

Uveitis: *OHS*

Histo: Basics

What is the causative organism in ocular histoplasmosis syndrome (OHS)?

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- 2) The profiled case is meshed
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*(Why the *Path* book doesn't, I have no idea.) Both refer to it as is done here, ie, as 'ocular histoplasmosis syndrome,' without the word *presumed* leading the way.*

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tl;dr As of this writing, the *BCSC* considers causality **likely**, but not **proven**.

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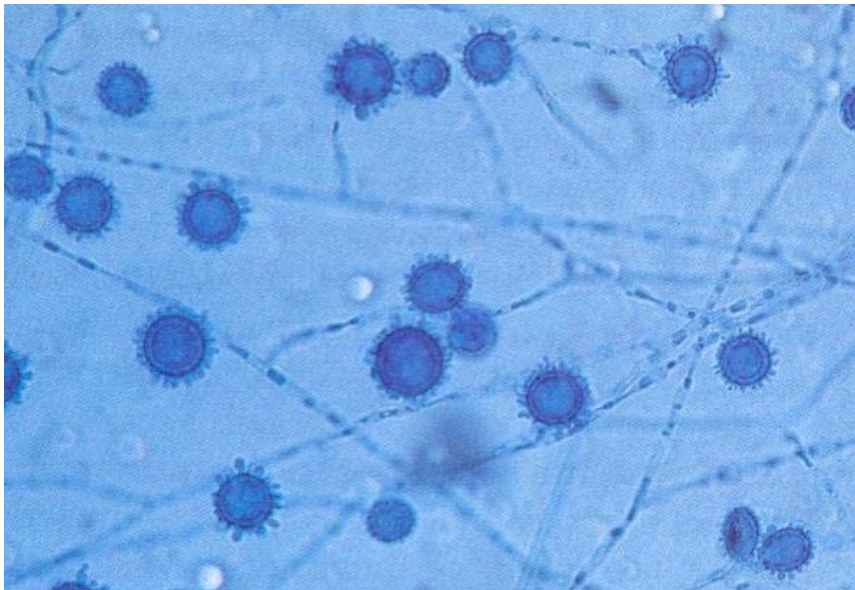
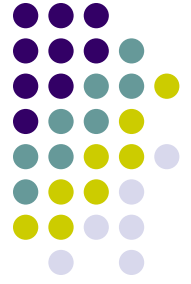
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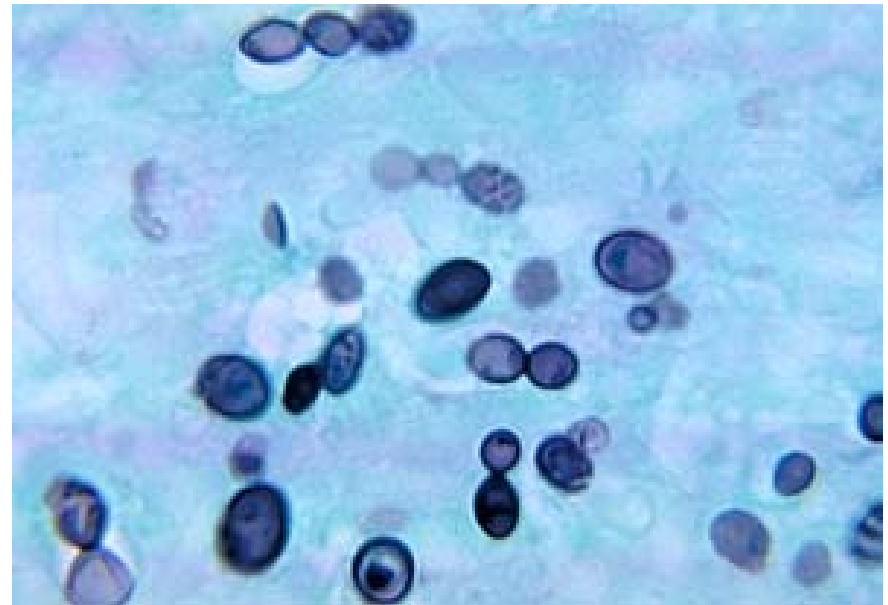
What does it mean to say a fungus is 'dimorphic'?

It means the organism has both a yeast form and a filamentous (mold) form

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H capsulatum: Mold (filamentous) form



H capsulatum: Yeast form

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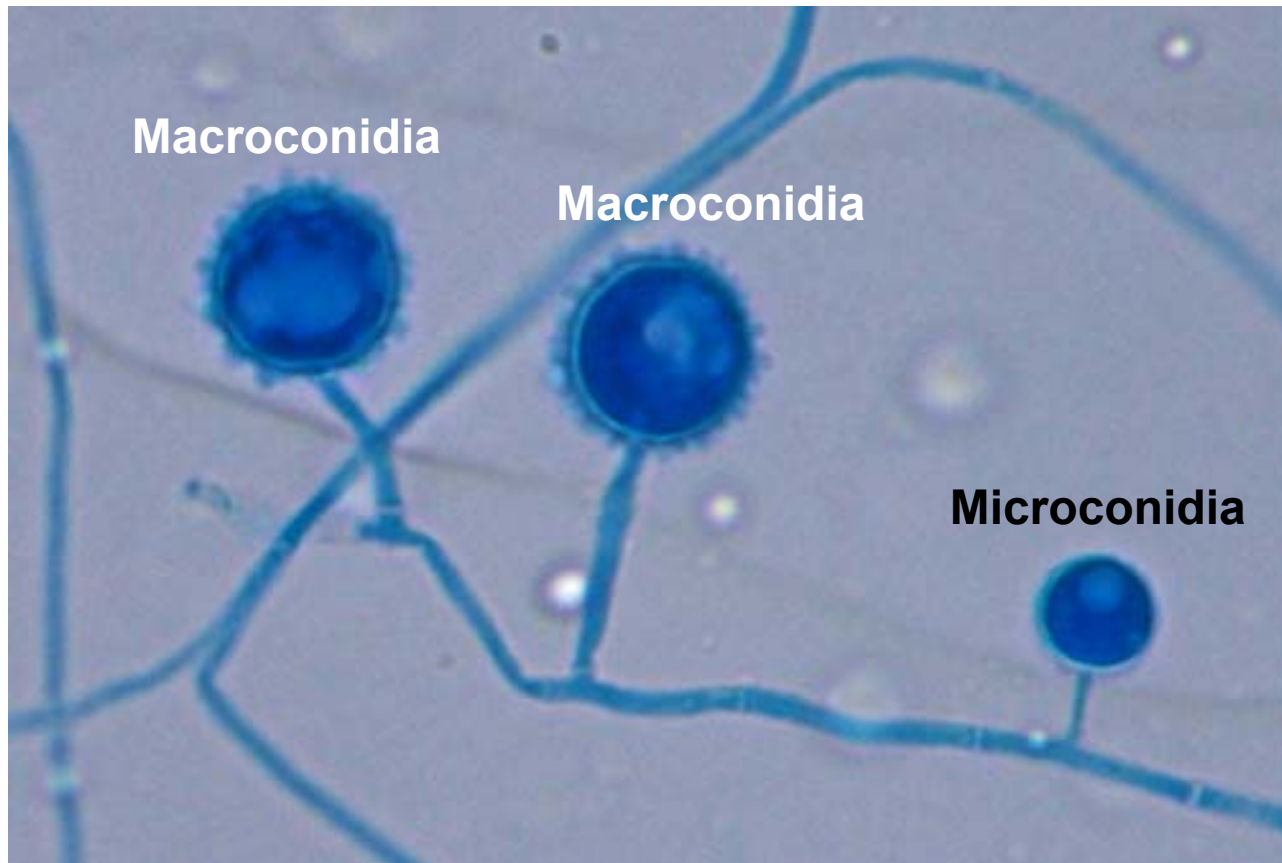
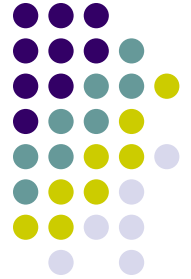
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Microconidia are the infectious particles of the mold, while the ***macroconidia*** are characteristic of the organism and provide a clue to its identification!

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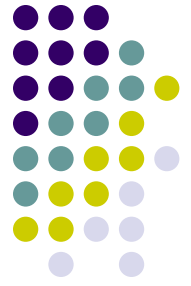
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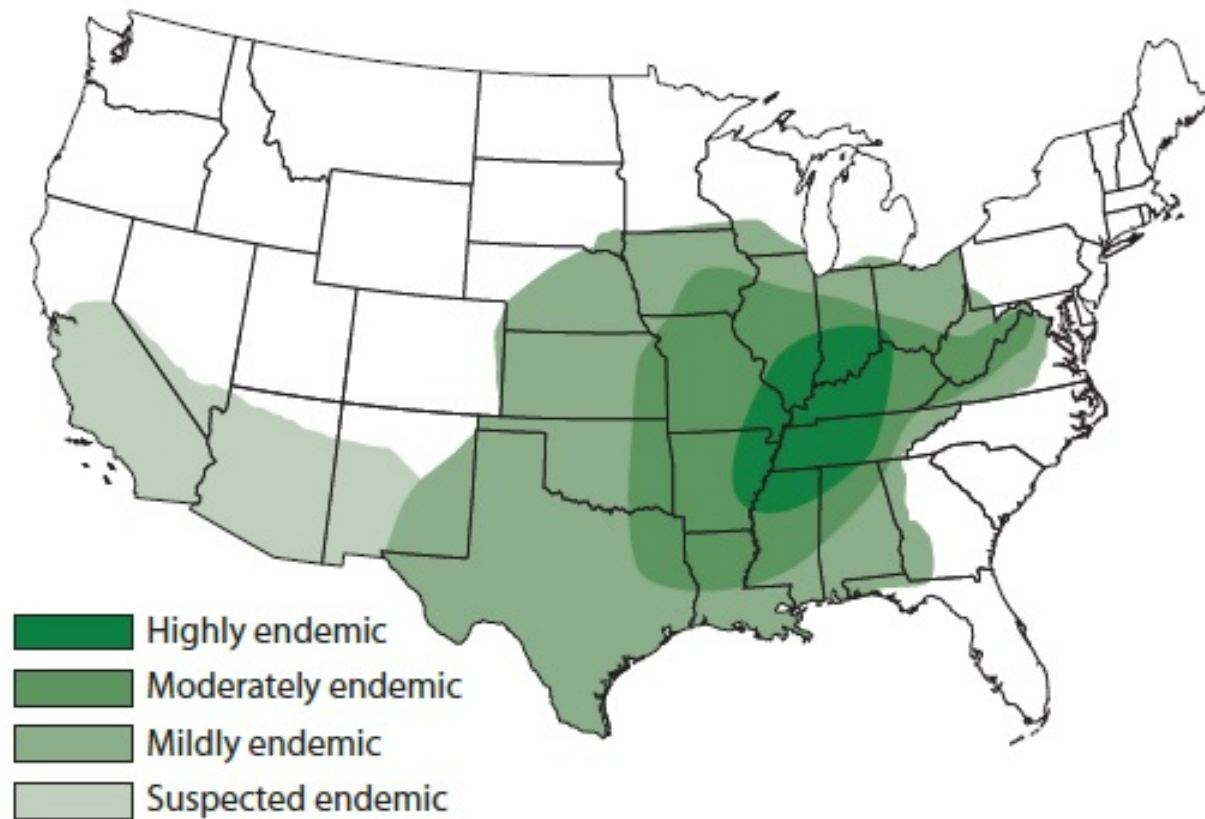
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Areas Endemic for Histoplasmosis



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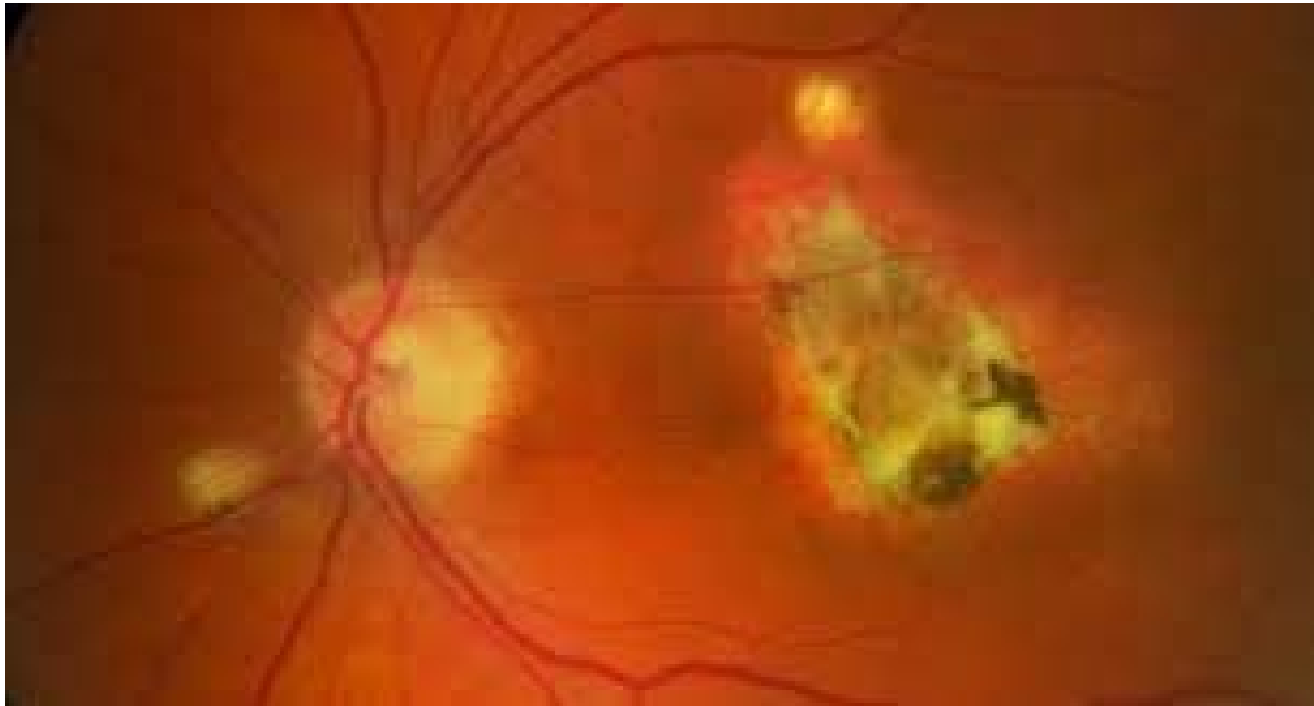
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Bilaterally (although it can be asymmetric)

Is OHS associated with vitritis?

Never. If vitritis is present, it's not OHS.

What about AC cell?

Never. If AC cell is present, it's not OHS.

If OHS is associated with vision loss, what is the culprit?

A subfoveal choroidal neovascular membrane (CNVM)

Is there an HLA association?

It seems so. Pts positive for HLA-DRw2 or HLA-B7 are much more likely to manifest the condition.

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Uveitis

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Panuveitis

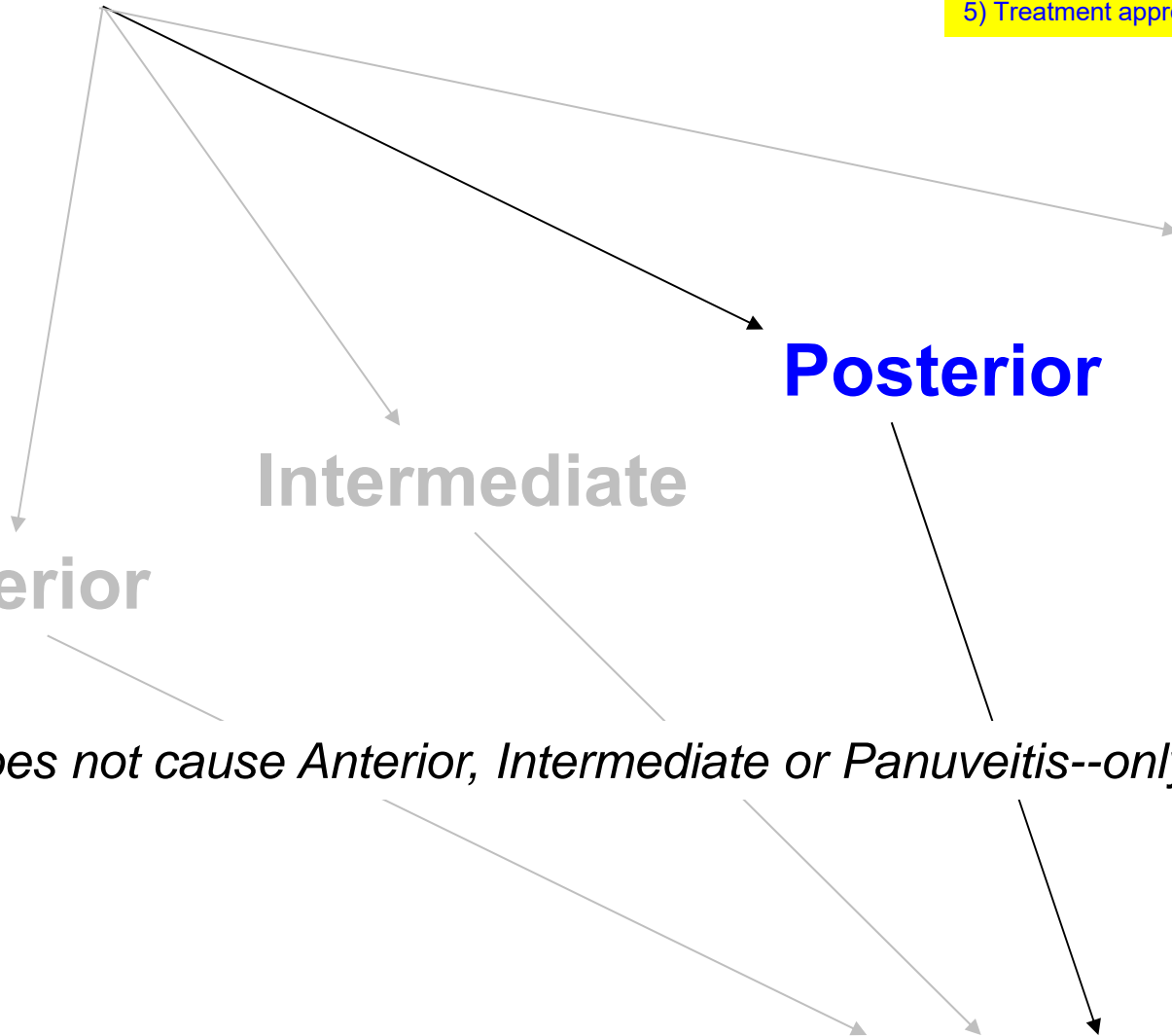
Posterior

Intermediate

Anterior

OHS does not cause Anterior, Intermediate or Panuveitis--only Posterior uveitis

OHS



Uveitis: *Posterior*

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**Chorioretinitis or
Retinochoroiditis**

Retinitis

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What is the classic posterior manifestation of OHS?
A multifocal chorioretinitis

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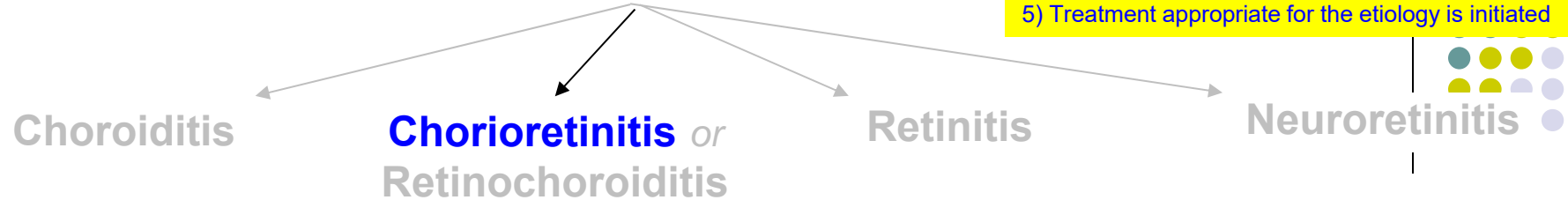
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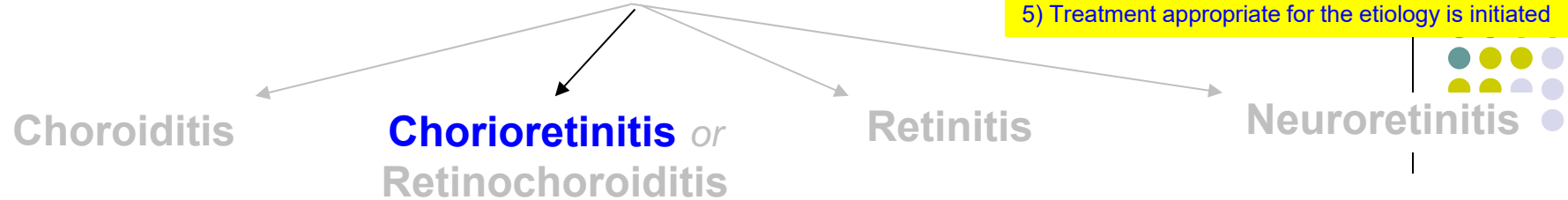
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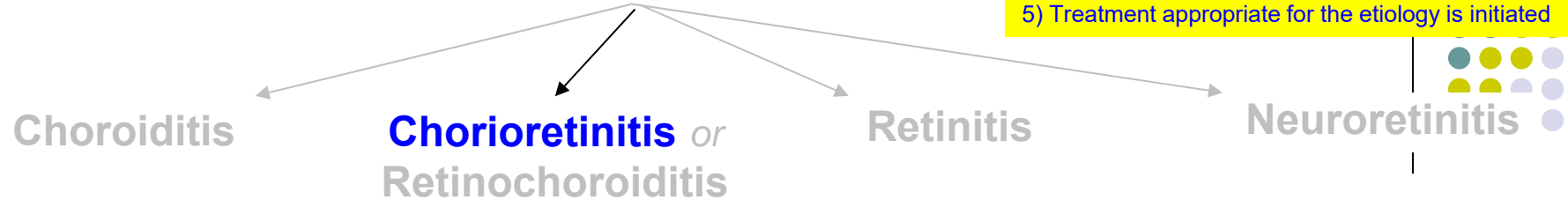
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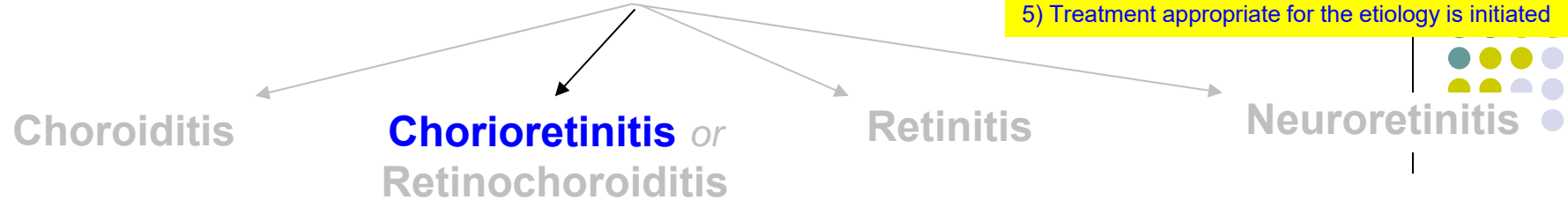
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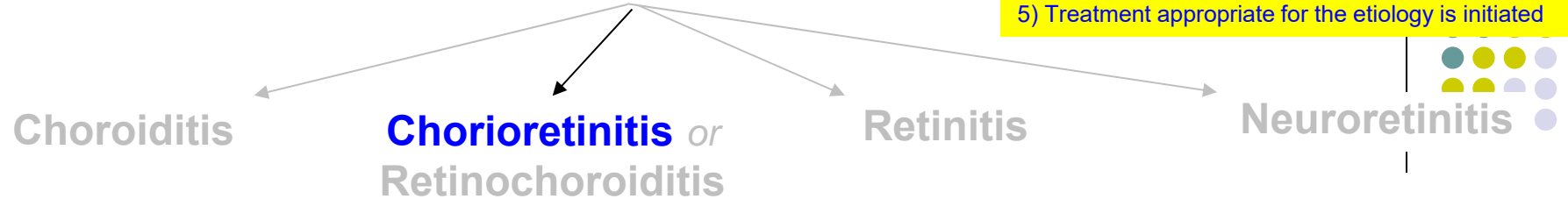
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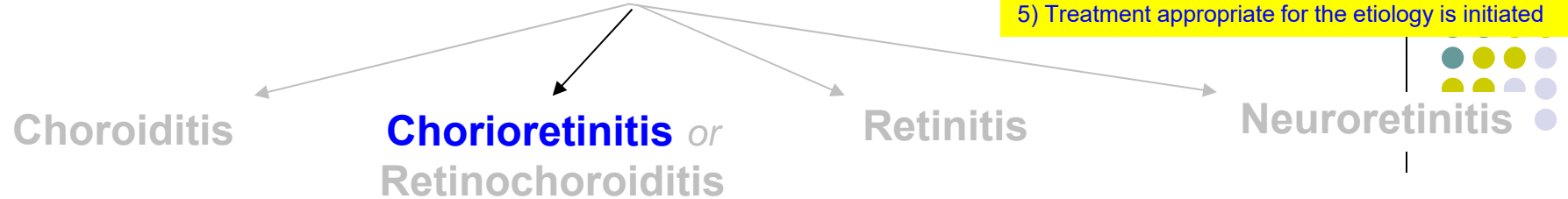
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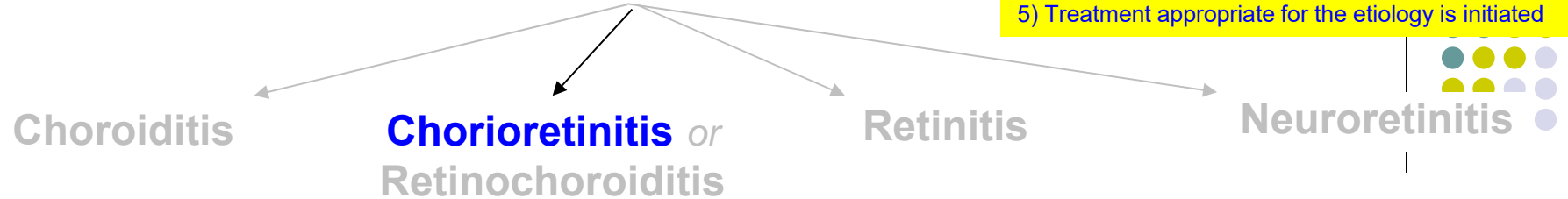
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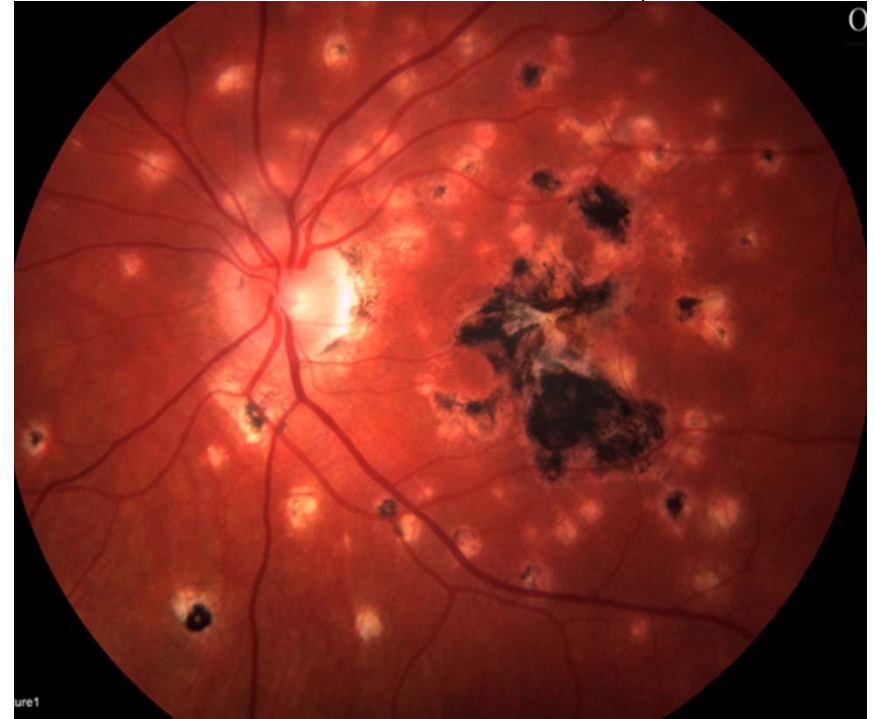
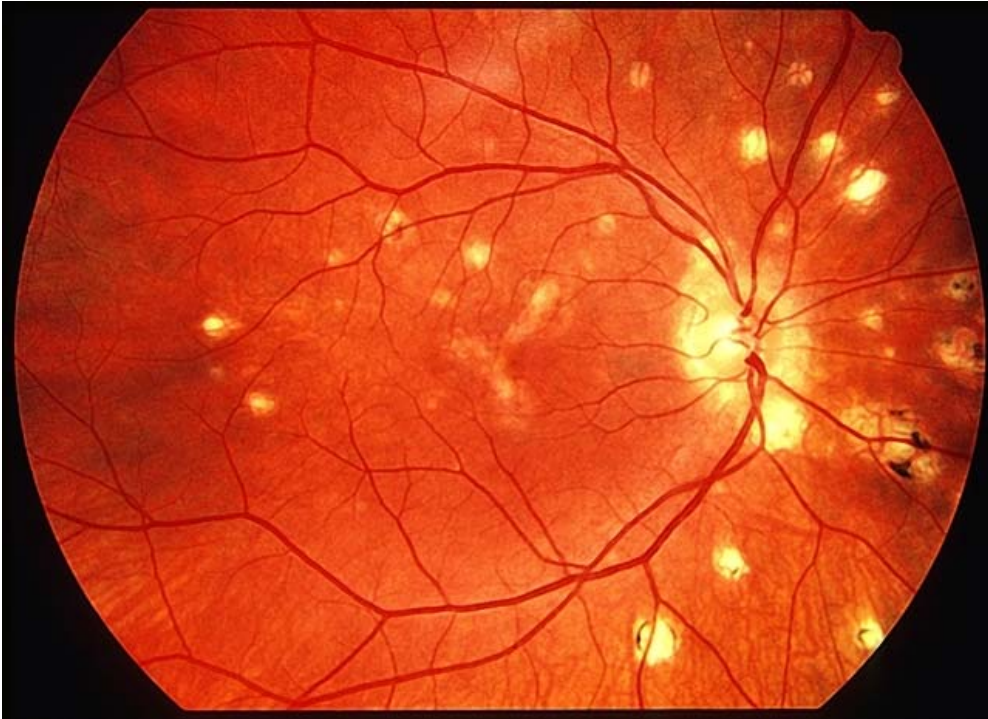
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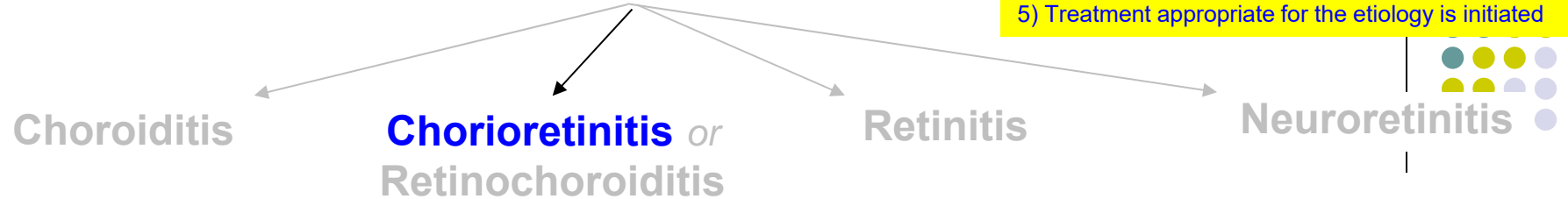
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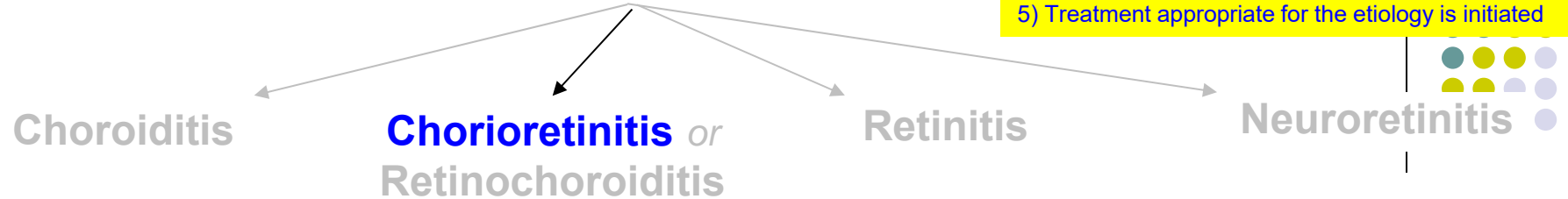
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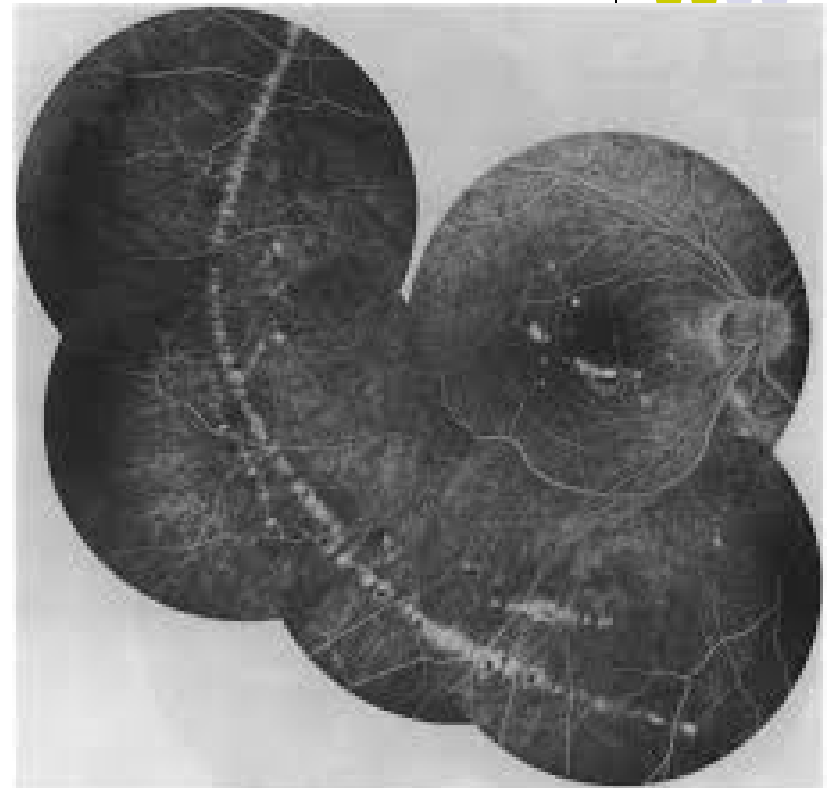
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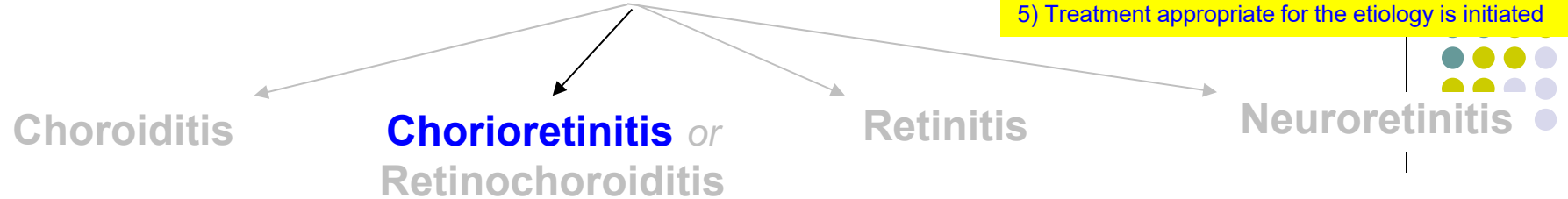
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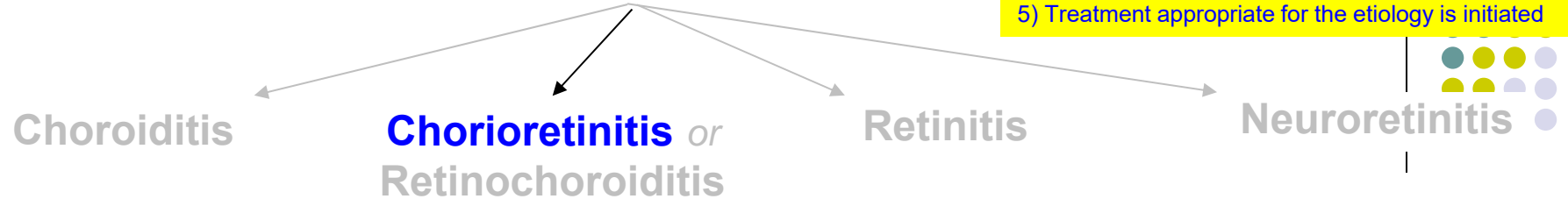
Do they evolve over time? Generally no

Re the three lesions of OHS: **Peripapillary atrophy**

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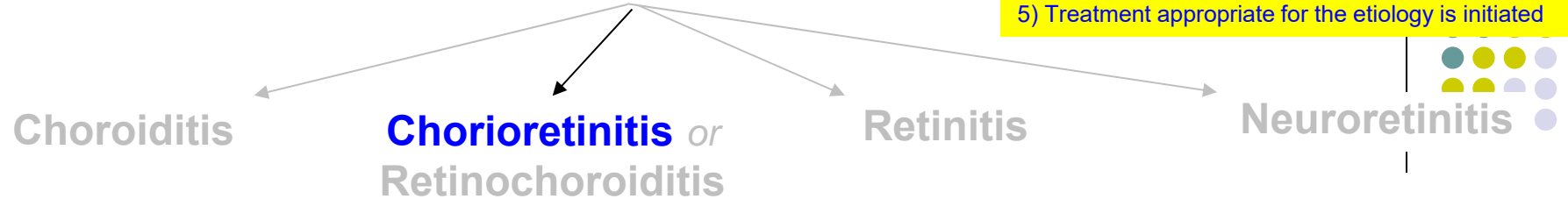
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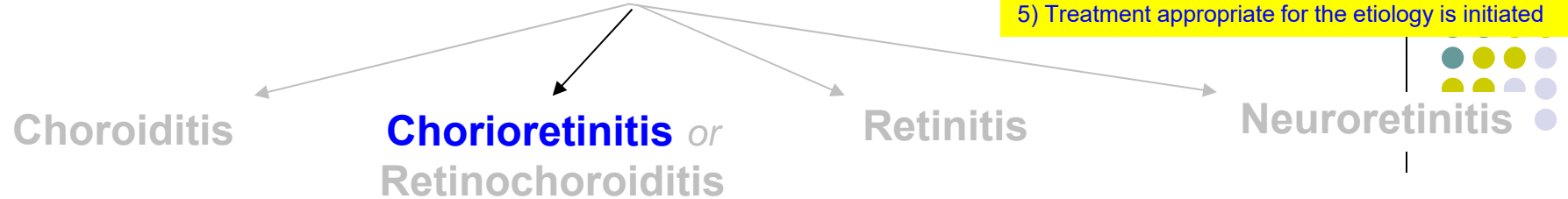
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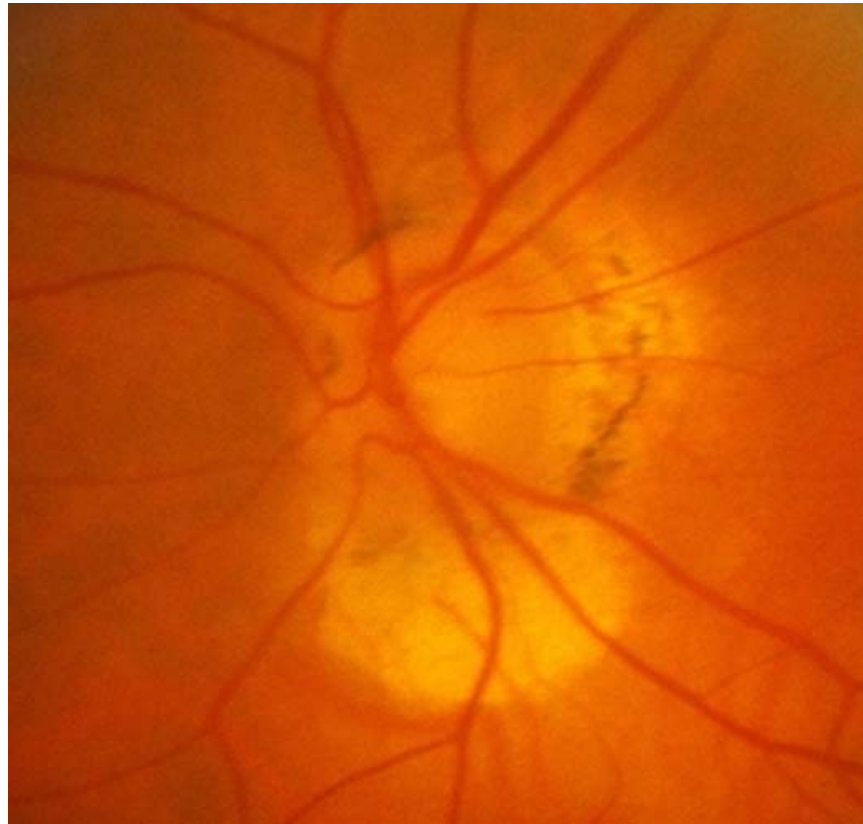
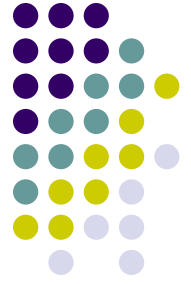
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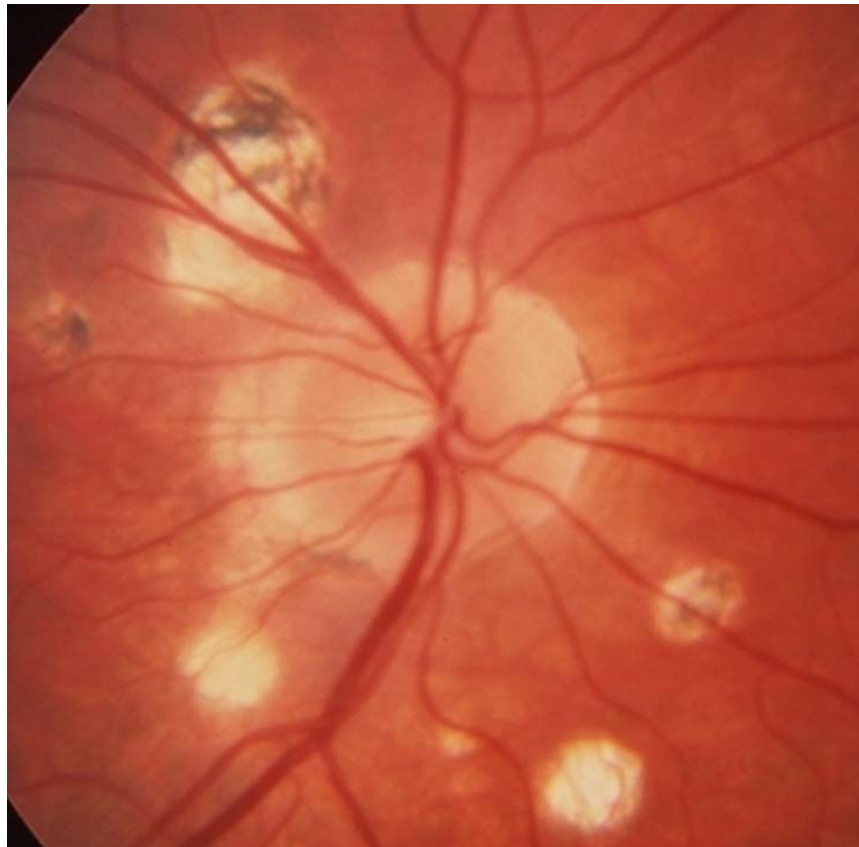
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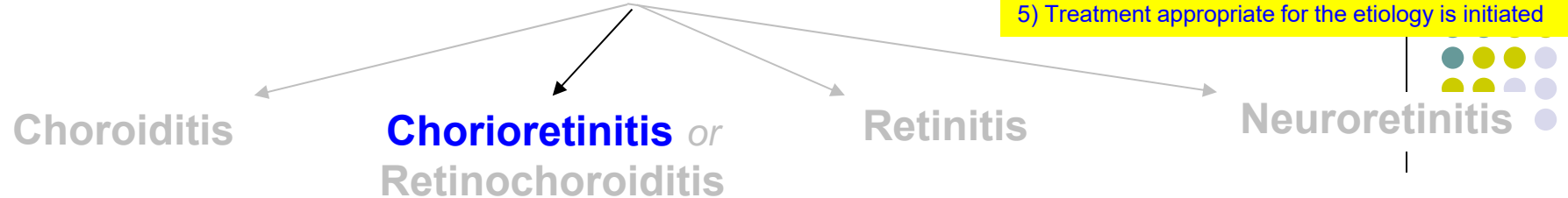
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Re the three lesions of OHS: **Macular disciform lesions**

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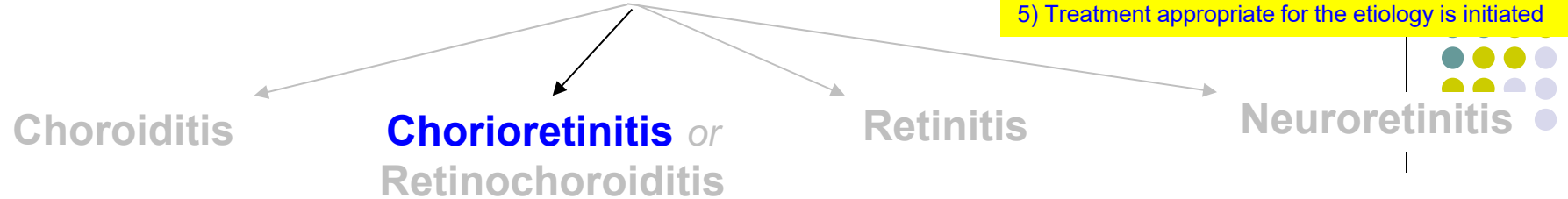
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What specific structure(s) is/are involved? Everything, including a defect in Bruch's membrane

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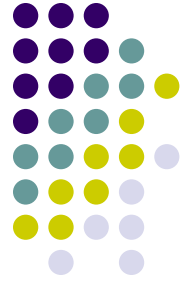
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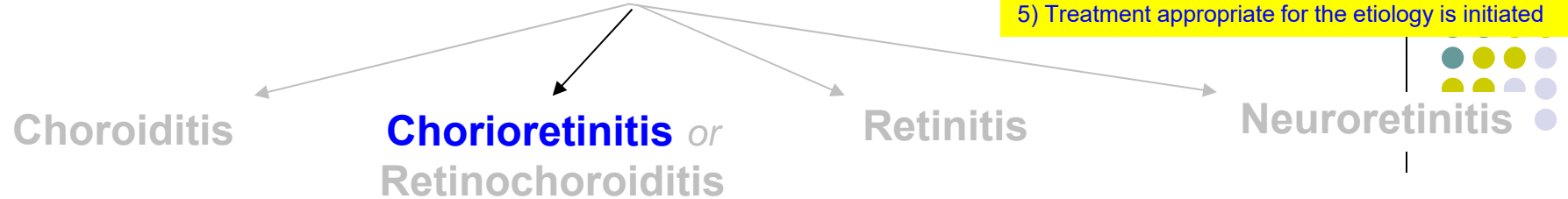
In a nutshell, what are they? **Active** lesions represent either the presence of CNVM under the retina, or a hemorrhagic retinal detachment



Histo: Macular disciform lesion

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Do they evolve over time? Generally no

Re the three lesions of OHS: **Peripapillary atrophy**

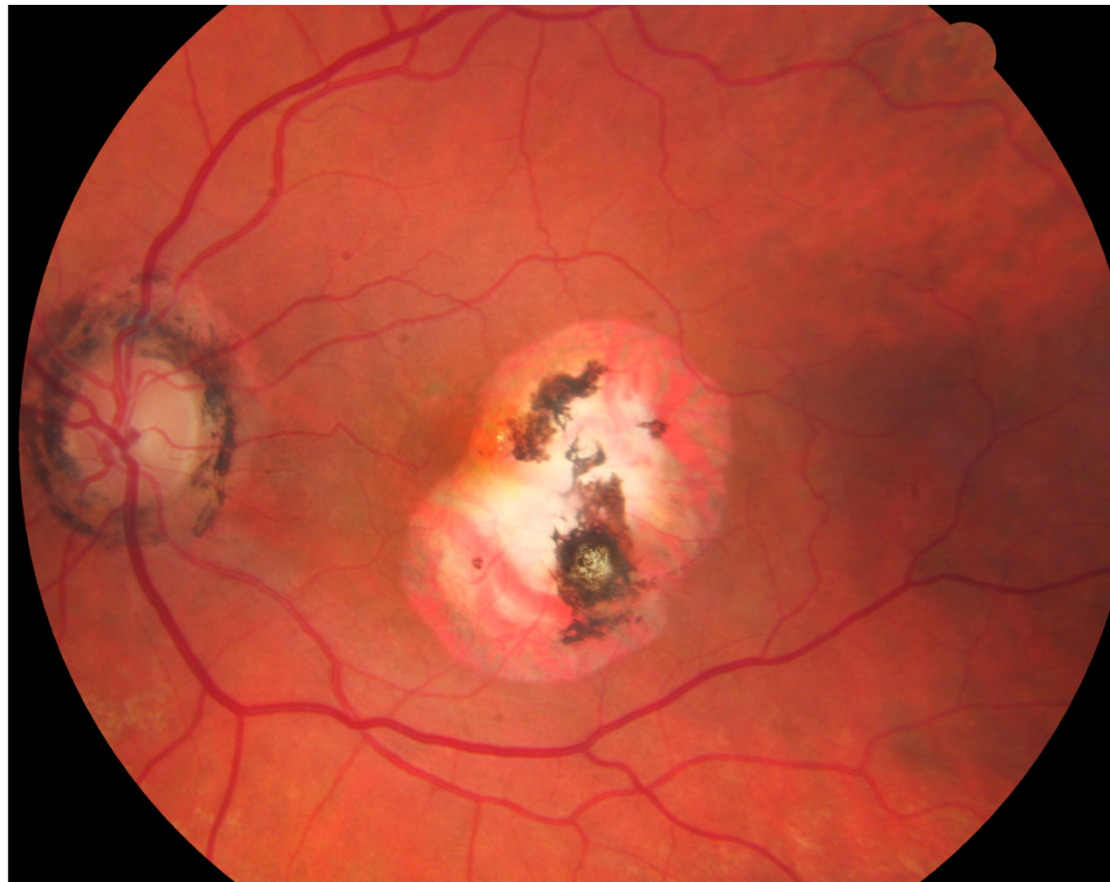
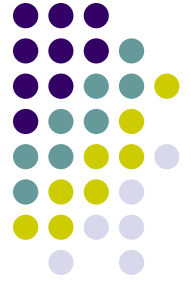
What specific structure(s) is/are involved? The inner choroid, RPE and retina

In a nutshell, what are they? Discrete, focal, atrophic scars

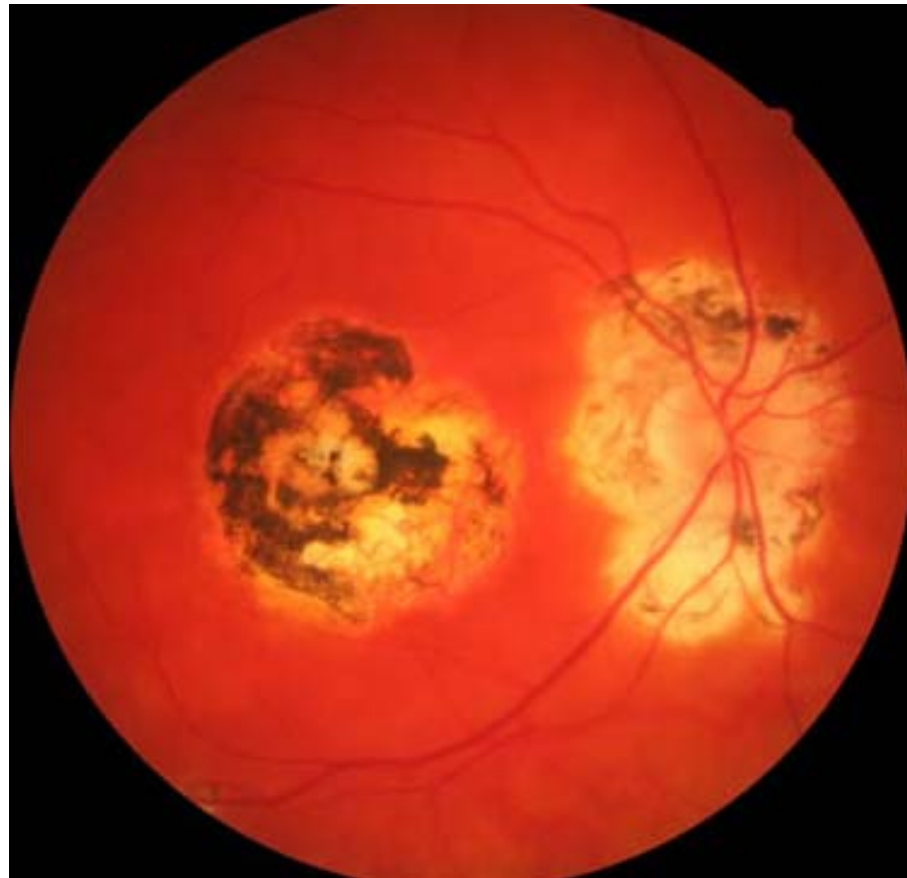
Re the three lesions of OHS: **Macular disciform lesions**

What specific structure(s) is/are involved? Everything, including a defect in Bruch's membrane

In a nutshell, what are they? **Active** lesions represent either the presence of CNVM under the retina, or a hemorrhagic retinal detachment. **Inactive** lesions (aka *disciform scars*) are fibrovascular remnants of previous CNVM and/or subretinal hemorrhage.



Histo: Macular disciform lesion



Histo: Macular disciform lesion

Uveitis: **OHS**

Diagnosis

How is the diagnosis of OHS made?

- 1) The uveitis is profiled
- 2) The profiled case is meshed
- 3) A differential diagnosis list is generated
- 4) Studies are obtained to identify the etiology
- 5) Treatment appropriate for the etiology is initiated



Uveitis: *OHS*

Diagnosis

How is the diagnosis of OHS made?

It is a clinical diagnosis based on DFE findings

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Do antifungals play a role in the treatment?

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Why no role for antifungals?

Because there's no evidence to indicate live organisms are present
(much less actively contributing to the CNVM process)

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- Photodynamic therapy (PDT)
- Anti-VEGF therapy
- Submacular surgery
- Intravitreal corticosteroids
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As a loss of 6 or more lines from initial presentation

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- The specific underlying condition responsible for the CNVM occurrence
- Whether the lesion was new, or recurrent

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With respect to distance from the foveal center

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There were a number of factors that influenced the outcome:

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--Whether the lesion

Four locations were used. What were they?

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--Extrafoveal

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--Subfoveal: Some portion of the CNVM was directly below the foveal center

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--Whether the lesion was *subfoveal*

Was OHS one of the CNVM-causing conditions included in the MPS?

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How is the diagnosis of OHS made?

What landmark clinical study evaluated the use of thermal laser for the treatment of CNVM?

The Macular Photocoagulation Study (MPS)

What two laser modalities were evaluated?

Argon (**blue-green**) and krypton (**red**)

What was the primary endpoint/outcome variable?

Percent of eyes experiencing severe vision loss (SVL) from baseline

There were a number of subgroup analyses in the MPS. Important subgroup analyses were based on:

--Lesion location

--**The specific underlying condition responsible for the CNVM occurrence**

--Whether the lesion was **new or recurrent**

Was OHS one of the CNVM-causing conditions included in the MPS?

Yes

What treatment modalities

--**Thermal laser**

--Photodynamic therapy (PDT)

--Anti-VEGF therapy

--Submacular surgery

--Intravitreal corticosteroids

--Combination therapy (of some of the above modalities)

Uveitis: *OHS*

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It found that thermal laser significantly reduced the risk of SVL in extrafoveal and juxtafoveal lesions. Treatment of subfoveal lesions provided a small long-term benefit, but was associated with an immediate, dramatic decrease in acuity. (Newsflash: If you laser the fovea, vision suffers.) Further, thermal laser treatment was associated with a high CNVM recurrence rate.

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Is thermal laser still an acceptable treatment for CNVM associated with OHS?

In select pts, yes. If a pt has extrafoveal (or even juxtafoveal) disease, thermal laser is a reasonable option. This is especially the case if the pt is not a good candidate for other treatment modalities.

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--Infusion-related two words pain

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A molecule is introduced into the eye that binds VEGF, thereby preventing it from binding its receptors in the budding CNVM complex

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- 3) A differential diagnosis list is generated
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What does VEGF stand for?

Vascular endothelial growth factor

Broadly, what is it?

A signaling molecule that promotes angiogenesis

What does VEGF have to do with CNVM formation?

It appears to play a vital role in initiation of the CNVM process, and thus provides a target for clinical intervention to interrupt the development of CNVM

What is the basic premise underlying anti-VEGF therapy?

A molecule is introduced into the eye that binds VEGF, thereby preventing it from binding its receptors in the budding CNVM complex

The currently-employed anti-VEGF meds work by one of two techniques for binding it--what are they?

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Photodynamic therapy (PDT)

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--Submacular surgery

--Intravitreal corticosteroids

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What two VEGF-Ab drugs currently dominate the market?

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'VEGF Trap'

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Yes--in fact, most clinicians probably consider it their first-line treatment

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At one time, submacular surgery was the only alternative to thermal laser, and thus a more compelling case could be made for its risk/benefit profile (especially with regard to subfoveal lesions). However, the advent of PDT and the development of anti-VEGF therapies have rendered submacular surgery considerably less popular.

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That said, in certain very select clinical situations (eg, a large peripapillary CNVM), and if other treatment modalities have proven ineffective, submacular surgery would be a reasonable option to consider.

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Corticosteroids are anti-inflammatory meds. What role does inflammation play in CNVM?

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The same drawbacks that apply to using them to treat anything--cataract formation, and ocular hypertension

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Is corticosteroid therapy an acceptable treatment for CNVM associated with OHS?

Absent highly extenuating circumstances, few if any clinicians would advocate for using steroids as a first-line treatment. However, they do have a role as an adjunctive therapy in combination with other treatments.

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--Combination therapy (of some of the above modalities)

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Treatment

Which lesion(s) require treatment?

Active disciform lesions

Do antifungals play a role in the treatment?

No

Is there any treatment known to reduce the risk of developing disciform lesions?

No

What treatment modalities are used to treat active disciform lesions?

-- *Which combinations seem to show particular promise?*

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-- Anti-VEGF + PDT

-- Anti-VEGF + thermal laser

-- PDT + corticosteroids

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