultation
--- Carotid imaging
--- Check the pt's pulse
--- Cardiac auscultation
--- Echo

--- Cardiac auscultation
--- Echo
Cholesterol emboli are suggestive of…
atheromatous dz of…
the carotids

- Carotid auscultation
- Carotid imaging

Platelet-fibrin emboli are suggestive of…
thrombotic dz of…
the atria in AFib

- Check the pt’s pulse
- Cardiac auscultation
- Echocardiography

Calcium emboli are suggestive of…
calcific dz of…
the heart valves

- Cardiac auscultation
- Echo

What are you looking for on echo vis a vis working up platelet-fibrin emboli?
Well, it would certainly cinch the diagnosis if an intra-atrial clot was observed…

Other than that, the main concern is for an atrial septal wall defect that would allow an embolus originating on the venous side of the circulatory system to gain access to the arterial side.

---

Embolization of retinal arterial tree

- Carotid auscultation
- Carotid imaging
- Echocardiography
- EKG
- Holter study
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

GCA

Diffuse ocular hypoperfusion

--OIS

--Stenosis of the great vessels

Occlusion in the retinal venous system

Vasospasm of the CRA

In a pt with TMVL, what should push you to suspect an embolic cause?

Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

Well, they need a workup

In a nutshell, what is the goal of the workup?

To identify the embolic source

Who should do the workup?

Ideally an ER-affiliated Stroke Center; otherwise, the ER

Who should not do the workup?

You, and/or the pt's PCP via an outpt referral. Remember, a retinal TIA is a TIA.

If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.

How does one go about identifying the embolic source?

Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…

**Platelet-fibrin emboli**

are suggestive of… thrombotic dz of…

the atria in AFib

--Check the pt's pulse

--Cardiac auscultation

--Echo

**Cholesterol emboli**

are suggestive of… atheromatous dz of…

the carotids

--Carotid auscultation

--Carotid imaging

**Calcium emboli**

are suggestive of… calcific dz of…

the heart valves

--Cardiac auscultation

--Echo

What are you looking for on echo vis a vis working up platelet-fibrin emboli?

Well, it would certainly cinch the diagnosis if an intra-atrial clot was observed…

Other than that, the main concern is for an atrial septal wall defect that would allow an embolus originating on the venous side of the circulatory system to gain access to the arterial side.

bearing vis a vis making a diagnosis.

--Check the pt's pulse

--Carotid auscultation

--Cardiac auscultation

--Carotid imaging

--Echocardiography

--EKG

--Holter study

Embolization of retinal arterial tree

--- Vasospasm of the CRA

--- Plaque rupture

--- Cholesterol emboli

--- Platelet-fibrin emboli

--- Calcification emboli

--- GCA

--- Group B strep
In a patient with suspected TMVL, the ultimate cause related to the vasculature is likely hypoperfusion of the retina and/or optic nerve. Various causes of diffuse ocular hypoperfusion include stenosis of the great vessels, occlusion in the retinal venous system, and vasospasm of the CRA.

In a pt with TMVL, what should push you to suspect an embolic cause? Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low.

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now? Well, they need a workup.

In a nutshell, what is the goal of the workup? To identify the embolic source.

Who should do the workup? Ideally an ER-affiliated Stroke Center; otherwise, the ER. Who should not do the workup? You, and/or the pt's PCP via an outpt referral. Remember, a retinal TIA is a TIA.

If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.

How does one go about identifying the embolic source? Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found...

**Cholesterol emboli** are suggestive of... atheromatous dz of... the carotids.

- Carotid auscultation
- Carotid imaging

**Platelet-fibrin emboli** are suggestive of... thrombotic dz of... the atria in AFib.

- Check the pt’s pulse
- Cardiac auscultation
- Echo

**Calcium emboli** are suggestive of... calcific dz of... the heart valves.

- Cardiac auscultation
- Echo

Now, the workup. What tests/exam maneuvers should be performed? For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

- Check the pt’s pulse
- Carotid auscultation
- Carotid imaging
- Echocardiography
- EKG
- Holter study

What are you looking for on echo in this regard? Evidence of cardiac valvular dz. This exam goal may necessitate a modification in echo technique. What mod? Employing a transesophageal (TEE) approach rather than the more commonly-employed transthoracic (TTE) approach. Why might a TEE be indicated? Because TTE doesn’t visualize the cardiac valves well.
In a pt with TMVL, what should push you to suspect an embolic cause?

Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low.

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

Well, they need a workup.

In a nutshell, what is the goal of the workup?

To identify the embolic source.

Who should do the workup?

Ideally an ER-affiliated Stroke Center; otherwise, the ER.

Who should not do the workup?

You, and/or the pt's PCP via an outpt referral. Remember, a retinal TIA is a TIA.

If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.

How does one go about identifying the embolic source?

Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…

Cholesterol emboli are suggestive of…atheromatous dz of… the carotids

--Carotid auscultation
--Carotid imaging

Platelet-fibrin emboli are suggestive of…thrombotic dz of… the atria in AFib

--Check the pt's pulse
--Cardiac auscultation
--Echo

Calcium emboli are suggestive of…calcific dz of… the heart valves

--Cardiac auscultation
--Echo

Now, the workup.

For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

--Check the pt's pulse
--Carotid auscultation
--Cardiac auscultation
--Carotid imaging
--Echocardiography
--EKG
--Holter study

What are you looking for on echo in this regard?

Evidence of cardiac valvular dz

Embolization of retinal arterial tree

--Carotid auscultation
--Carotid imaging
--Check the pt's pulse
--Cardiac auscultation
--Echo

What mod?

Employing a transesophageal (TEE) approach rather than the more commonly-employed transthoracic (TTE) approach.

Why might a TEE be indicated?

Because TTE doesn't visualize the cardiac valves well.
In a pt with TMVL, what should push you to suspect an embolic cause?
Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low
OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?
Well, they need a workup
In a nutshell, what is the goal of the workup?
To identify the embolic source
Who should do the workup?
Ideally an ER-affiliated Stroke Center; otherwise, the ER
Who should not do the workup?
You, and/or the pt's PCP via an outpt referral. Remember, a retinal TIA is a TIA.
If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.
How does one go about identifying the embolic source?
Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…
Cholesterol emboli are suggestive of…atheromatous dz of…the carotids
--Carotid auscultation
--Carotid imaging
Platelet-fibrin emboli are suggestive of…thrombotic dz of…the atria in AFib
--Check the pt’s pulse
--Cardiac auscultation
--Echo
Calcium emboli are suggestive of…calcific dz of…the heart valves
--Cardiac auscultation
--Echo
Now, the workup. What tests/exam maneuvers should be performed? For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:
--Check the pt’s pulse
--Carotid auscultation
--Cardiac auscultation
--Carotid imaging
--Echocardiography
--EKG
--Holter study
Embolization of retinal arterial tree
--Carotid auscultation
--Carotid imaging
--Check the pt’s pulse
--Cardiac auscultation
--Echo
--Echocardiography
What are you looking for on echo in this regard?
Evidence of cardiac valvular dz
This exam goal may necessitate a modification in echo technique. What mod?
Employing a transesophageal (TEE) approach rather than the more commonly-employed transthoracic (TTE) approach
Because TTE doesn’t visualize the cardiac valves well
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

- Hypoperfusion of the retina and/or optic nerve

In a pt with TMVL, what should push you to suspect an embolic cause?

- Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

- They need a workup

In a nutshell, what is the goal of the workup?

- To identify the embolic source

Who should do the workup?

- Ideally an ER-affiliated Stroke Center; otherwise, the ER

Who should not do the workup?

- You, and/or the pt's PCP via an outpt referral. Remember, a retinal TIA is a TIA.

If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.

How does one go about identifying the embolic source?

Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found...

- Platelet-fibrin emboli are suggestive of... those thrombotic dz of...
  - the atria in AFib

- Cholesterol emboli are suggestive of... atheromatous dz of...
  - the carotids

- Calcium emboli are suggestive of... calcific dz of...
  - the heart valves

Now, the workup. What tests/exam maneuvers should be performed?

- Check the pt's pulse
- Carotid auscultation
- Cardiac auscultation
- Carotid imaging
- Echocardiography
- EKG
- Holter study

--Check the pt's pulse
--Carotid auscultation
--Cardiac auscultation
--Carotid imaging
--Echocardiography
--EKG
--Holter study

What are you looking for on echo in this regard?

Evidence of cardiac valvular dz

This exam goal may necessitate a modification in echo technique.

What mod?

Employing a more commonly-employed approach rather than the approach.

This exam goal may necessitate a modification in echo technique.

What mod?

Employing a more commonly-employed approach rather than the approach.

What are you looking for on echo in this regard?

Evidence of cardiac valvular dz

This exam goal may necessitate a modification in echo technique.

What mod?

Employing a more commonly-employed approach rather than the approach.

What are you looking for on echo in this regard?

Evidence of cardiac valvular dz

This exam goal may necessitate a modification in echo technique.

What mod?

Employing a more commonly-employed approach rather than the approach.

What are you looking for on echo in this regard?

Evidence of cardiac valvular dz

This exam goal may necessitate a modification in echo technique.

What mod?

Employing a more commonly-employed approach rather than the approach.

What are you looking for on echo in this regard?

Evidence of cardiac valvular dz

This exam goal may necessitate a modification in echo technique.

What mod?

Employing a more commonly-employed approach rather than the approach.
In a pt with TMVL, what should push you to suspect an embolic cause? Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low.

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now? Well, they need a workup.

In a nutshell, what is the goal of the workup? To identify the embolic source.

Who should do the workup? Ideally an ER-affiliated Stroke Center; otherwise, the ER.

Who should not do the workup? You, and/or the pt's PCP via an outpt referral. Remember, a retinal TIA is a TIA.

If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.

Now, the workup. What tests/exam maneuvers should be performed? For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

- Check the pt's pulse
- Carotid auscultation
- Carotid imaging
- Cardiac auscultation
- Echocardiography

Emboli of retinal arterial tree
- Carotid auscultation
- Carotid imaging
- Check the pt's pulse
- Cardiac auscultation
- Echocardiography
- EKG
- Holter study

What are you looking for on echo in this regard? Evidence of cardiac valvular dz.

This exam goal may necessitate a modification in echo technique. What mod? Employing a transesophageal (TEE) approach rather than the more commonly-employed transthoracic (TTE) approach.

How does one go about identifying the embolic source? Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…
In a patient with TMVL, what might be the ultimate cause related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve.

Ocular vascular disease can lead to diffuse hypoperfusion.

- GCA
- Diffuse ocular hypoperfusion
- Stenosis of the great vessels
- Occlusion in the retinal venous system
- Vasospasm of the CRA

In a pt with TMVL, what should push you to suspect an embolic cause?

Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low.

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

Well, they need a workup.

In a nutshell, what is the goal of the workup?

To identify the embolic source.

Who should do the workup?

Ideally an ER-affiliated Stroke Center; otherwise, the ER.

Who should not do the workup?

You, and/or the pt's PCP via an outpt referral. Remember, a retinal TIA is a TIA.

If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.

How does one go about identifying the embolic source?

Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found...

- **Cholesterol emboli**
  - are suggestive of...atheromatous dz of...the carotids
  - --Carotid auscultation
  - --Carotid imaging

- **Platelet-fibrin emboli**
  - are suggestive of...thrombotic dz of...the atria in AFib
  - --Check the pt’s pulse
  - --Cardiac auscultation
  - --Echo

- **Calcium emboli**
  - are suggestive of...calcific dz of...the heart valves
  - --Cardiac auscultation
  - --Echo

Now, the workup. What tests/exam maneuvers should be performed? For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

- Check the pt’s pulse
- Carotid auscultation
- Cardiac auscultation
- Carotid imaging
- Echocardiography
- EKG
- Holter study

What are you looking for on echo in this regard?

Evidence of cardiac valvular dz.

This exam goal may necessitate a modification in echo technique. What mod?

Employing a transesophageal (TEE) approach rather than the more commonly-employed transthoracic (TTE) approach.

Why might a TEE be indicated?

Because TTE doesn’t visualize the cardiac valves well.
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

- Hypoperfusion of the retina and/or optic nerve
- Diffuse ocular hypoperfusion
- Stenosis of the great vessels
- Occlusion in the retinal venous system
- Vasospasm of the CRA

In a pt with TMVL, what should push you to suspect an embolic cause?

- Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

- They need a workup

In a nutshell, what is the goal of the workup?

- Identify the embolic source

Who should do the workup?

- Ideally an ER-affiliated Stroke Center; otherwise, the ER

Who should not do the workup?

- You, and/or the pt’s PCP via an outpt referral. Remember, a retinal TIA is a TIA.

If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.

How does one go about identifying the embolic source?

- Recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found...

  Cholesterol emboli are suggestive of…atheromatous dz of…the carotids
  - Carotid auscultation
  - Carotid imaging

  Platelet-fibrin emboli are suggestive of…thrombotic dz of…the atria in AFib
  - Check the pt’s pulse
  - Cardiac auscultation
  - Echo

  Calcium emboli are suggestive of…calcific dz of…the heart valves
  - Cardiac auscultation
  - Echo

Now, the workup. What are you looking for on echo in this regard?

- Evidence of cardiac valvular dz

This exam goal may necessitate a modification in echo technique.

- What mod?
- Employing a transesophageal (TEE) approach rather than the more commonly-employed transthoracic (TTE) approach

Why might a TEE be indicated?

- Because TTE doesn’t visualize the cardiac valves well

Embolization of retinal arterial tree

- Check the pt’s pulse
- Carotid auscultation
- Carotid imaging
- Echocardiography
- EKG
- Holter study
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

In a pt with TMVL, what should push you to suspect an embolic cause?

Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

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How does one go about identifying the embolic source?

Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…

Cholesterol emboli are suggestive of… atheromatous dz of… the carotids

Platelet-fibrin emboli are suggestive of… thrombotic dz of… the atria in AFib

Calcium emboli are suggestive of… calcific dz of… the heart valves

---Carotid auscultation
---Carotid imaging
---Check the pt’s pulse
---Cardiac auscultation
---Echo
---Cardiac auscultation
---Echo

What are you looking for on echo in this regard?

Evidence of cardiac valvular dz

This exam goal may necessitate a modification in echo technique.

What mod?

Employing a transesophageal (TEE) approach rather than the more commonly-employed transthoracic (TTE) approach

Why might a TEE be indicated?

Because TTE doesn’t visualize the cardiac valves well

Now, the workup. What are you looking for on echo in this regard?

Embolization of retinal arterial tree

---Carotid auscultation
---Carotid imaging
---Check the pt’s pulse
---Cardiac auscultation
---Echo
In a pt with TMVL, what should push you to suspect an embolic cause?
Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low.

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

Well, they need a workup.

In a nutshell, what is the goal of the workup?
To identify the embolic source.

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Ideally an ER-affiliated Stroke Center; otherwise, the ER.

Who should not do the workup?
You, and/or the pt's PCP via an outpt referral. Remember, a retinal TIA is a TIA.

If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.

How does one go about identifying the embolic source?
Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…

**Cholesterol emboli** are suggestive of… atheromatous dz of… the carotids
- Carotid auscultation
- Carotid imaging

**Platelet-fibrin emboli** are suggestive of… thrombotic dz of… the atria in AFib
- Check the pt's pulse
- Cardiac auscultation
- Echo

**Calcium emboli** are suggestive of… calcific dz of… the heart valves
- Cardiac auscultation
- Echo

Now, the workup. What tests/exam maneuvers should be performed?
For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:
- Check the pt's pulse
- Carotid auscultation
- Cardiac auscultation
- Carotid imaging
- Echocardiography
- EKG?
- Holter study

Embolization of retinal arterial tree

Vasospasm of the CRA
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

Diffuse ocular hypoperfusion --OIS

Stenosis of the great vessels

Occlusion in the retinal venous system

Vasospasm of the CRA

In a pt with TMVL, what should push you to suspect an embolic cause?

Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

Well, they need a workup

In a nutshell, what is the goal of the workup?

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How does one go about identifying the embolic source?

Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found...

Platelet-fibrin emboli are suggestive of... a thrombotic dz of...

Cholesterol emboli are suggestive of... an atheromatous dz of...

Calcium emboli are suggestive of... a calcific dz of...

Embolization of retinal arterial tree

--Carotid auscultation
--Carotid imaging
--Echocardiography
--Check the pt's pulse
--Cardiac auscultation
--Echo
--EKG

--Carotid auscultation
--Carotid imaging
--Check the pt's pulse
--Cardiac auscultation
--Echo
--EKG

--Check the pt's pulse

--Carotid auscultation
--Carotid imaging
--Echo
--EKG

--Carotid imaging
--Echocardiography
--Check the pt's pulse
--Cardiac auscultation
--Echo
--EKG

Now, the workup. What tests/exam maneuvers should be performed?

For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

--Check the pt's pulse
--Carotid auscultation
--Cardiac auscultation
--Carotid imaging
--Echocardiography
--EKG
--Holter study

Embolization of retinal arterial tree

How does one go about identifying the embolic source?
In a patient with TMVL, what is the ultimate cause related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

GCA

Diffuse ocular hypoperfusion--OIS

Stenosis of the great vessels

Occlusion in the retinal venous system

Vasospasm of the CRA

In a pt with TMVL, what should push you to suspect an embolic cause?

Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

Well, they need a workup

In a nutshell, what is the goal of the workup?

To identify the embolic source

Who should do the workup?

Ideally an ER-affiliated Stroke Center; otherwise, the ER

Who should not do the workup?

You, and/or the pt’s PCP via an outpt referral. Remember, a retinal TIA is a TIA.

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Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found...

Cholesterol emboli are suggestive of... atheromatous dz of... the carotids

--Carotid auscultation
--Carotid imaging

Platelet-fibrin emboli are suggestive of... thrombotic dz of... the atria in AFib

--Check the pt’s pulse
--Cardiac auscultation
--Echo
--EKG

Calcium emboli are suggestive of... calcific dz of... the heart valves

--Cardiac auscultation
--Echo

Now, the workup. What tests/exam maneuvers should be performed?

For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

--Check the pt’s pulse
--Carotid auscultation
--Cardiac auscultation
--Carotid imaging
--Echocardiography
--EKG
--Holter study?

Embolization of retinal arterial tree

Vasospasm of the CRA

---
In a pt with TMVL, finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low, OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

Well, they need a workup. In a nutshell, what is the goal of the workup?

identify the embolic source

How does one go about identifying the embolic source? Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…

Cholesterol emboli are suggestive of… atheromatous dz of…the carotids

--Carotid auscultation
--Carotid imaging

Platelet-fibrin emboli are suggestive of… thrombotic dz of…the atria in AFib

--Check the pt's pulse
--Cardiac auscultation
--Echo
--EKG
--Holter study

Calcium emboli are suggestive of… calcific dz of…the heart valves

--Cardiac auscultation
--Echo

Now, the workup. What tests/exam maneuvers should be performed? For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

--Check the pt's pulse
--Carotid auscultation
--Cardiac auscultation
--Carotid imaging
--Echocardiography
--EKG
--Holter study

Embolization of retinal arterial tree
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

GCA

Diffuse ocular hypoperfusion

--OIS

--Stenosis of the great vessels

Occlusion in the retinal venous system

Vasospasm of the CRA

In a pt with TMVL, what should push you to suspect an embolic cause?

Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

Well, they need a workup

In a nutshell, what is the goal of the workup?

To identify the embolic source

Who should do the workup?

Ideally an ER-affiliated Stroke Center; otherwise, the ER

Who should not do the workup?

You, and/or the pt's PCP via an outpt referral. Remember, a retinal TIA is a TIA.

If a pt reported a recent two-minute spell of aphasia, would you refer them to their PCP for evaluation? (Please tell me you said no.) A retinal TIA is no different.

How does one go about identifying the embolic source?

Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found...

- Platelet-fibrin emboli are suggestive of...
  - atheromatous dz of...
- Cholesterol emboli are suggestive of...
  - atheromatous dz of...
- Calcium emboli are suggestive of...
  - calcific dz of...

--Carotid auscultation
--Carotid imaging
--Echocardiography
--EKG
--Holter study

And last but by no means least: No matter what the putative embolism type is, all pts with acute retinal ischemia need another imaging study. What is it? (This is the ‘another important study’ alluded to earlier.)

Embolization of retinal arterial tree

--Diffusion-weighted imaging (DWI)

Why this particular study?

It is especially good for revealing brain infarctions

If DWI reveals acute infarction, what should be done?

The pt should be admitted to the Stroke service
In a patient with TMVL, what is the ultimate cause related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

Diffuse ocular hypoperfusion

--OIS

--Stenosis of the great vessels

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--Carotid imaging

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Embolization of retinal arterial tree

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How does one go about identifying the embolic source?

Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…

**Cholesterol emboli** are suggestive of…

atheromatous dz of the carotids

**Platelet-fibrin emboli** are suggestive of…

thrombotic dz of the atria in AFib

**Calcium emboli** are suggestive of…

calcific dz of the heart valves

--Carotid auscultation
--Carotid imaging
--Echocardiography
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Embolization of retinal arterial tree

And last but by no means least: No matter what the putative embolism type is, all pts with acute retinal ischemia need another imaging study. What is it? (This is the 'another important study’ alluded to earlier.)

**MRI-ing everyone with retinal ischemia seems like overkill. Why bother?**

**Diffusion-weighted imaging (DWI)**

Why this particular study?

It is especially good for revealing brain infarctions

If DWI reveals acute infarction, what should be done?

The pt should be admitted to the Stroke service

MRI-ing everyone with retinal ischemia seems like overkill. Why bother?

OK, but then why not just image those who present with S/S of TIA/CVA?

Because as many as 25% of retinal ischemia pts have infarctions on DWI, and these infarctions are often 'silent” (ie, neurologically asymptomatic)
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

- Hypoperfusion of the retina and/or optic nerve
- Diffuse ocular hypoperfusion
- Stenosis of the great vessels
- Occlusion in the retinal venous system
- Vasospasm of the CRA

In a pt with TMVL, what should push you to suspect an embolic cause?

- Finding that you've ruled out ocular and orbital causes, as well as having concluded that the likelihood of GCA is low

OK, so I've concluded the acute case of TMVL sitting in front of me is likely embolic in origin. What now?

- They need a workup

In a nutshell, what is the goal of the workup?

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Who should do the workup?

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How does one go about identifying the embolic source?

- Well, recall that each embolus type is suggestive of a particular sort of pathology, which in turn is suggestive of particular locales at which the path might be found…

  - Platelet-fibrin emboli are suggestive of… thombothrombotic dz of…
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Now, the workup. What tests/exam maneuvers should be performed?

- For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:
  - Check the pt's pulse
  - Carotid auscultation
  - Carotid imaging
  - Echocardiography
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- Calcium emboli are suggestive of calcific dz of the heart valves.

- Carotid auscultation
- Carotid imaging
- Check the pt's pulse
- Cardiac auscultation
- Echo
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Diffuse ocular hypoperfusion

--OIS

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thrombotic dz of…

the atria in AFib

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MRI

There's a particular MRI sequence the Neuro book stresses must be obtained—what is it?

Diffusion-weighted imaging (DWI)

Why this particular study?

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If DWI reveals acute infarction, what should be done?

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  - Carotid imaging
  - Echocardiography
  - EKG
  - Holter study

- **Cholesterol emboli** are suggestive of atheromatous dz of:
  - Carotid auscultation
  - Carotid imaging

- **Calcium emboli** are suggestive of calcific dz of:
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Embolization of retinal arterial tree

- Vasospasm of the CRA

If DWI reveals acute infarction, what should be done? The pt should be admitted to the Stroke service.
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

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GCA

Diffuse ocular hypoperfusion -- OIS -- Stenosis of the great vessels

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- Carotid auscultation

- Carotid imaging

- Check the pt's pulse

- Cardiac auscultation

- Echocardiography

- EKG

- Holter study

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**Calcium emboli** are suggestive of... calcific dz of the heart valves.

- Carotid auscultation
- Carotid imaging
- Echocardiography
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- Holter study

And last but by no means least: No matter what the putative embolism type is, **all pts with acute retinal ischemia need another imaging study.** What is it? (This is the ‘another important study’ alluded to earlier.)

**MRI**

**There's a particular MRI sequence the Neuro book stresses must be obtained**—what is it?

Diffusion-weighted imaging (DWI)

**Why this particular study?**

It is especially good for revealing brain infarctions.

**If DWI reveals acute infarction, what should be done?**

- Carotid imaging
- Echocardiography
- EKG
- Holter study

**Embolization of retinal arterial tree**

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**Diffuse ocular hypoperfusion--OIS**

**Stenosis of the great vessels**

**Occlusion in the retinal venous system**

**Hypoperfusion of the retina and/or optic nerve**

**GCA**
And last but by no means least: No matter what the putative embolism type is, all pts with acute retinal ischemia need another imaging study. What is it? (This is the ‘another important study’ alluded to earlier.) MRI

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It is especially good for revealing brain infarctions

If DWI reveals acute infarction, what should be done?
The pt should be admitted to the Stroke service

--Carotid imaging
--Echocardiography
--EKG
--Holter study

Embolization of retinal arterial tree
What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

Hypoperfusion of the retina and/or optic nerve

GCA

Diffuse ocular hypoperfusion — OIS

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In a nutshell, what is the goal of the workup?

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— carotids

Calcium emboli are suggestive of calcific dz of

— heart valves

— Carotid auscultation

— Carotid imaging

— Check the pt’s pulse

— Cardiac auscultation

— Echo

— EKG

— Holter study

Now, the workup. What tests/exam maneuvers should be performed?

For each, indicate the type(s) of emboli for which the test has direct bearing vis a vis making a diagnosis:

— Check the pt’s pulse

— Carotid auscultation

— Cardiac auscultation

— Carotid imaging

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And last but by no means least: No matter what the putative embolism type is, all pts with acute retinal ischemia need another imaging study. What is it? (This is the ‘another important study’ alluded to earlier.)

MRI

There’s a particular MRI sequence the Neuro book stresses must be obtained—what is it?

Diffusion-weighted imaging (DWI)

Why this particular study?

It is especially good for revealing brain infarctions

But silent

If DWI reveals acute infarction, what should be done?

The pt should be admitted to the Stroke service

But silent

What should be done if the DWI-revealed acute infarct is silent?

Makes no difference—the pt should still be admitted

— Holter study

— Carotid auscultation

— Carotid imaging

— Cardiac auscultation

— Echocardiography

— EKG

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What is the ultimate cause of TMVL related to the vasculature? (Looking for a single word here.)

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In a pt with TMVL, what should push you to suspect an embolic cause?
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In a nutshell, what is the goal of the workup?
To identify the embolic source

Who should do the workup?
Ideally an ER-affiliated Stroke Center; otherwise, the ER

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MRI

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Diffusion-weighted imaging (DWI)

Why this particular study?
It is especially good for revealing brain infarctions but silent

If DWI reveals acute, infarction, what should be done?
The pt should be admitted to the Stroke service

What should be done if the DWI-revealed acute infarct is silent?
Makes no difference—the pt should still be admitted
(This is a good point in the set to take a break)
The Neuro book discusses three causes of binocular TVL. What are they?
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Actually, the book does touch upon a fourth cause—what is it?
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Occipital seizures
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Are occipital seizures a common cause of binocular TVL?
The Neuro book discusses three causes of binocular TVL. What are they?

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No—they are “very uncommon” to quote the Neuro book
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How long does seizure-induced TVL last? Seconds, usually (an important diagnostic consideration)
Of these, which is most common?
Of these, which is most common?
What buzzword is the general term for vision changes associated with migraine?

Migraine

- Posterior circulation abnormality
- Occipital structural abnormality

What is the classic presentation of bilateral vision loss secondary to migraine?

Homonymous hemianopia

How does it present?

It starts as a small scotoma surrounded by scintillations. Over a period of a few minutes, it enlarges, and then begins to slowly fade. Vision returns to normal in no more than about 60 minutes.
What buzzword is the general term for vision changes associated with migraine? ‘Aura’
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Does migraine aura precede, or follow the migraine HA itself?
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Precede
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Is aura always followed by a HA?
**Q/A**

**TVL**

**TMVL**

**Binocular**

**Migraine**

*What buzzword is the general term for vision changes associated with migraine?*

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*Does migraine aura precede, or follow the migraine HA itself?*

Precede

*Is aura always followed by a HA?*

No, some pts have what are called *acephalgic migraines* in which they get an aura but no subsequent HA.

*Posterior circulation abnormality*  

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Migraine is a recurrent condition. Is the same hemifield always involved?

Migraine
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Migraine is a recurrent condition. Is the same hemifield always involved?
No—in fact, if only one hemifield is involved over and over again, the migraine dx should be questioned

Migraine

Posterior circulation abnormality
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In such cases, what sort of condition rises to the top of the DDx?
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How do migraine-related vision changes present and proceed?

It starts as a small scotoma surrounded by scintillations. Over a period of a few minutes it enlarges, and then begins to slowly fade. Vision returns to normal in no more than about 60 minutes.
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What term is often used to describe the geometric quality of these scintillations?
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‘Fortification spectrum’
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How do migraine-related vision changes present and proceed? It starts as a small scotoma surrounded by scintillations.

What term is often used to describe the geometric quality of these scintillations? ‘Fortification spectrum’

Note: Fortification spectra are closely associated with migraines (as they should be). However, any occipital-based cause of TVL can present with fortification spectra.
**What buzzword is the general term for vision changes associated with migraine?**

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**What term is often used to describe the geometric quality of these scintillations?**

‘Fortification spectrum’

Note: Fortification spectra are closely associated with migraines (as they should be). However, any occipital-based cause of TVL can present with fortification spectra. So when you hear *fortification spectra*, you mos def should think migraine first—but keep occipital circulatory and structural issues in the back of your mind.
What buzzword is the general term for vision changes associated with migraine? ‘Aura’

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How do migraine-related vision changes present and proceed? It starts as a small scotoma surrounded by scintillations. Over a period of a few unit of time, it enlarges v shrinks, and then begins to slowly fade.
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It starts as a small scotoma surrounded by scintillations. **Over a period of a few minutes it enlarges, and then begins to slowly fade.** Vision returns to normal in no more than about [amount of time].
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Visual aura of migraine. A, The aura commonly begins with a small scotoma near fixation that gradually expands into the peripheral vision (B–C) and then breaks up (D). The times shown represent minutes from the onset of the visual aura.
What buzzword is the general term for vision changes associated with migraine?

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While we’re on this subject: What is a retinal migraine?

It is the same as a garden-variety migraine-with-aura, with one huge difference—the aura is confined to one eye as opposed to one hemifield.

Seems like we see retinal migraine pts a lot in clinic. Why is it so common?

Here’s the thing—it’s not common; in fact, it is considered to be very rare. Despite this, in clinical practice retinal migraine is often used as a wastebasket diagnosis for unexplained TMVL. Don’t do this!

OK, if it’s not a retinal migraine, what is it?

Most likely, it’s migraine-with-aura for which the pt has (mis)identified their bilateral hemifield loss as monocular (as we mentioned they are wont to do).
**Q/A**

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What sorts of 'circulatory abnormalities' are we talking about here?
--?
--?
--?

Three mentioned in the Neuro book

Migraine

Posterior circulation abnormality

Occipital structural abnormality
What sorts of ‘circulatory abnormalities’ are we talking about here?

- Embolism
- Vasculitis
- Atherosclerosis

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What sorts of ‘circulatory abnormalities’ are we talking about here?
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What is the final common pathway by which such abnormalities lead to TVL?
What sorts of ‘circulatory abnormalities’ are we talking about here?
--Embolism
--Vasculitis
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What is the final common pathway by which such abnormalities lead to TVL?
They lead to occipital ischemia

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HA 2ndry to these ischemic events…Where does it tend to localize?
What sorts of 'circulatory abnormalities' are we talking about here? 
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- Vasculitis 
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Yes

HA 2ndry to these ischemic events...Where does it tend to localize? 
In the region
What sorts of ‘circulatory abnormalities’ are we talking about here?
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The HA of these events differ in an important respect from that of migraine-with-aura—what is it?
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The HA of these events differ in an important respect from that of migraine-with-aura—what is it?
Whereas a migraine HA follows the vision loss, in occipital ischemic events the HA occurs simultaneous with vs long before the vision loss
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Speaking of important differences between the presentations of migraine-with-aura vs occipital ischemia: In what two ways does vision loss differ between them?

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Are these associated with HA?
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Speaking of important differences between the presentations of migraine-with-aura vs occipital ischemia: In what two ways does vision loss differ between them?
--The onset of vision loss in occipital ischemia is sudden (as opposed to the gradual onset characteristic of migraine-with-aura)
--The duration of vision loss in occipital ischemia is brief (just a few minutes, as opposed to the 30-60 minutes typical of migraine-with-aura)

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