

# Uveitis: *OHS*

## *Histo: Basics*

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

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Fair—that was probably presumptuous on my part. Two volumes address OHS: *Uveitis*, and *Retina*. (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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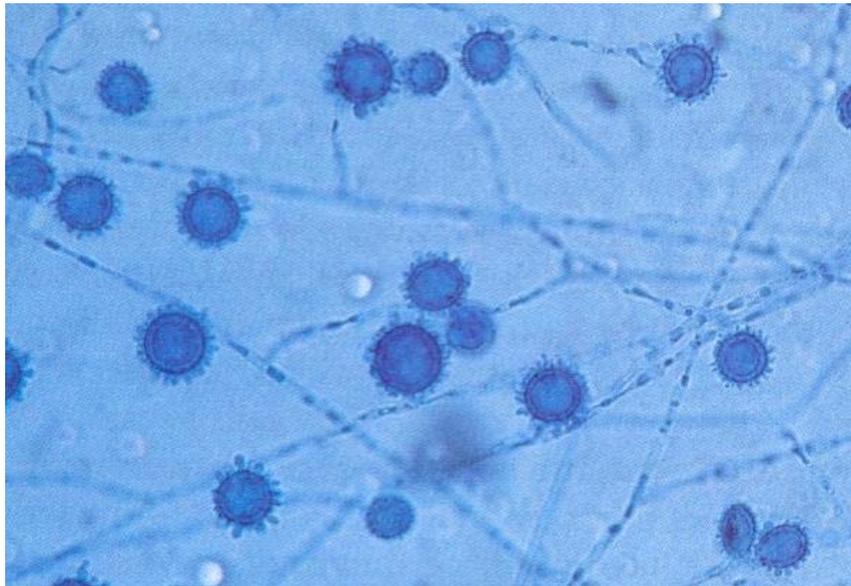
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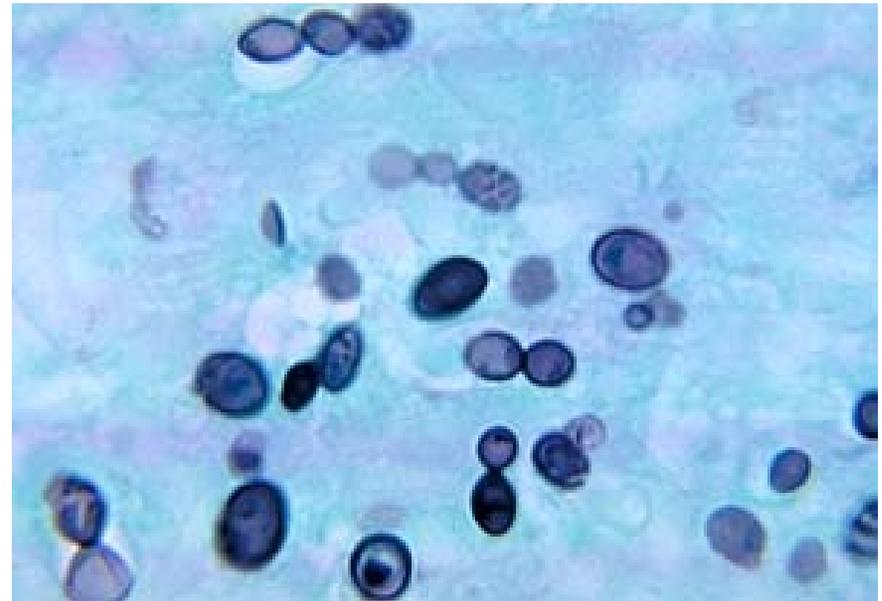
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*H capsulatum*: Mold (filamentous) form



*H capsulatum*: Yeast form

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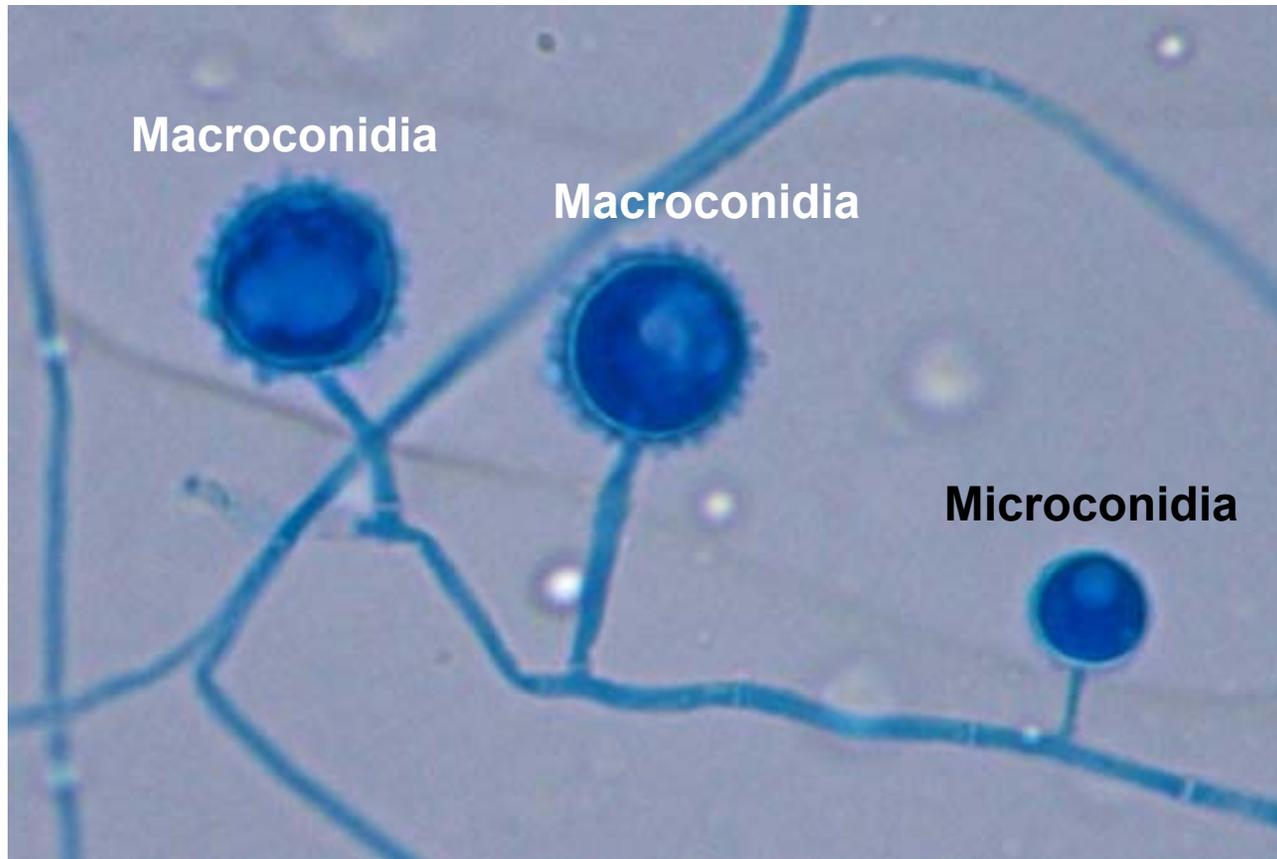
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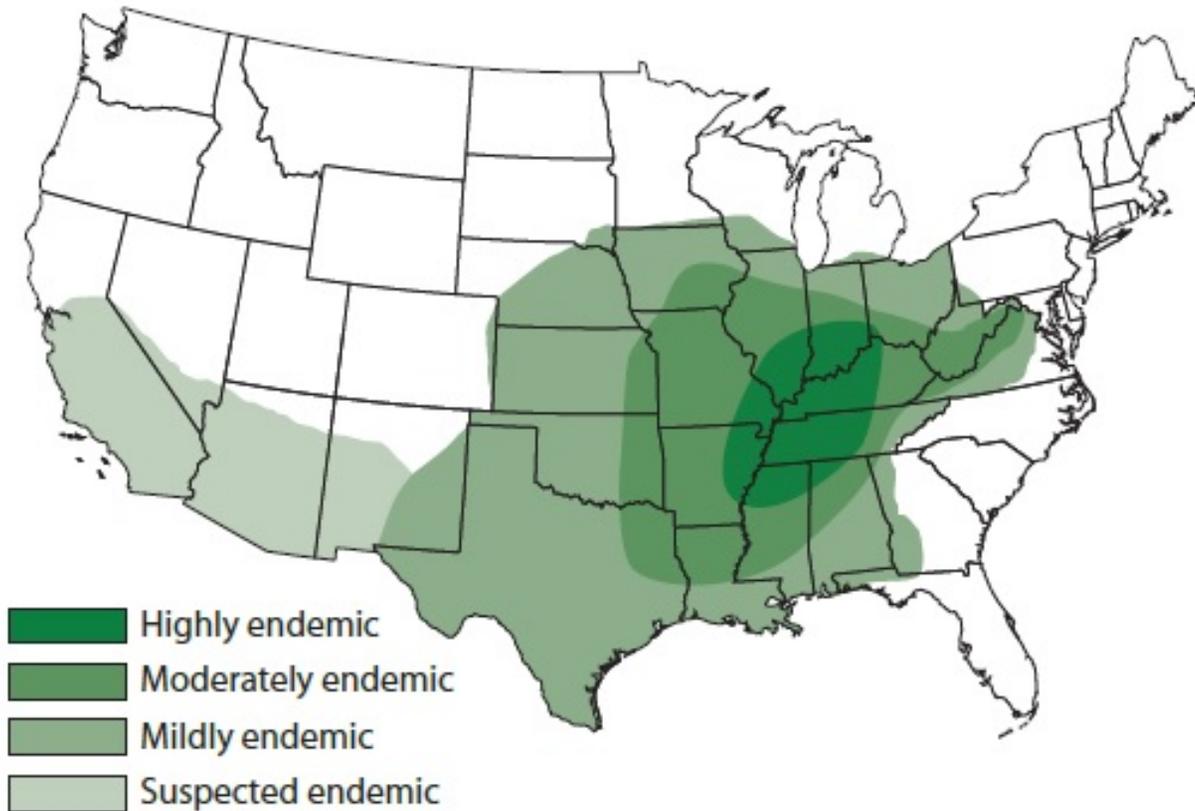
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Areas Endemic for Histoplasmosis



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Yes, OHS occurs almost exclusively among **ethnicity** of **geographic area** heritage

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--Disciform macular lesion(s)

*Does OHS manifest unilaterally, or bilaterally?*

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?*

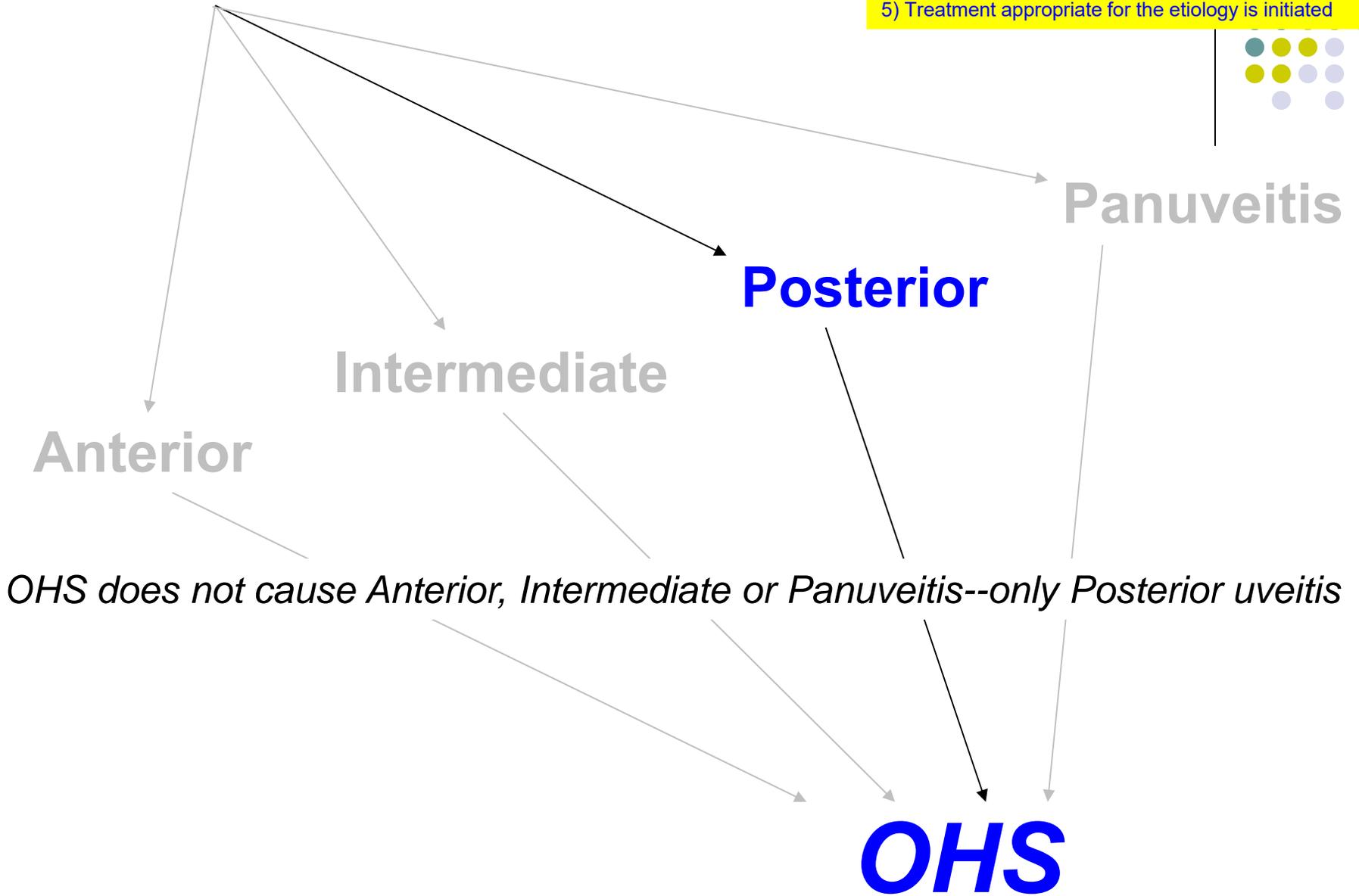
It seems so. Pts positive for HLA-DRw2 or HLA-B7 are much more likely to manifest the condition.

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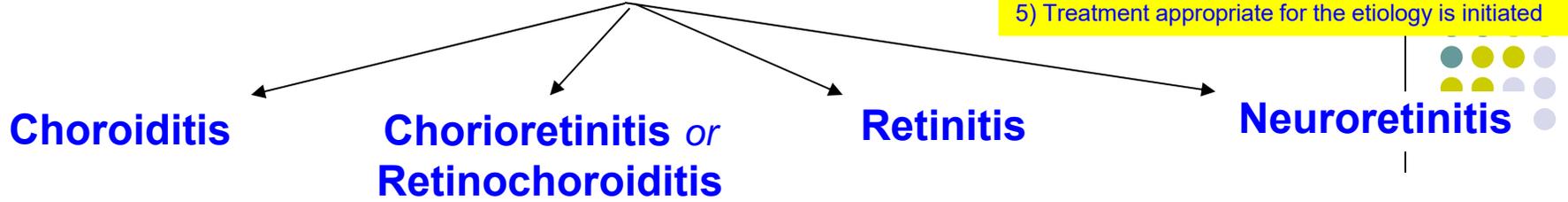
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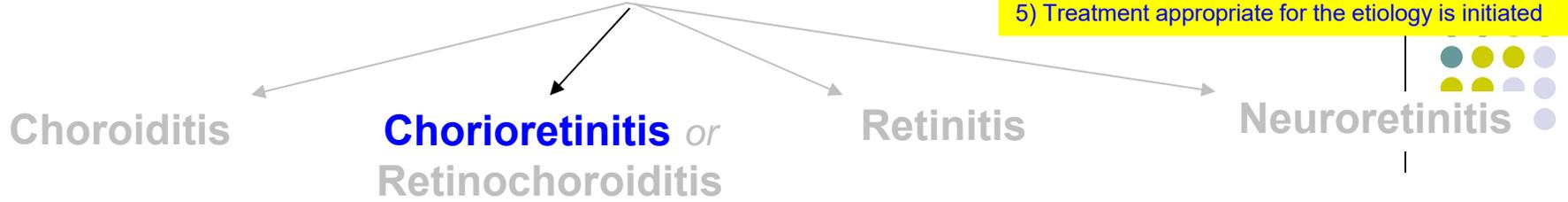
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*What is the classic posterior manifestation of OHS?*

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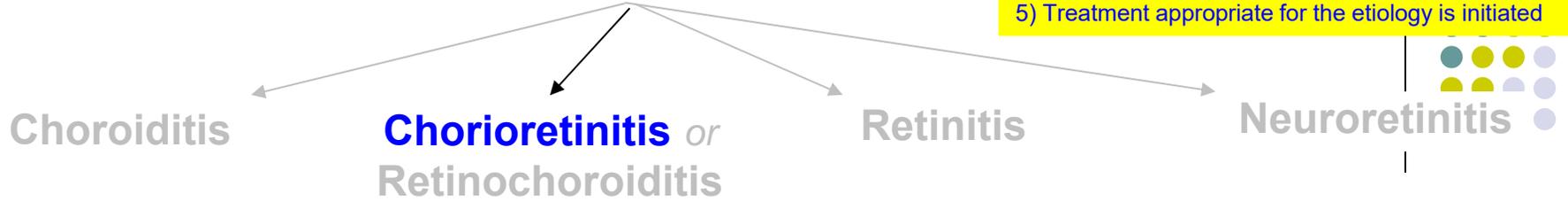
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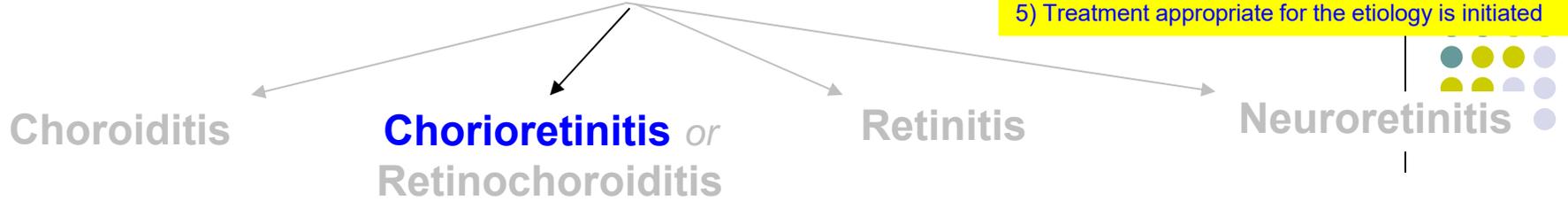


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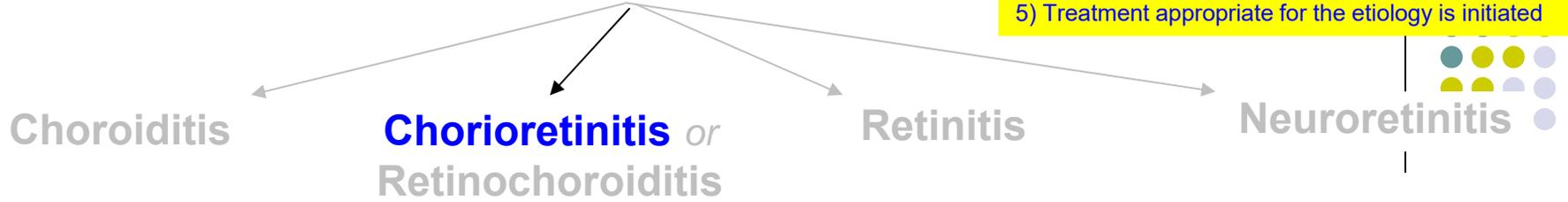


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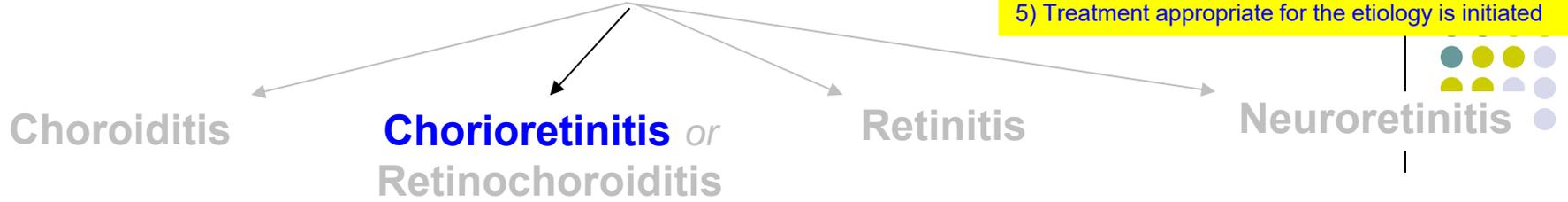


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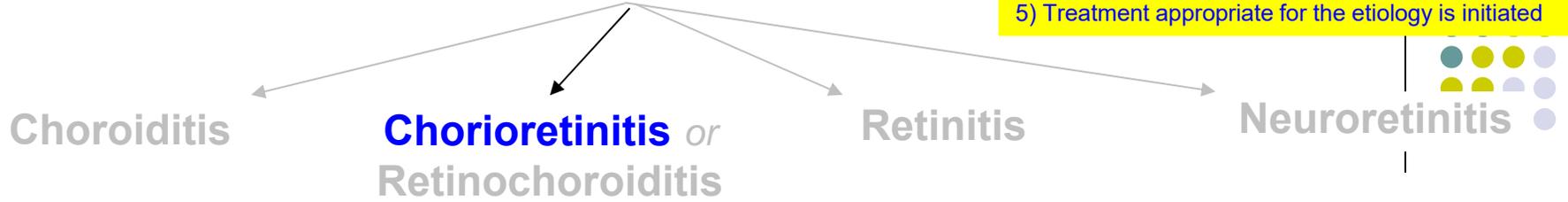


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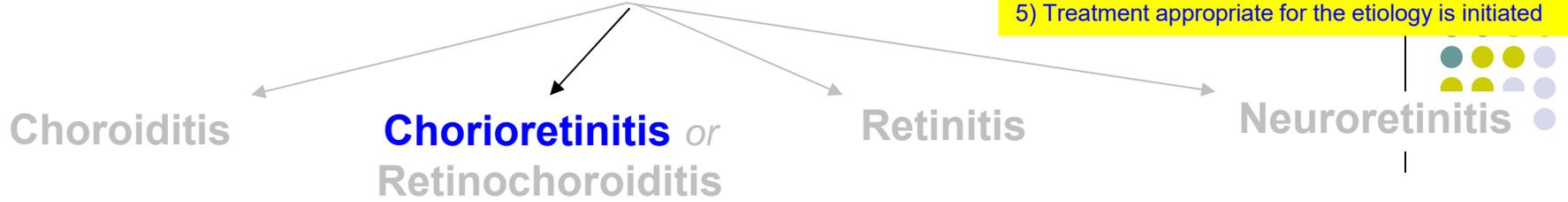


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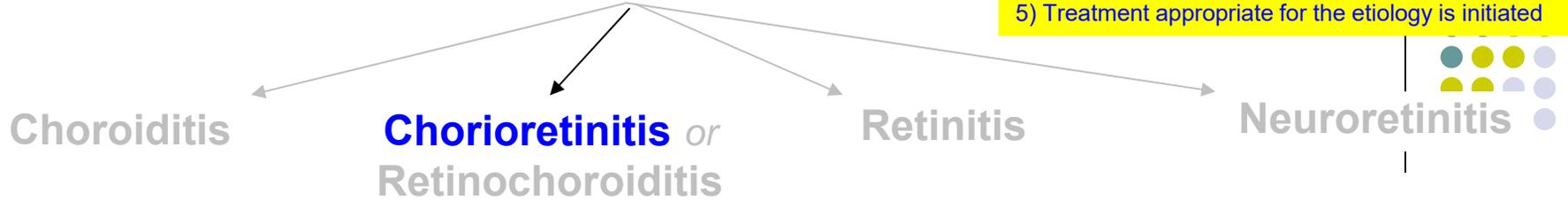


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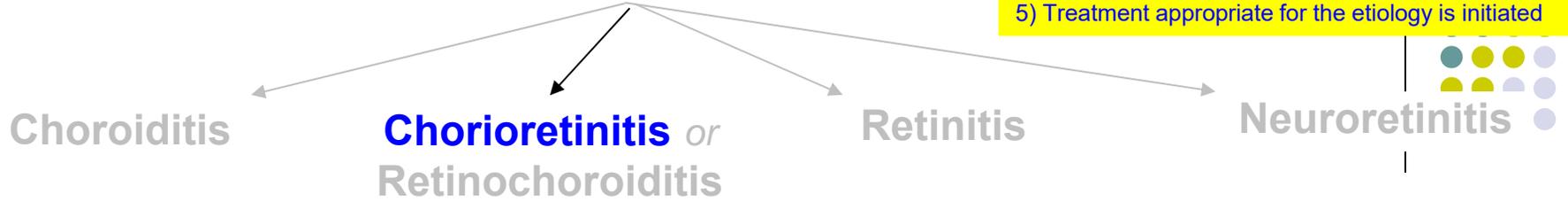


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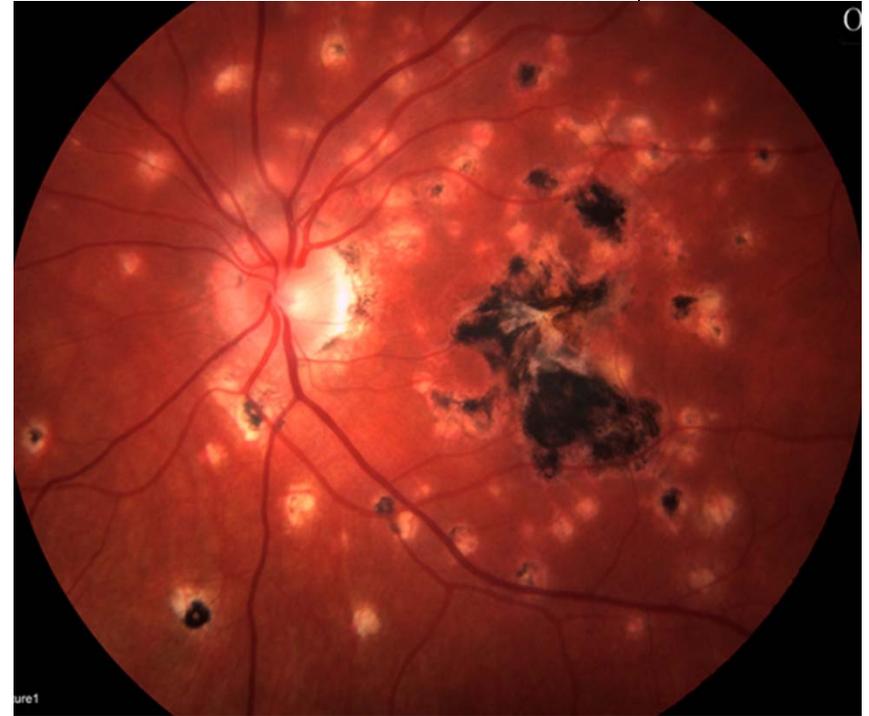
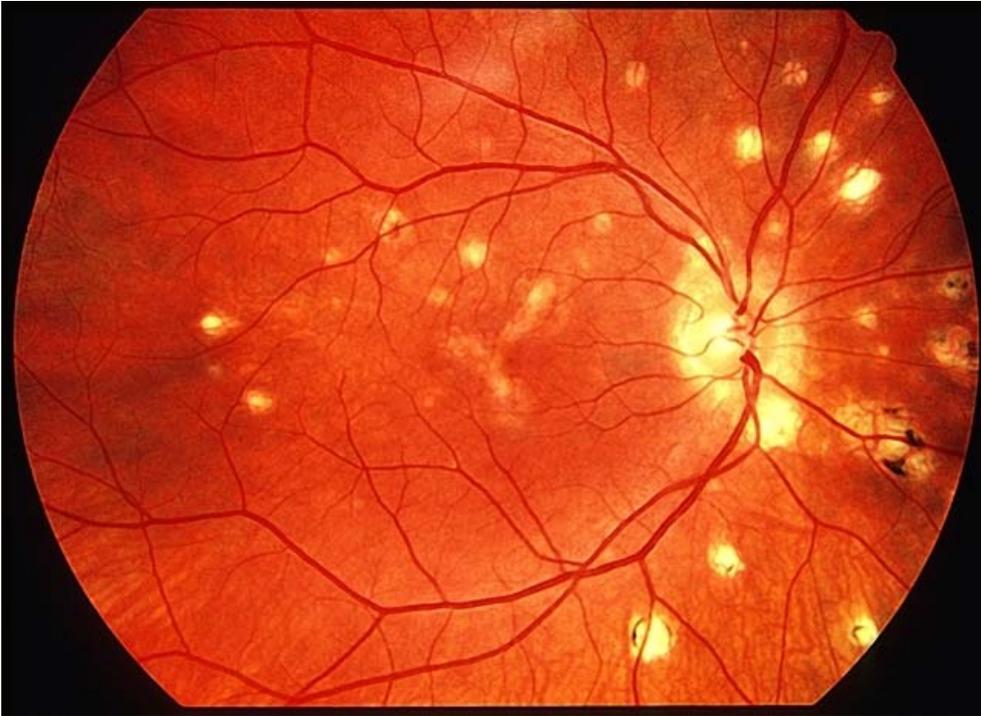
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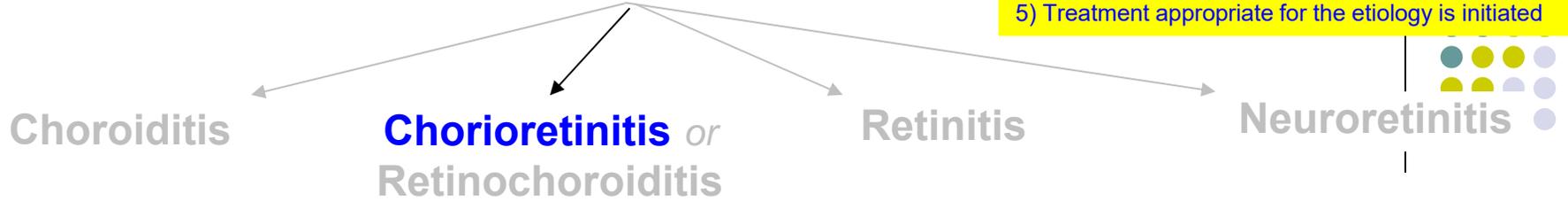
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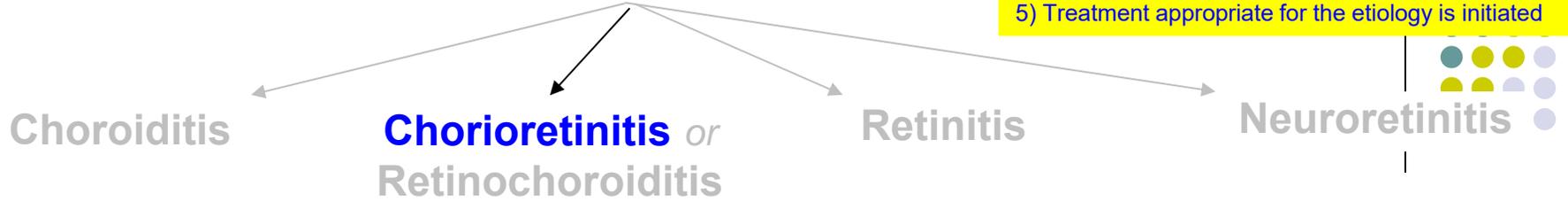


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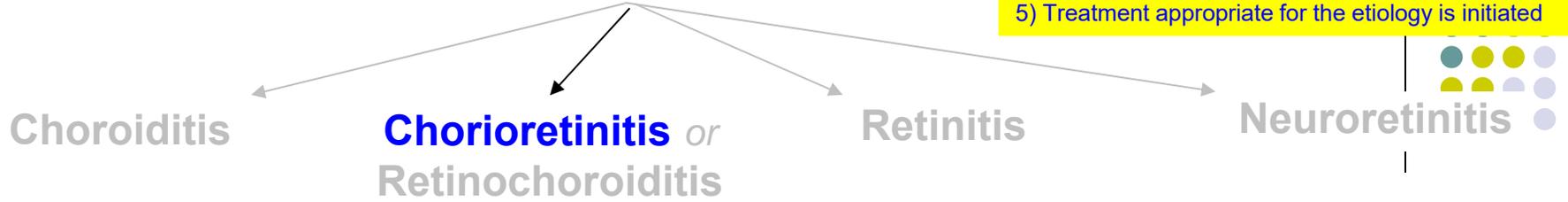


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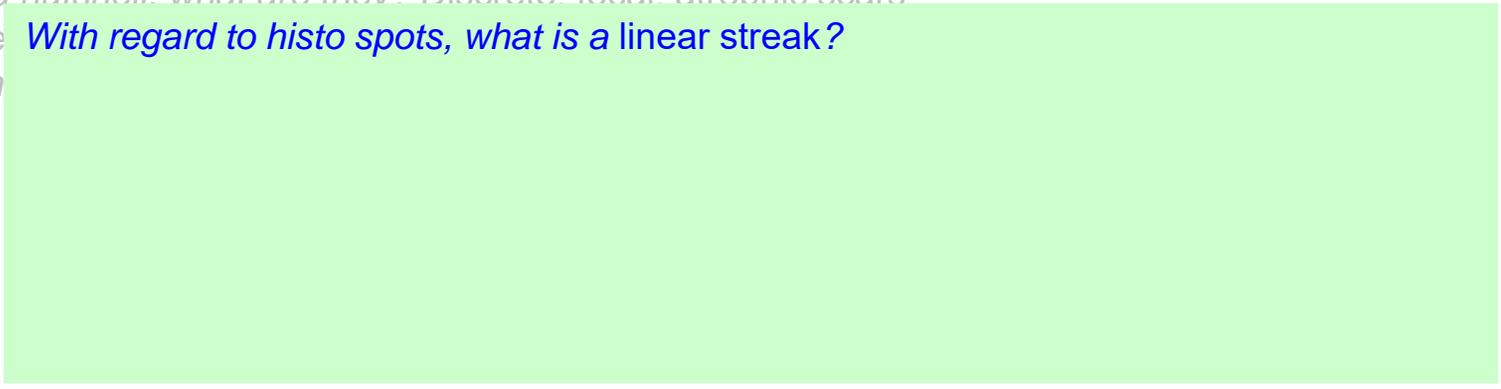
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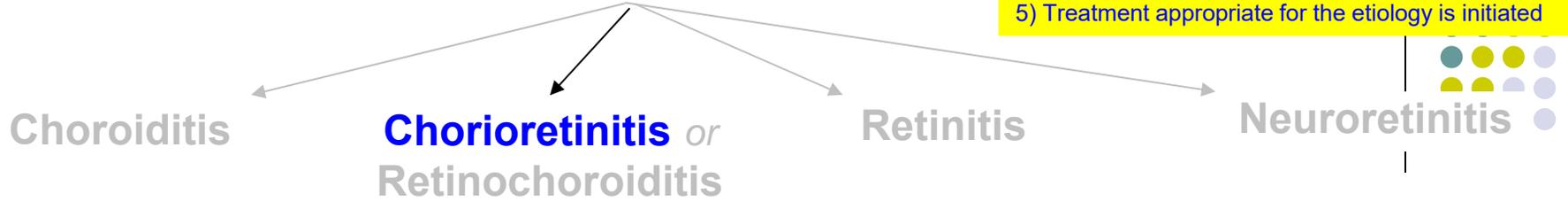
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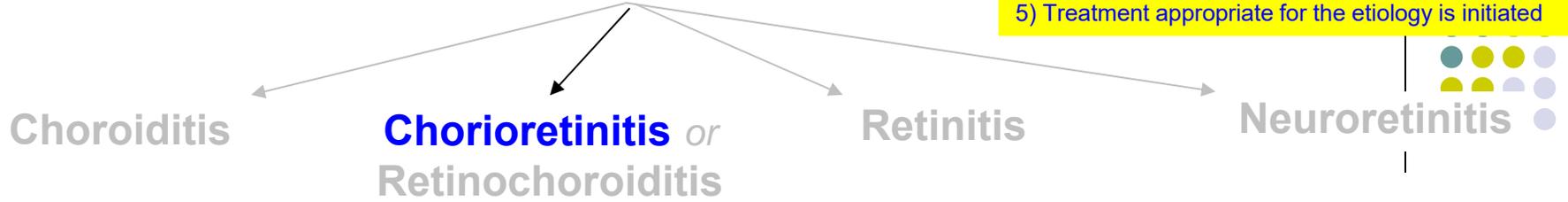
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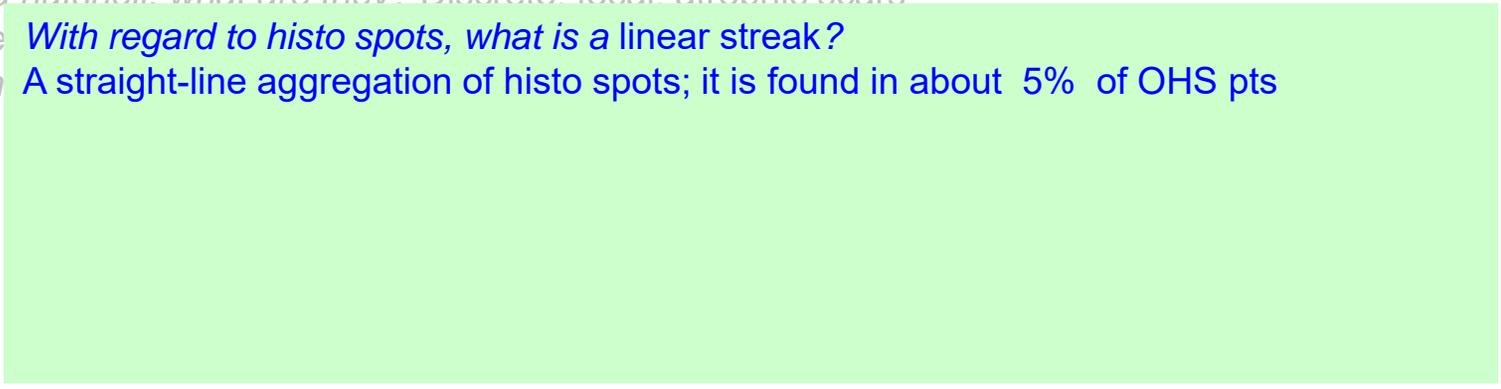
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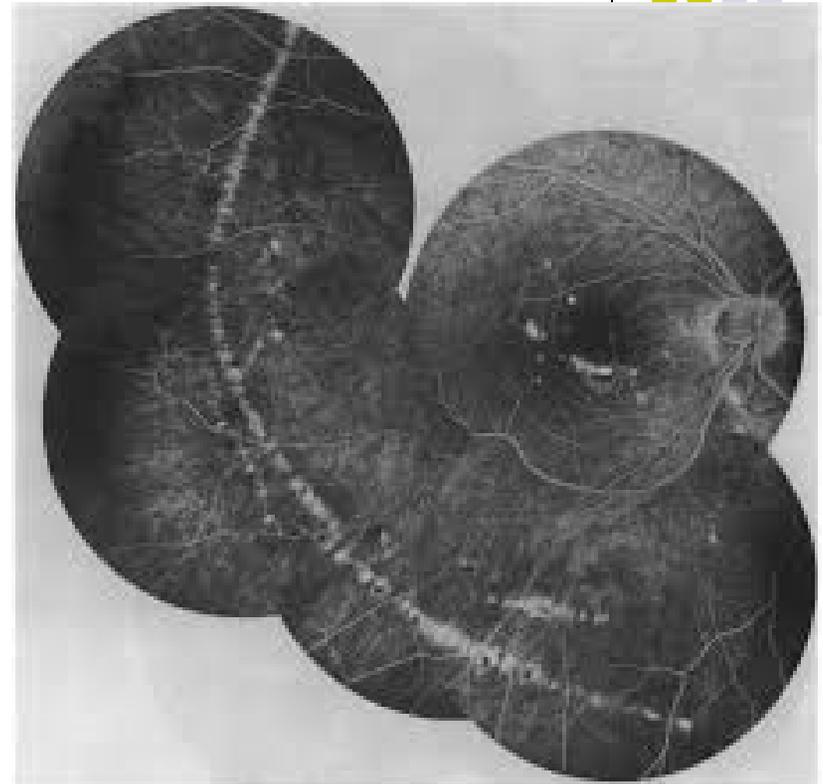
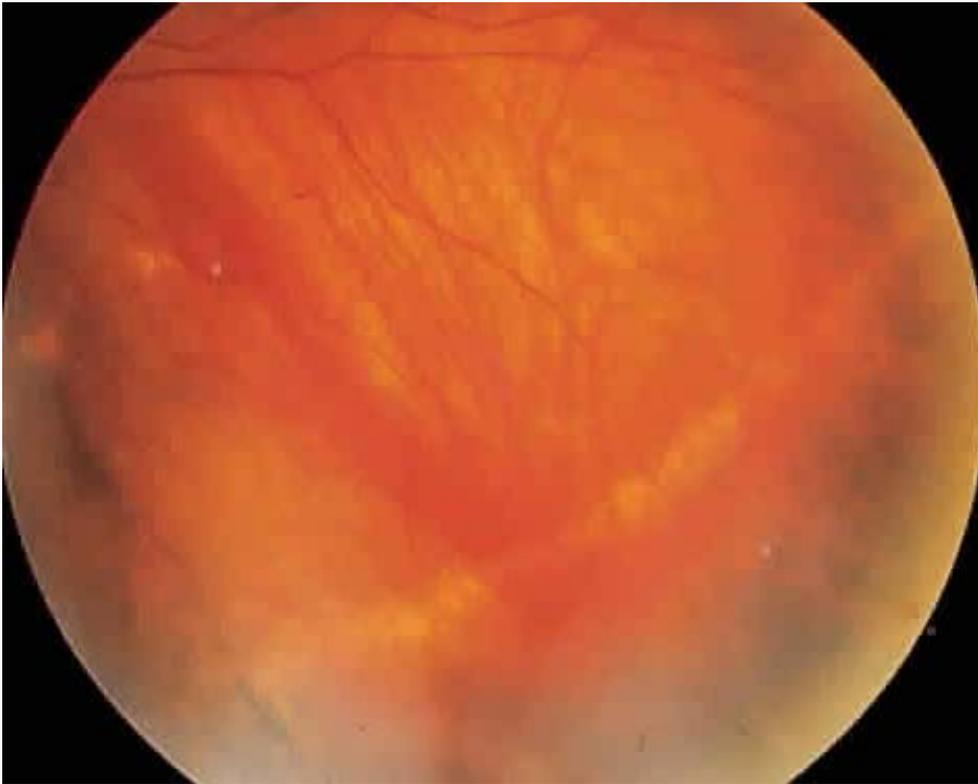
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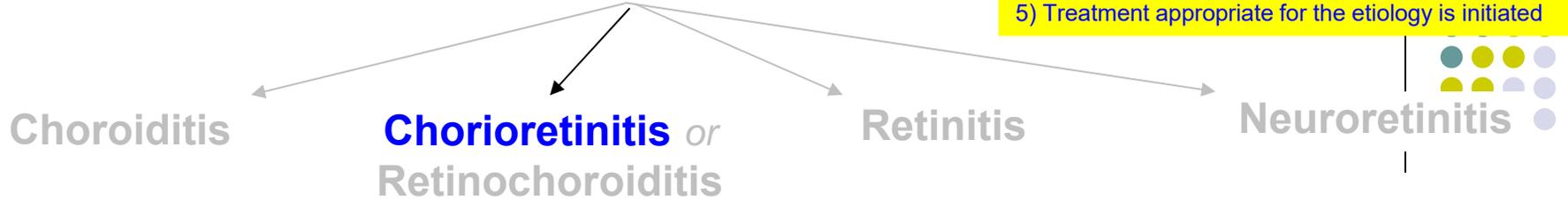




Histo: Linear streaks

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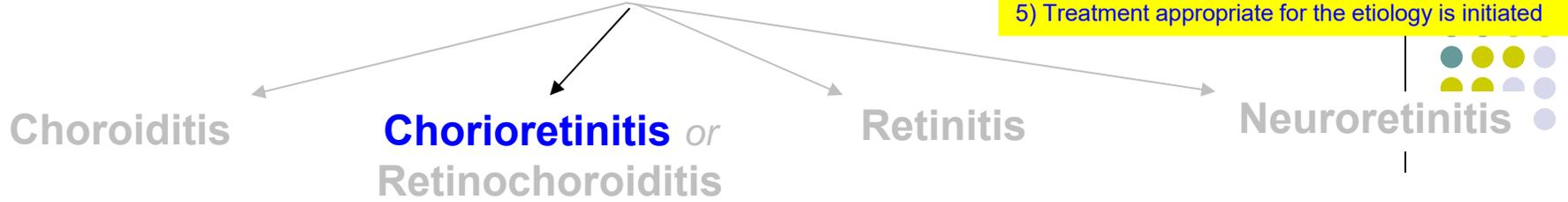
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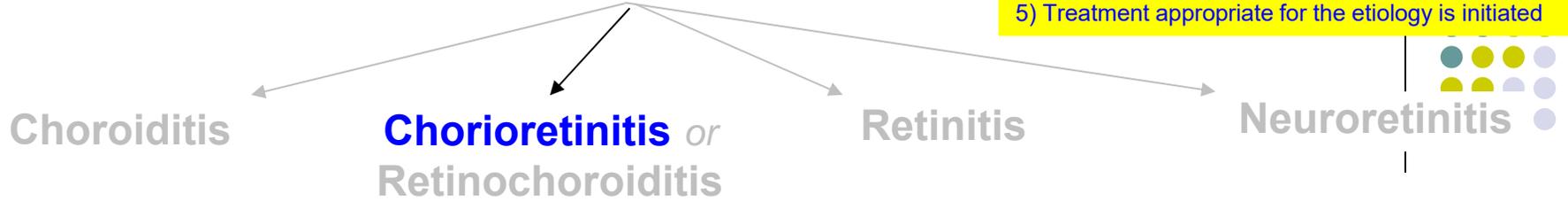
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***At what location with the globe are linear streaks found?***

***At the equator***

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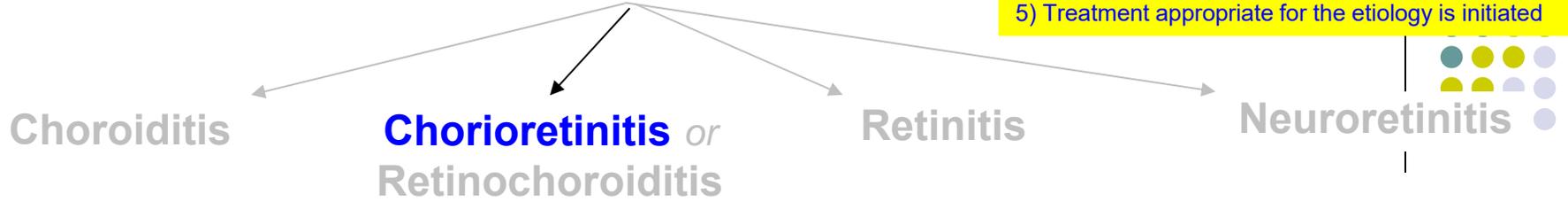
***At what location with the globe are linear streaks found?***

**At the equator**

***How is the streak oriented; ie, is it parallel, or perpendicular, to the equator of the globe?***

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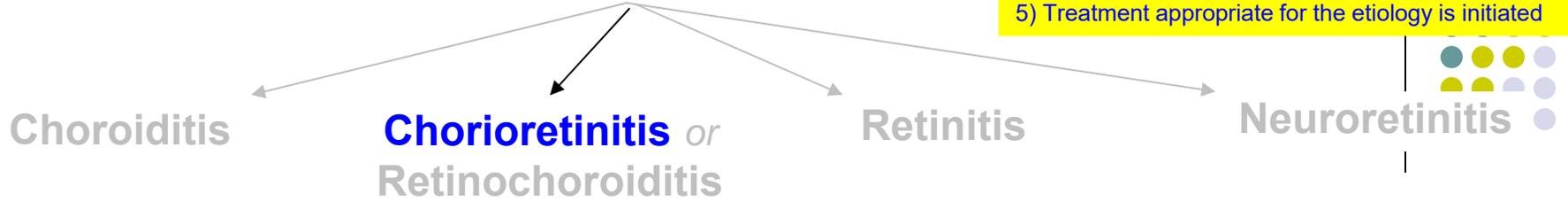
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***How is the streak oriented; ie, is it parallel, or perpendicular, to the equator of the globe?***

**Parallel**

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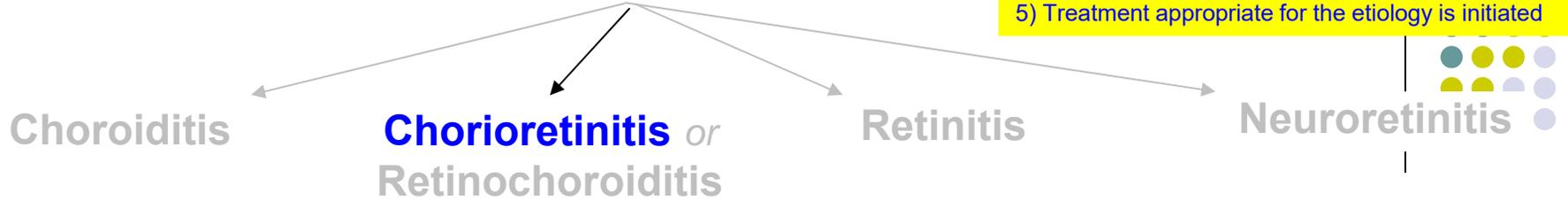
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Re the three lesions of OHS: **Peripapillary atrophy**  
*What specific structure(s) is/are involved?*

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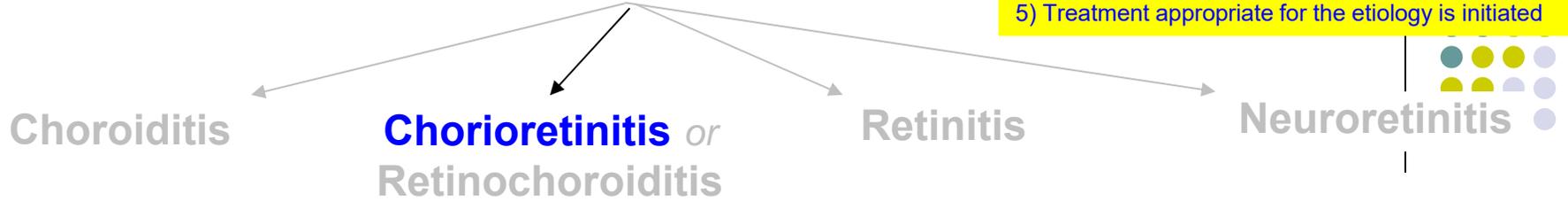
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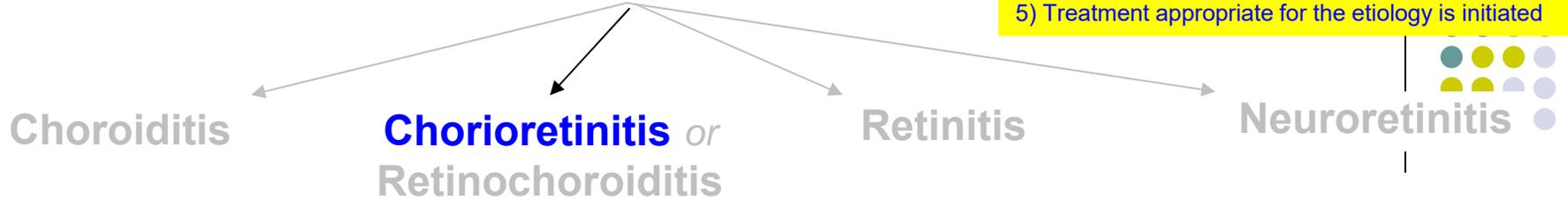
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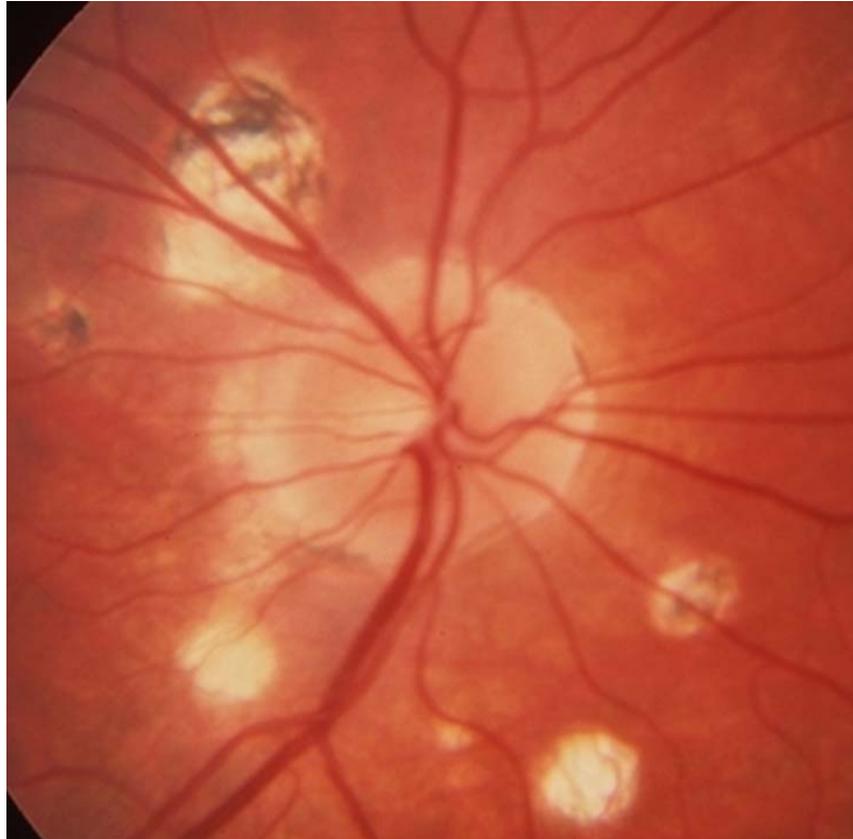
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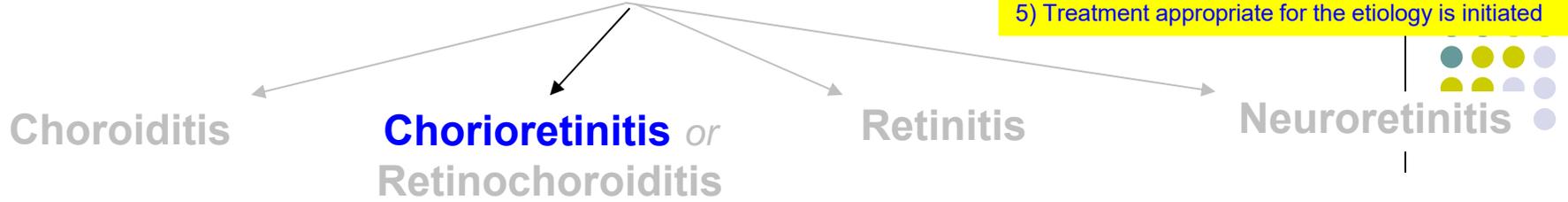
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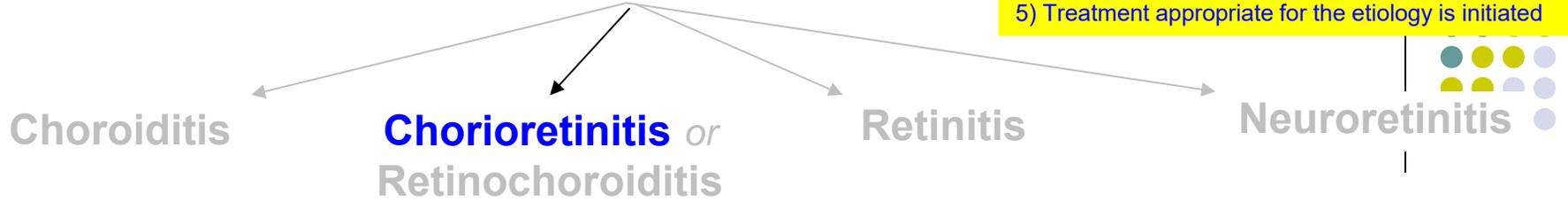
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Re the three lesions of OHS: **Macular disciform lesions**  
*What specific structure(s) is/are involved?*

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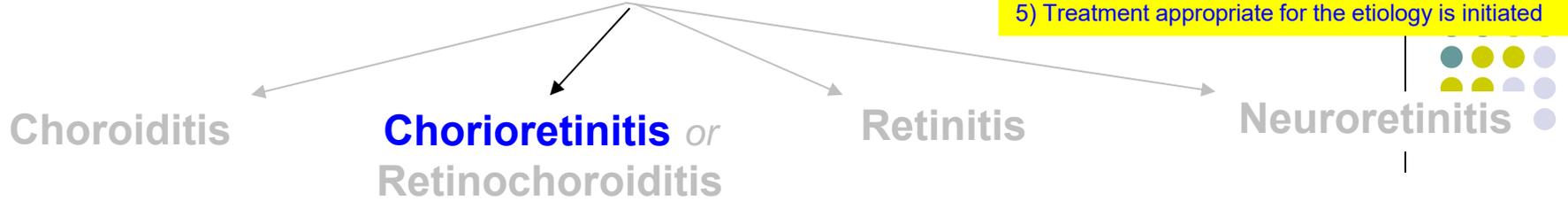
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*In a nutshell, what are they?* Discrete, focal, atrophic scars  
*Are histo spots larger, or smaller than the ONH?* Smaller  
*What two-word phrase is used to describe them?* 'Punched out'  
*Do they evolve over time?* Generally no

Re the three lesions of OHS: **Peripapillary atrophy**  
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Re the three lesions of OHS: **Macular disciform lesions**  
*What specific structure(s) is/are involved?* Everything, including a defect in Bruch's membrane

# Uveitis: *Posterior*

- 1) The uveitis is profiled
- 2) The profiled case is meshed
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*What is the classic posterior manifestation of OHS?*  
A multifocal chorioretinitis

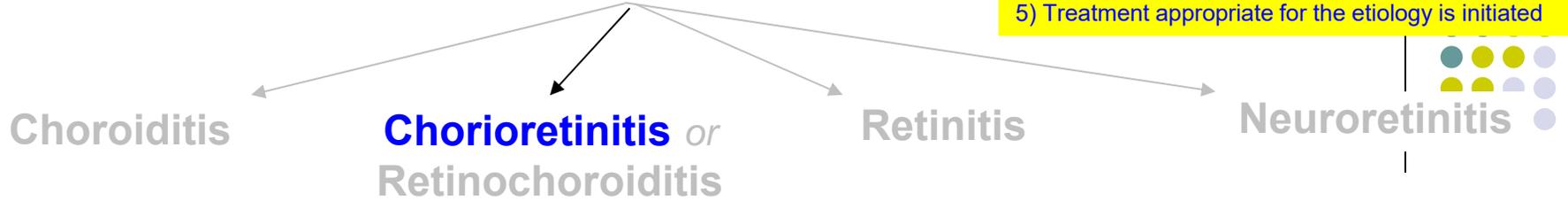
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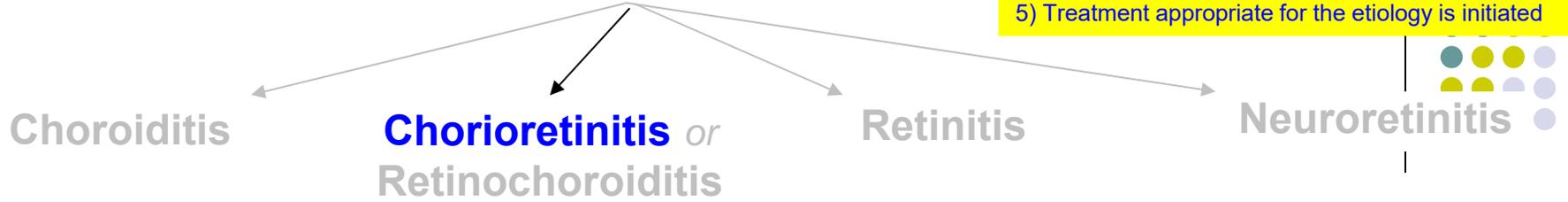
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Histo: Macular disciform lesion

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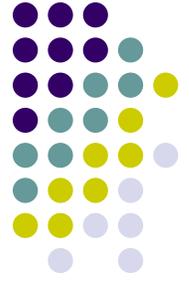
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Histo: Macular disciform lesion



Histo: Macular disciform lesion

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

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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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*There were a number of locations:*

### --Lesion location

--The specific underlying

--Whether the lesion

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--Extrafoveal

--Juxtafoveal

--Subfoveal

--Papillomacular bundle

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--Extrafoveal: Posterior edge of the CNVM >200 mm from foveal center

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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?*

Percent of eyes experiencing

*How was lesion location defined; ie, in terms of what structure?*

With respect to distance from the foveal center

*There were a number of locations:*

### --Lesion location

--The specific underlying

--Whether the lesion

*Four locations were used. What were they? **How were they defined?***

--Extrafoveal: Posterior edge of the CNVM >200 mm from foveal center

--Juxtafoveal: Posterior edge 1-200 mm from foveal center

--Subfoveal:

--Papillomacular bundle

*What treatment modalities are used to treat active disciform lesions?*

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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:*

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

--Whether the lesion was subfoveal or non-subfoveal

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

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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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--Transient **skin photosensitivity** (pts should avoid sunlight for 5 days post-PDT)

--Infusion-related two words pain

# Uveitis: OHS

## Diagnosis

*How is the diagnosis of OHS made?*

It is a clinical diagnosis based on DFE findings

- 1) The uveitis is profiled
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*What is the basic mechanism of PDT? How is it performed; how does it work?*

PDT destroys the CNVM net 'from the inside.' A light-sensitive chemical is injected intravascularly (note: NOT intravitreally). Time enough for the chemical to accumulate in the net is allowed to pass, then the net is illuminated with a low-power laser tuned to the specific wavelength the chemical is sensitive to. When activated by the light, the chemical reacts with nearby oxygen molecules to produce highly volatile oxygen/hydroxyl free radicals. These free radicals induce intralesional platelet aggregation, which in turn leads to thrombosis of the pathologic vasculature. In this way, the abnormal vasculature of the CNVM can be targeted in a relatively selective manner (in sharp contrast to the indiscriminate destruction produced by a thermal laser).

*Is PDT still an acceptable treatment for CNVM associated with OHS?*

Yes, although it is probably best utilized as an adjunctive therapy in concert with anti-VEGF meds

*What treatment modalities are used?*

--Thermal laser

--**Photodynamic therapy (PDT)**

--Anti-VEGF therapy

--Submacular surgery

--Intravitreal corticosteroids

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

*What are the important side effects of PDT?*

--Transient vision disturbances

-- Injection-site adverse effects (eg rash; extravasation)

--Transient **skin photosensitivity** (pts should avoid sunlight for 5 days post-PDT)

--Infusion-related **low-back pain**

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## **--Anti-VEGF therapy**

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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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*Is there clinical evidence indicating the effectiveness of steroids in treating CNVM in OHS?*

Yes (although not of the prospective, RCT sort)

*What is the main drawback to using intravitreal steroids to treat CNVM in OHS?*

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

*Combination therapy*

**--Intravitreal corticosteroids**

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

# Uveitis: *OHS*

## Diagnosis

*How is the diagnosis of OHS made?*

It is a clinical diagnosis based on DFE findings

- 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



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

Fundamentally, CNVM formation is a wound-healing response. And like all wound-healing responses, it is subject to modulation by inflammatory mediators.

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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.

**--Intravitreal corticosteroids**

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# Uveitis: *OHS*

## Diagnosis

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

-- Anti-VEGF + PDT

-- Anti-VEGF + thermal laser

-- PDT + corticosteroids

-- Intravitreal corticosteroids

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

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