

# **Glaucoma After Intraocular Bleed**

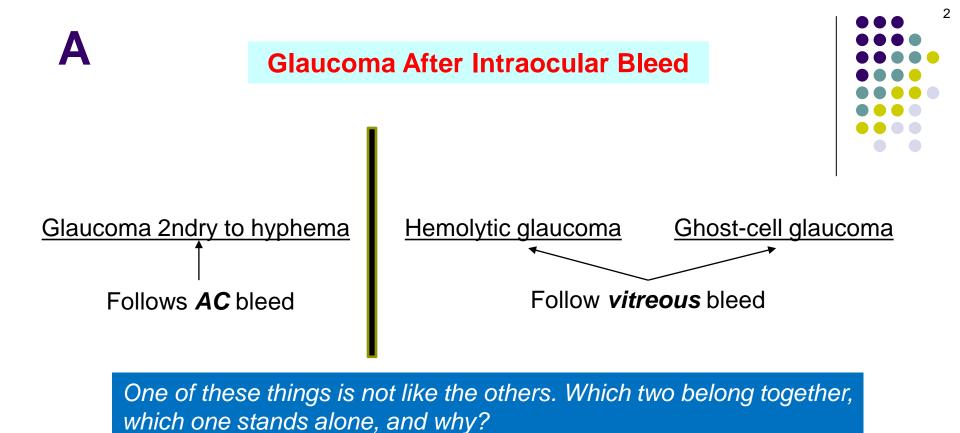


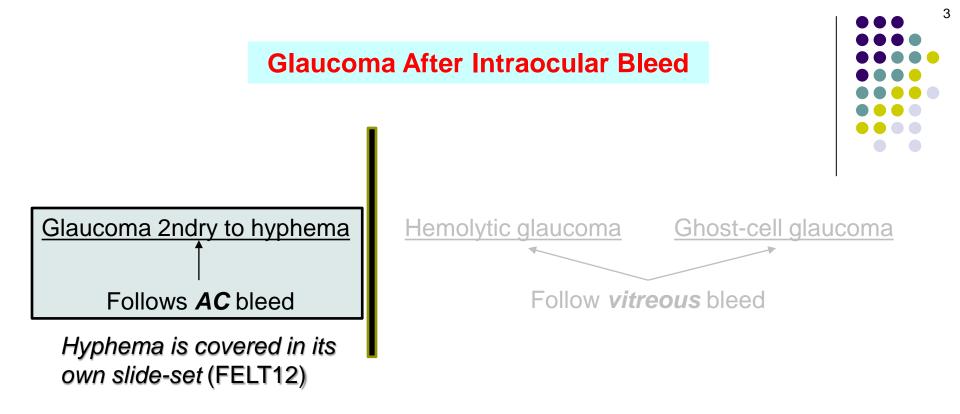
Glaucoma 2ndry to hyphema

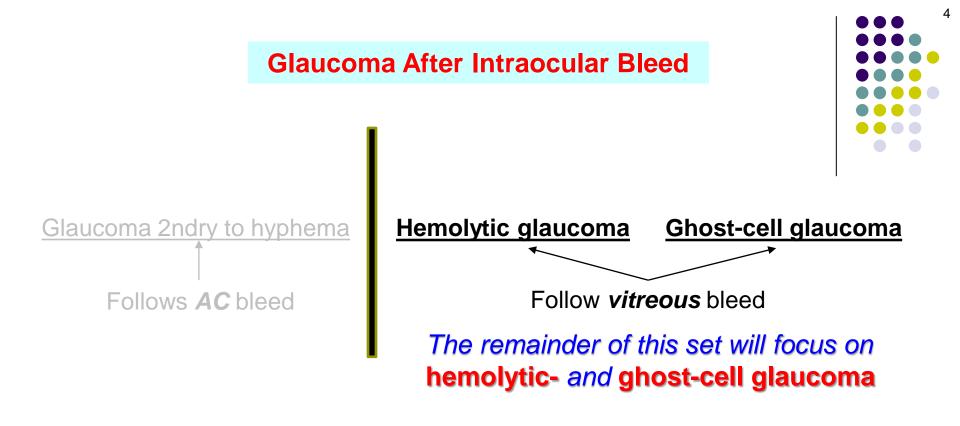
Hemolytic glaucoma

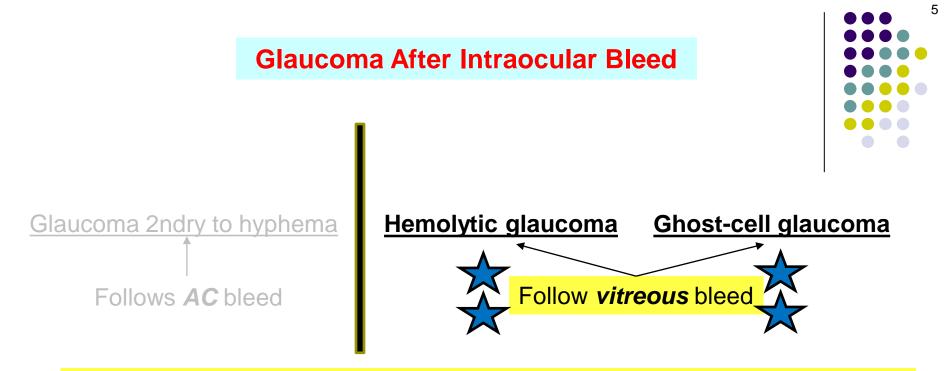
Ghost-cell glaucoma

One of these things is not like the others. Which two belong together, which one stands alone, and why?

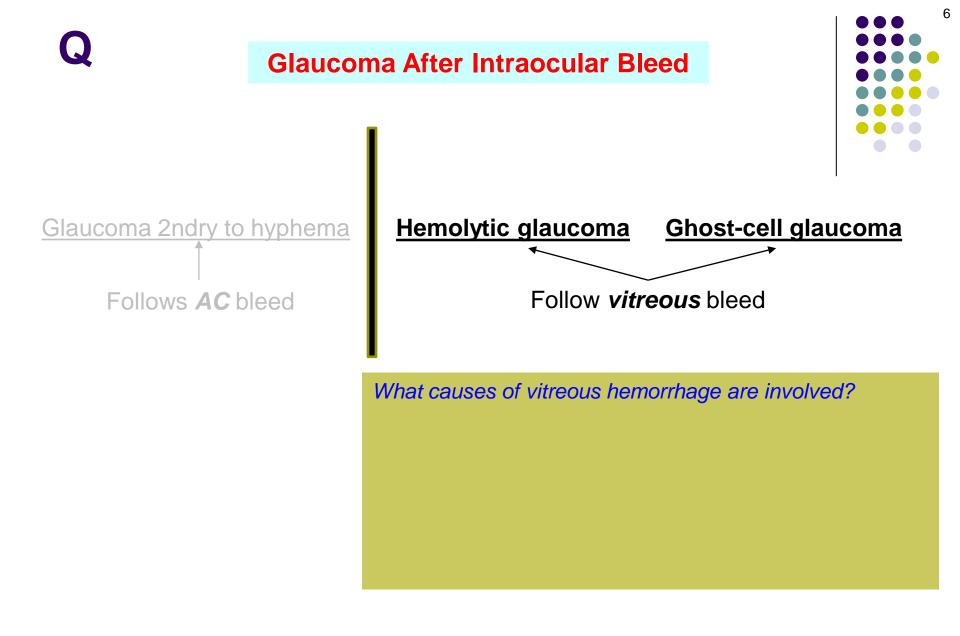


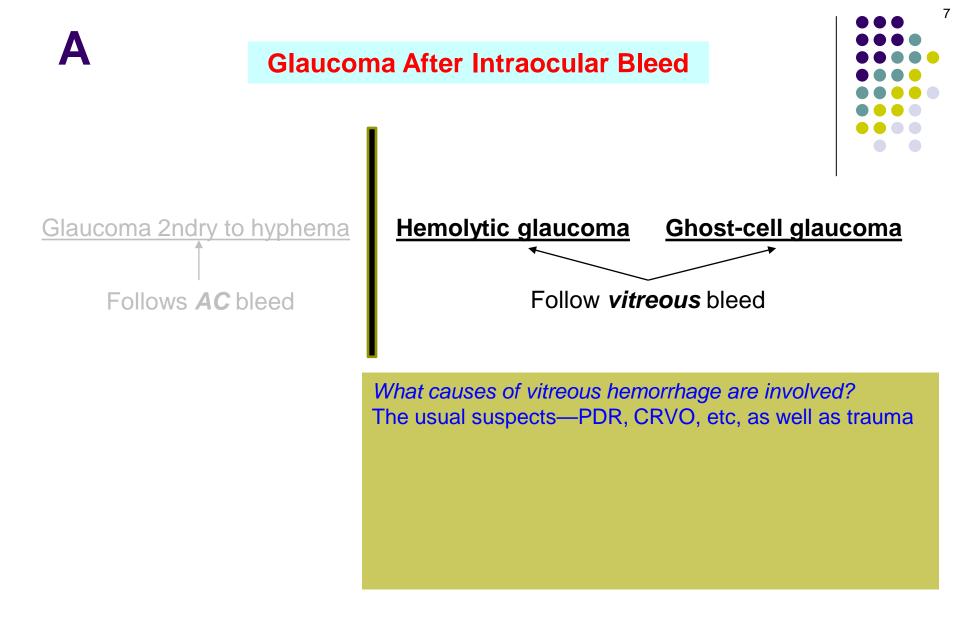


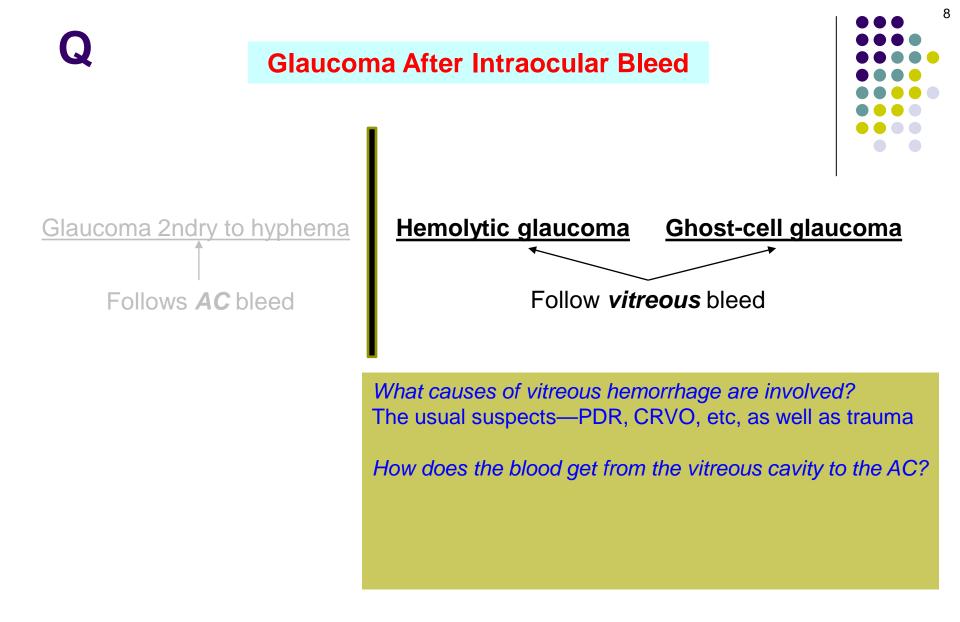


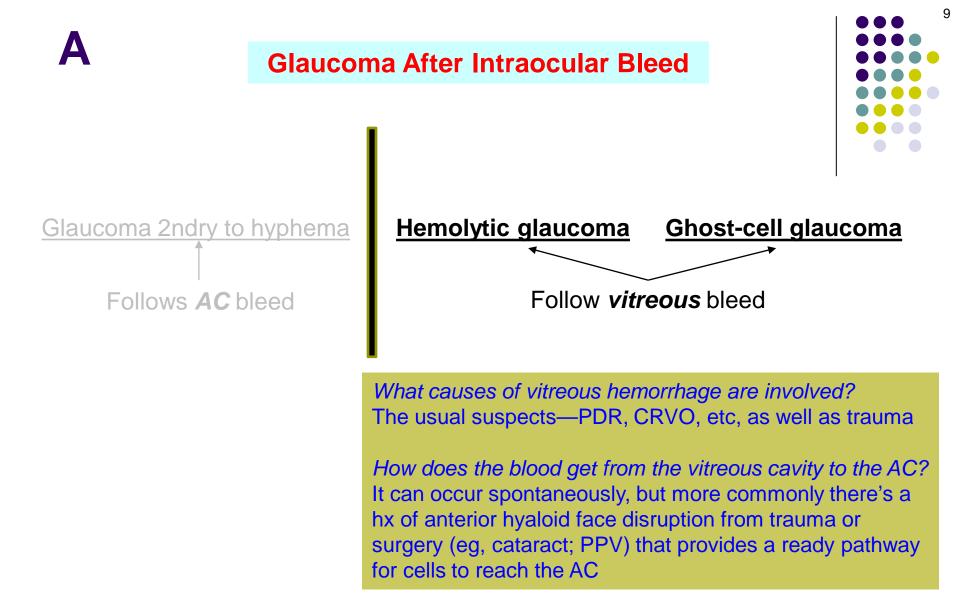


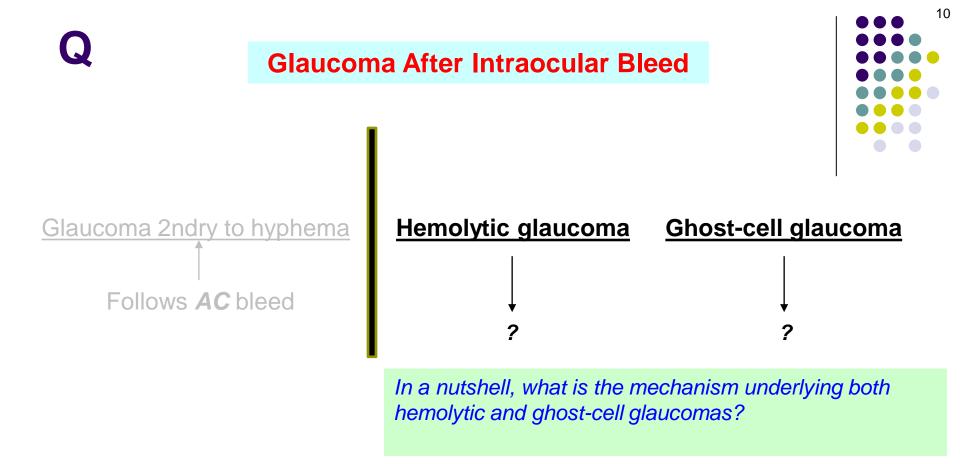
THIS IS IMPORTANT! Take a moment to file a mental note before proceeding: Hemolytic- and ghost-cell glaucoma follow a *vitreous* bleed, not an *AC* bleed!

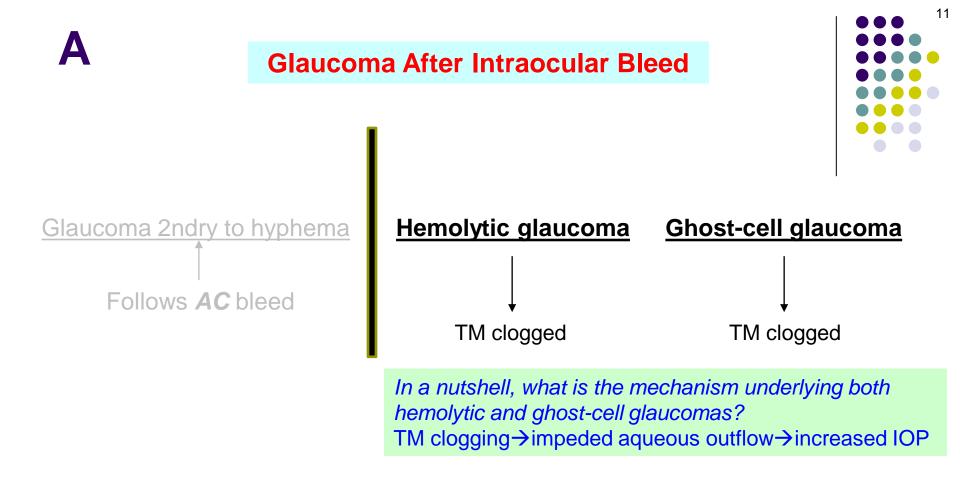


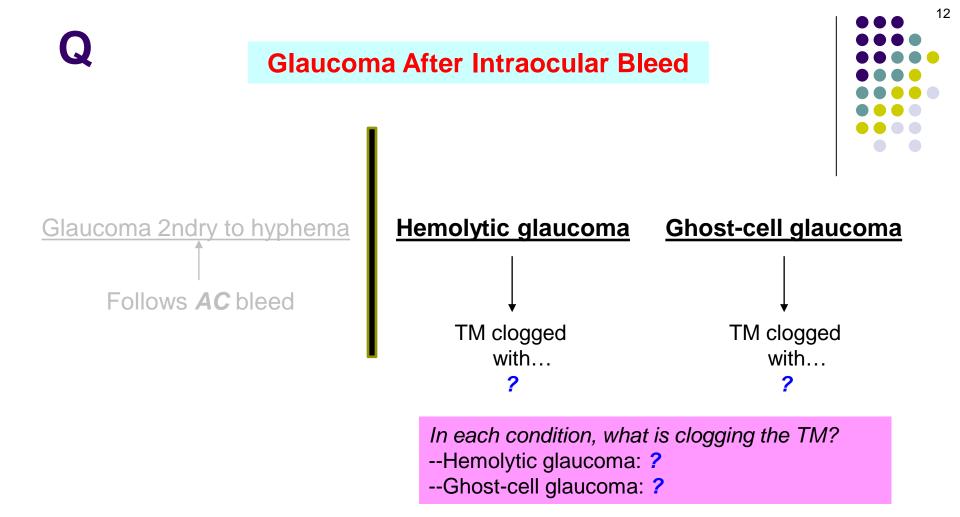


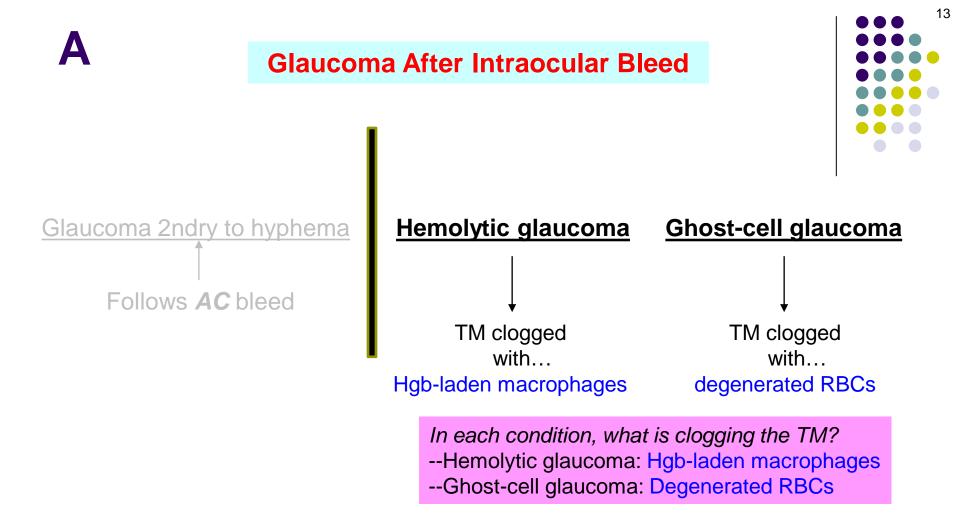


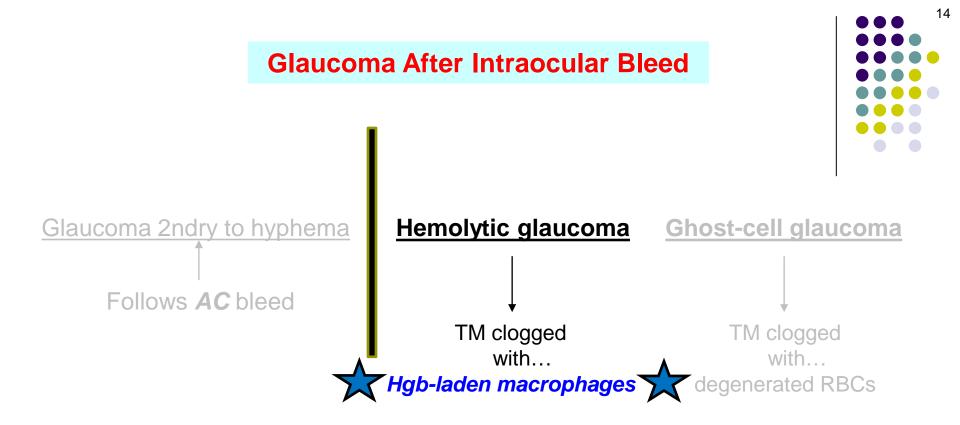




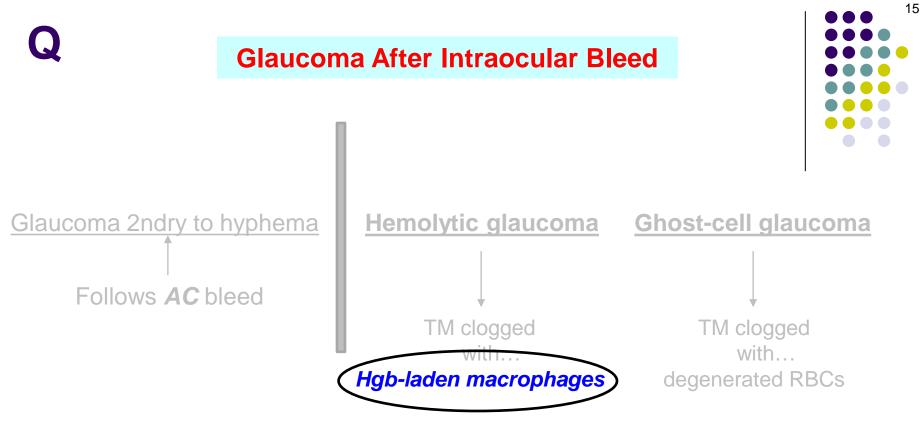




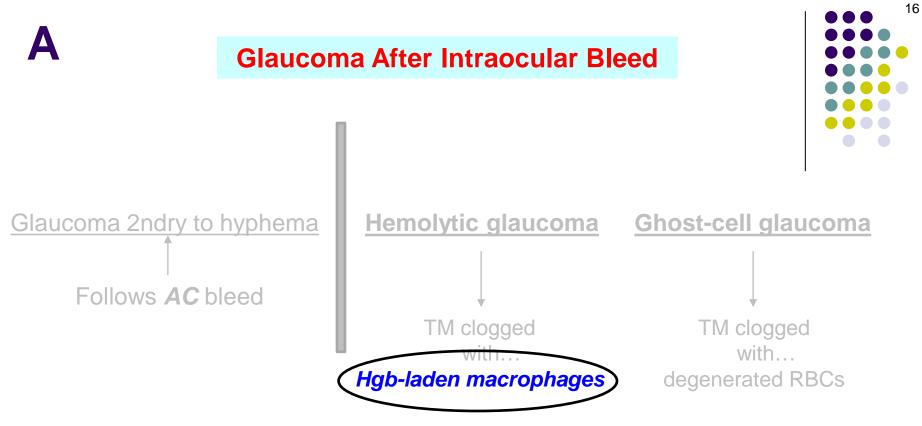




Make another mental note: While ghost-cell glaucoma involves RBCs as would be expected in a hemorrhage-related condition, in hemolytic glaucoma the culprit is not RBCs—it's macrophages



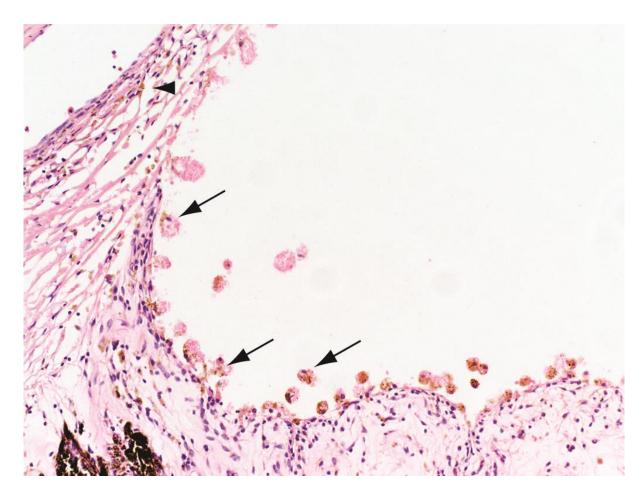
What's up with the macrophages? How do they figure in all this?



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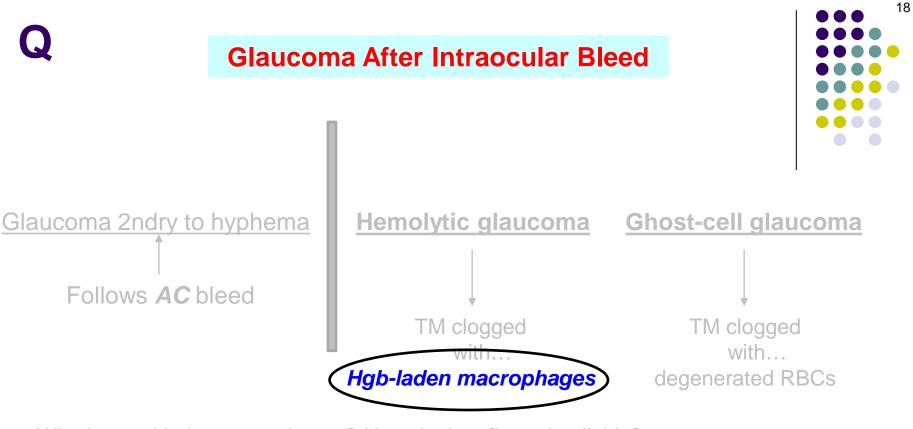
RBCs in a vitreous hemorrhage start to break down after a week or two. The degeneration of these cells attracts macrophages, which consume both the effete RBCs as well as the hemoglobin-related material they release. Heavy-laden with globules of degenerated Hgb and other RBC detritus, these macrophages end up in the AC, and ultimately the angle.

# **Glaucoma After Intraocular Bleed**

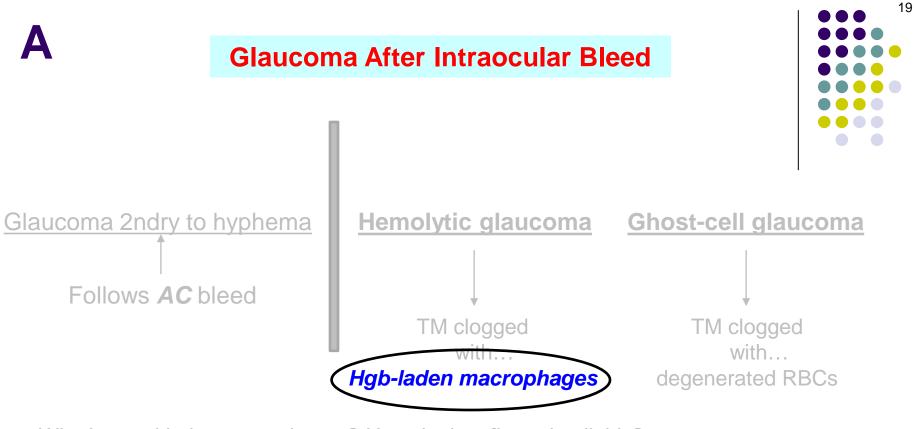


**Hemolytic glaucoma**. The anterior chamber angle contains macrophages with erythrocytic debris and rust-colored intracytoplasmic material (arrows).



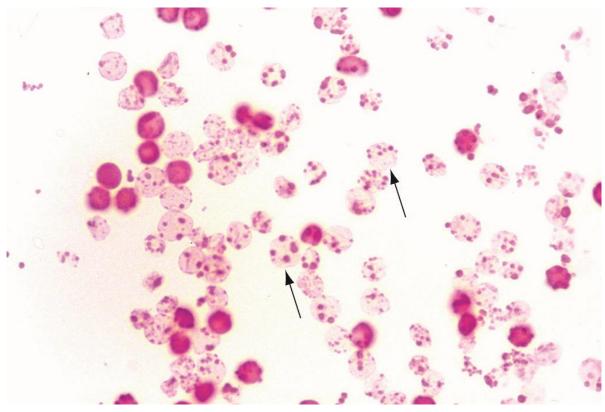


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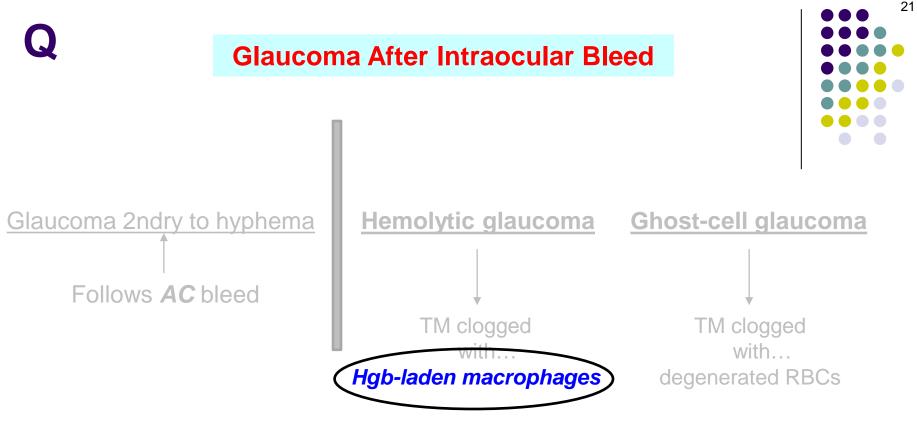
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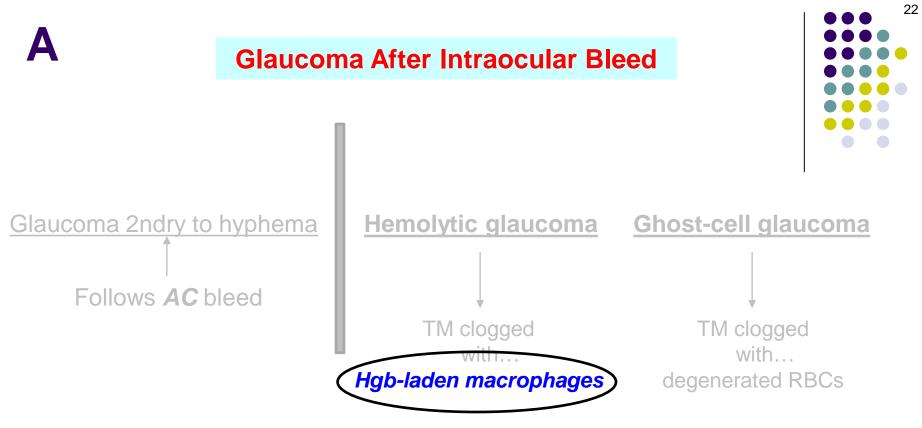
**Hemolytic glaucoma**. The degenerating hemoglobin is present as small globules known as Heinz bodies (arrows).





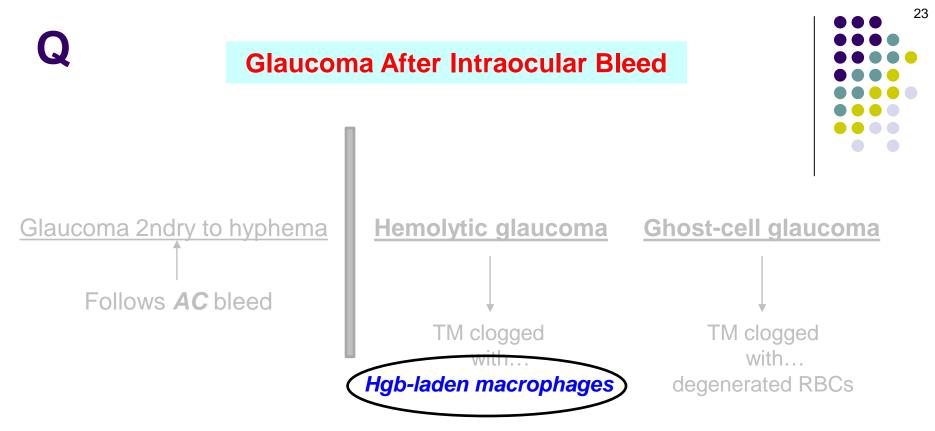
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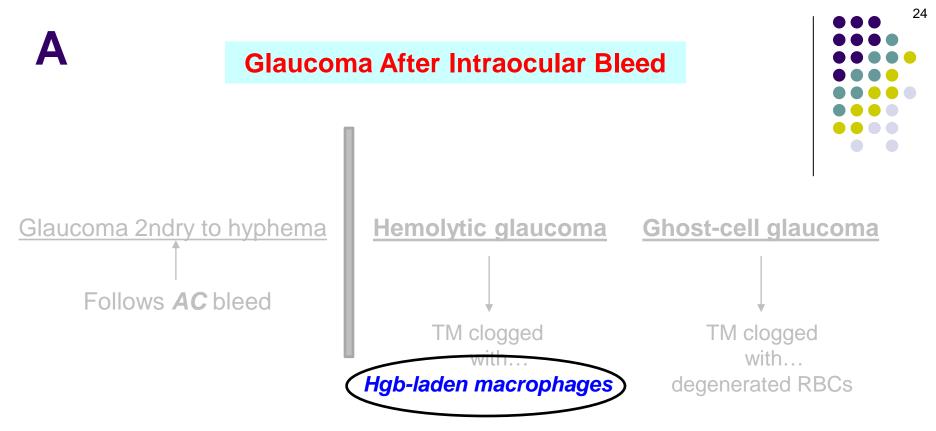
'Heinz bodies'? *Bruh, the BCSC* Glaucoma *book does* **not** *mention Heinz bodies. Why are you including details we don't need to know?* I wouldn't do you like that bruh—the *Pathology* book mentions Heinz bodies in *its* discussion of hemolytic- and ghost-cell glaucoma, so it's fair game for the OKAP



What's up with the macrophages? How do they figure in all this? 'Macrophages clogging the TM' should bring to mind another form of 2ndry OAG—what is it?

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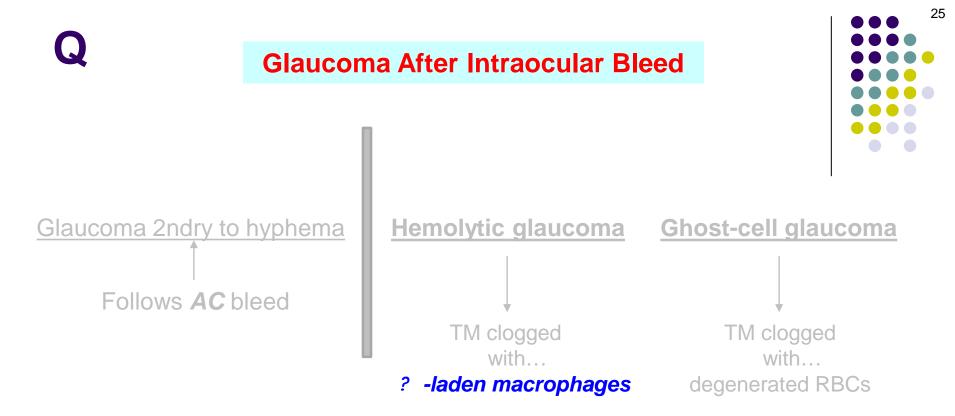
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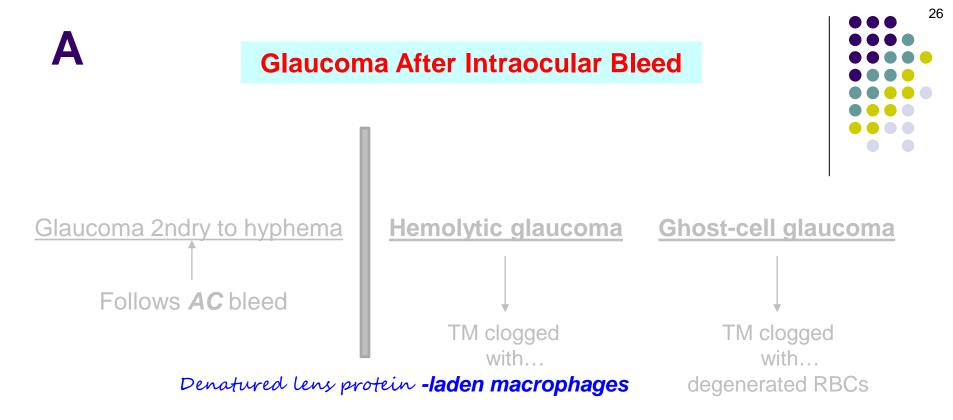


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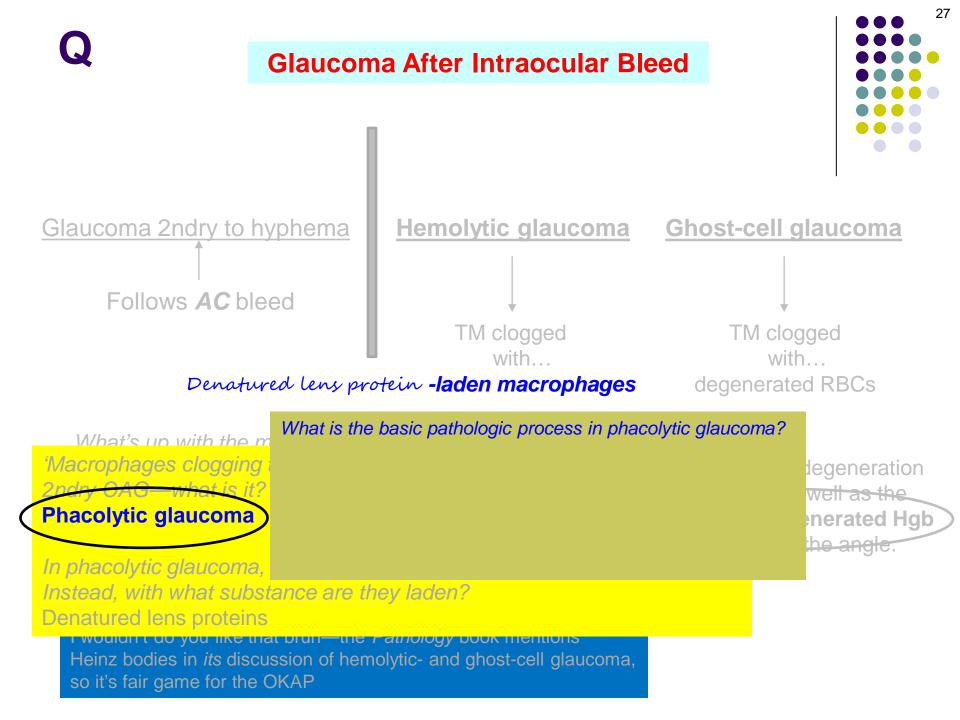


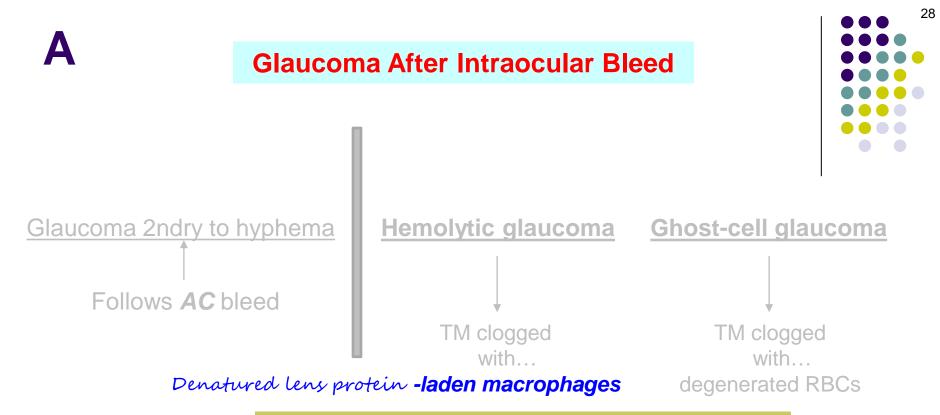
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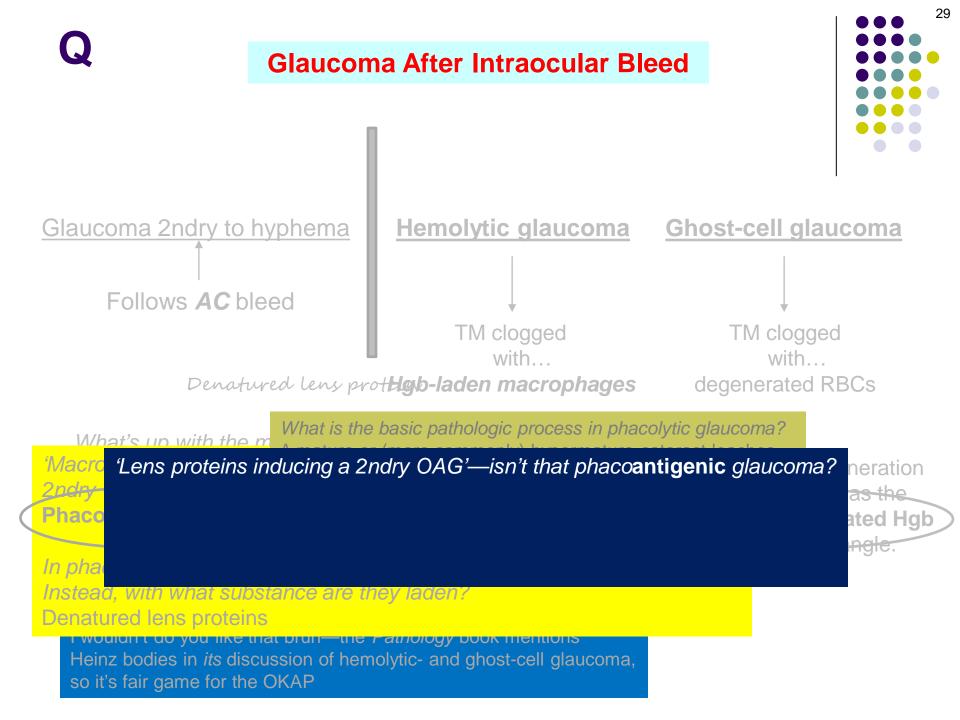
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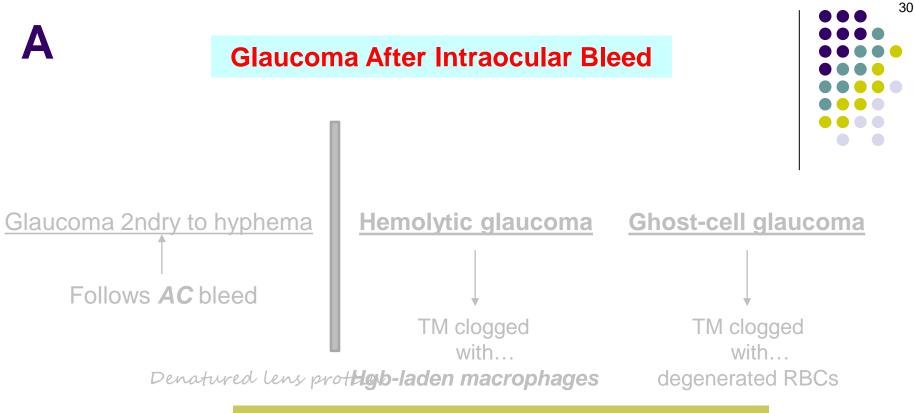
What is the basic pathologic process in phacolytic glaucoma? A mature or (more commonly) hypermature cataract leaches denatured lens proteins through its lens capsule. The proteins attract macrophages, which attempt to clear the protein from the AC. The protein-laden macrophages (and the protein) are swept into the angle, where they clog the TM and cause an IOP spike.

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#### What is the basic pathologic process in phacolytic glaucoma?

Macro 'Lens proteins inducing a 2ndry OAG'—isn't that phacoantigenic glaucoma? neration No. Phacoantigenic glaucoma is a rare condition in which the appearance 2pdr/ **Phace** of *normal* (ie, not denatured) lens proteins in the AC after a breach in the anterior capsule (either traumatic or surgical) provokes a granulomatous *In pha* immune response.

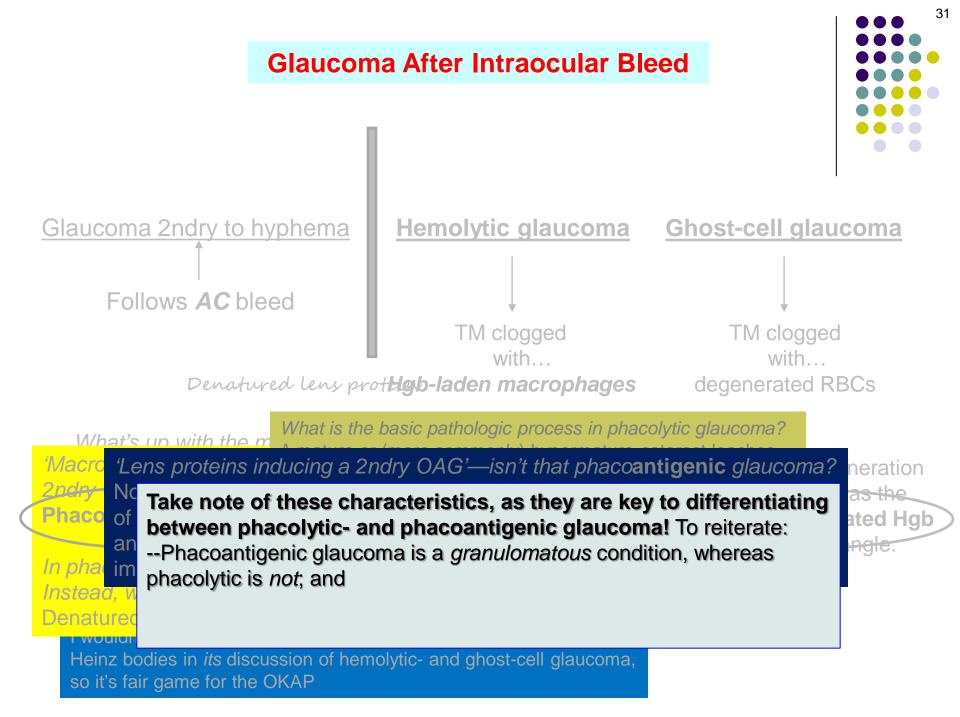
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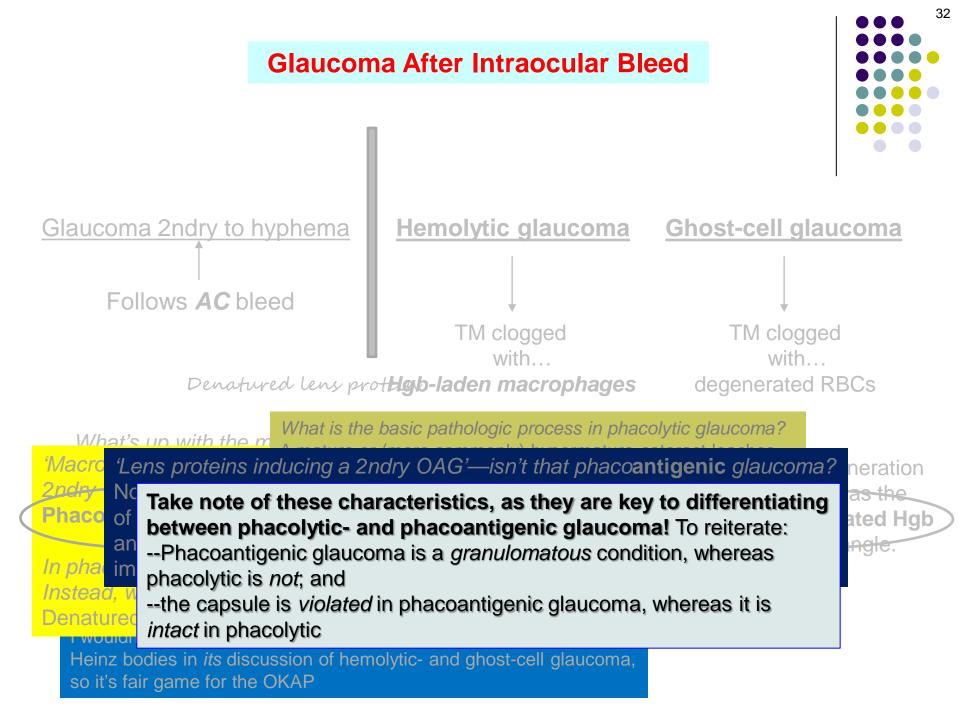
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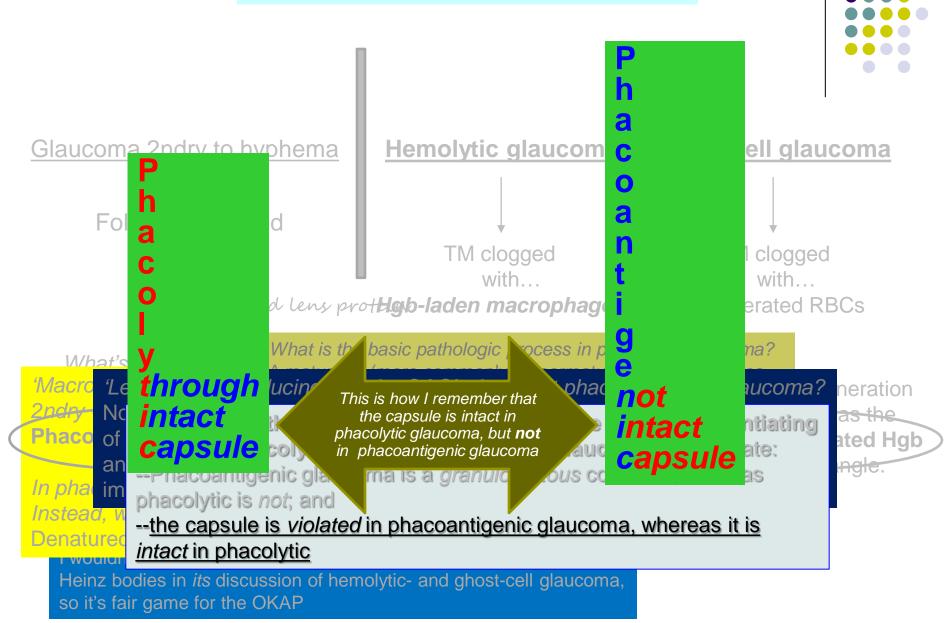
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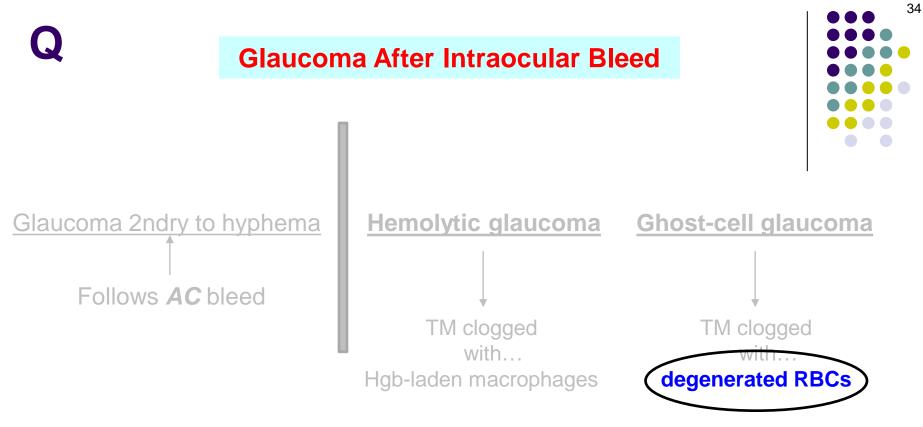
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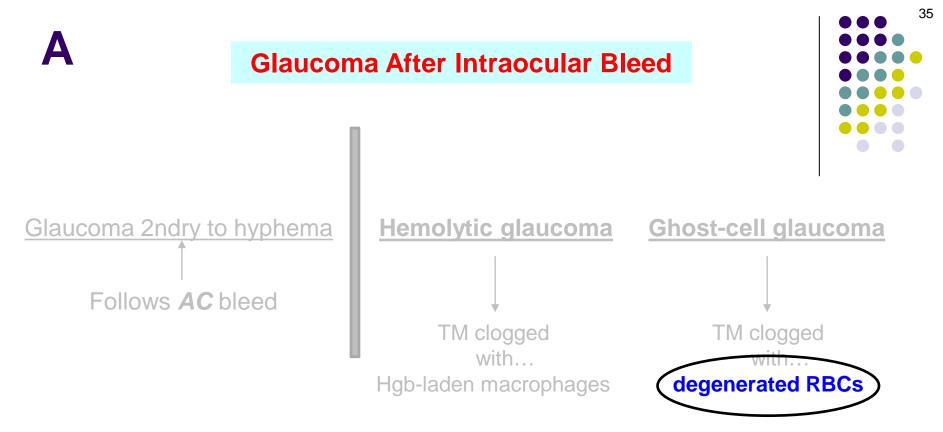


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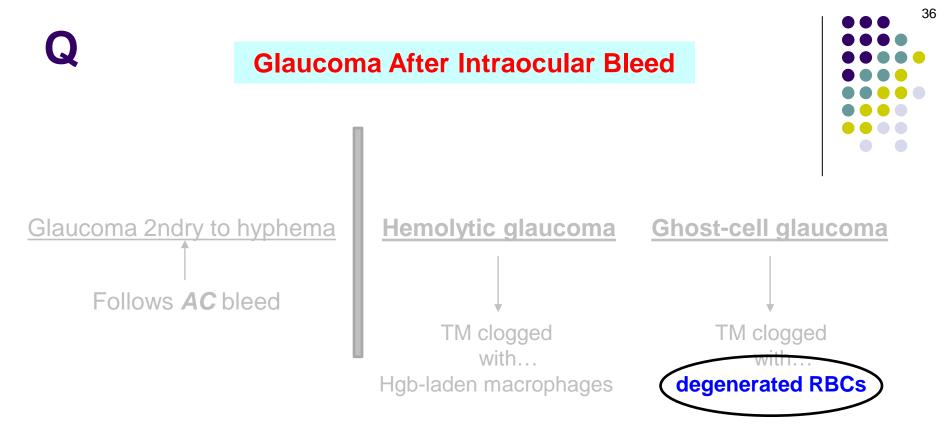




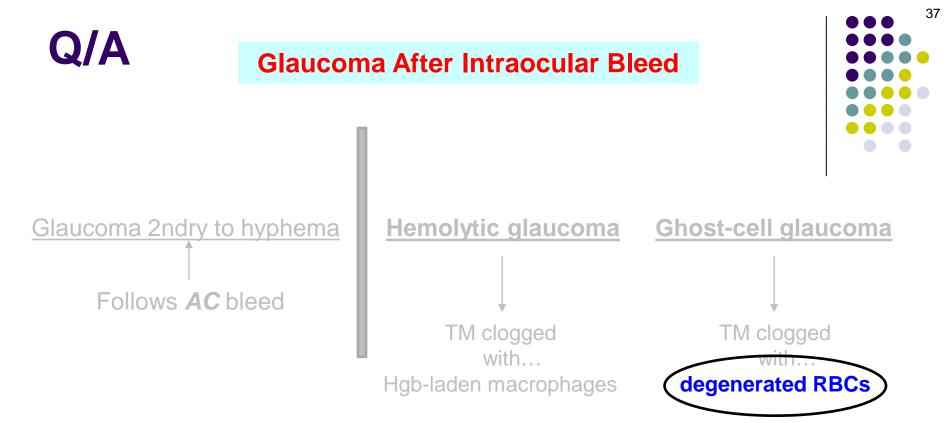
Degenerated RBCs pose a special problem for the TM--why?



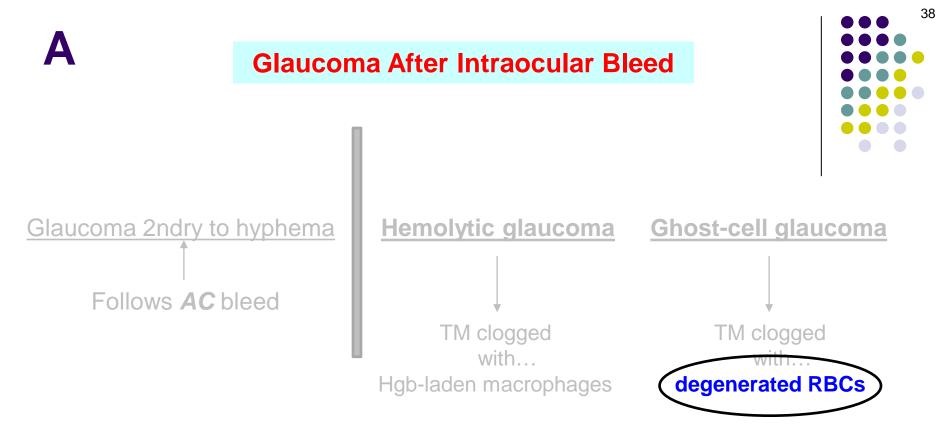
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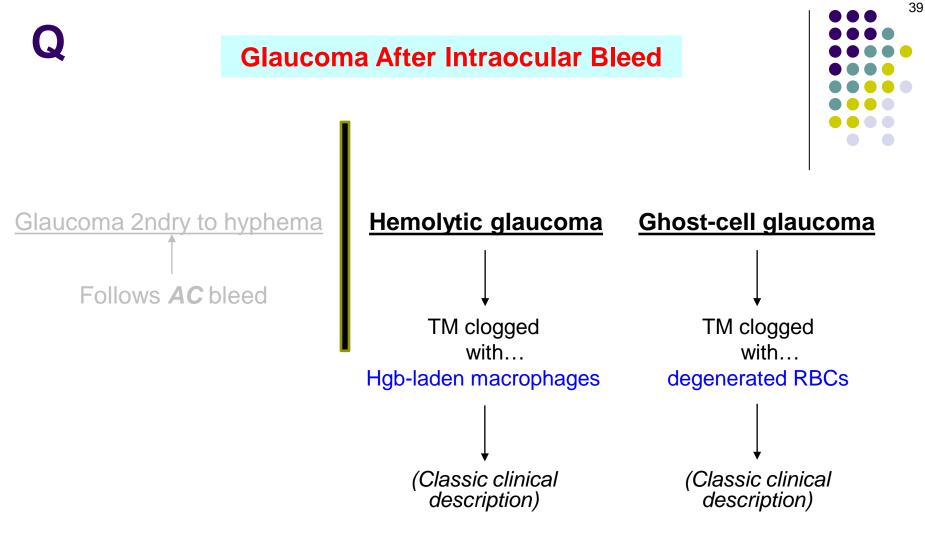
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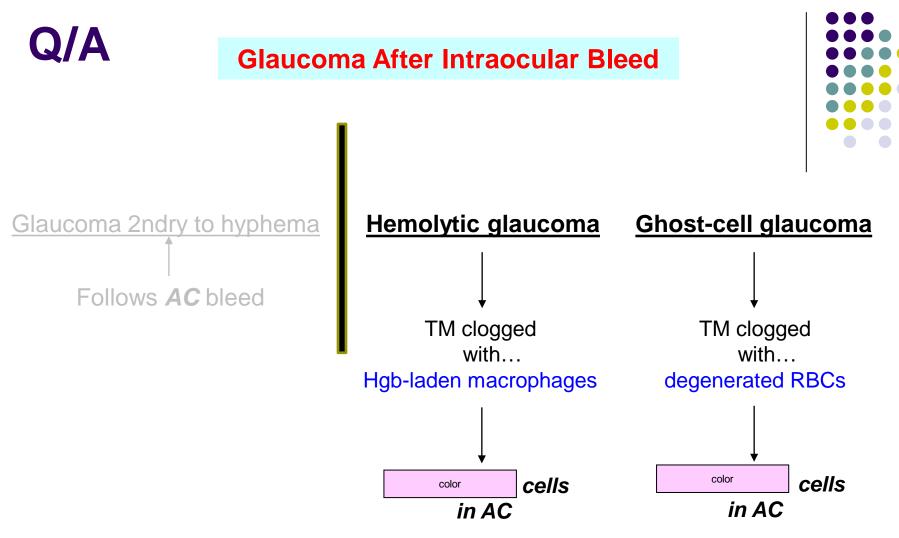
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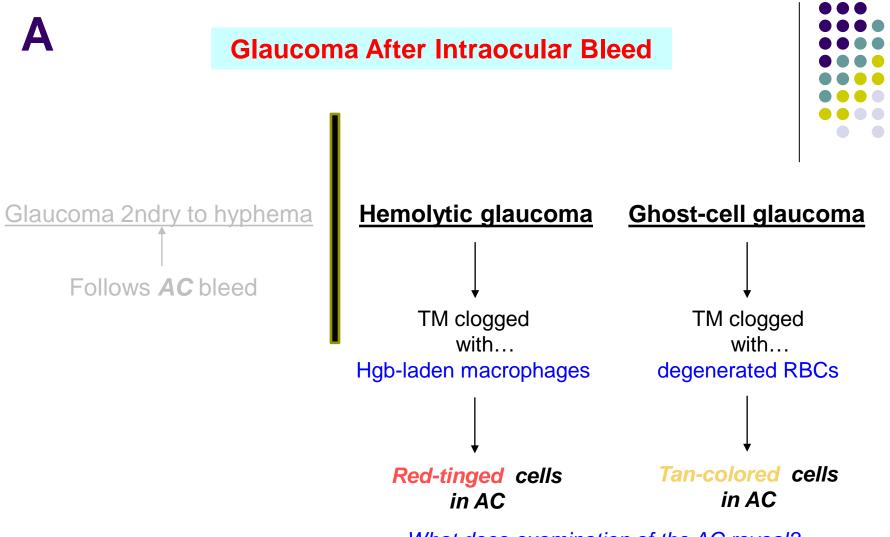


What does examination of the AC reveal?



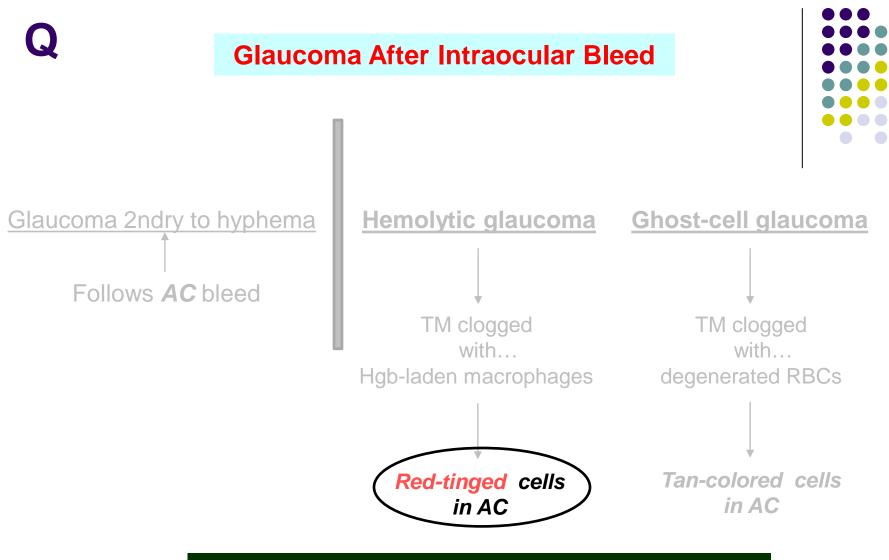
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40



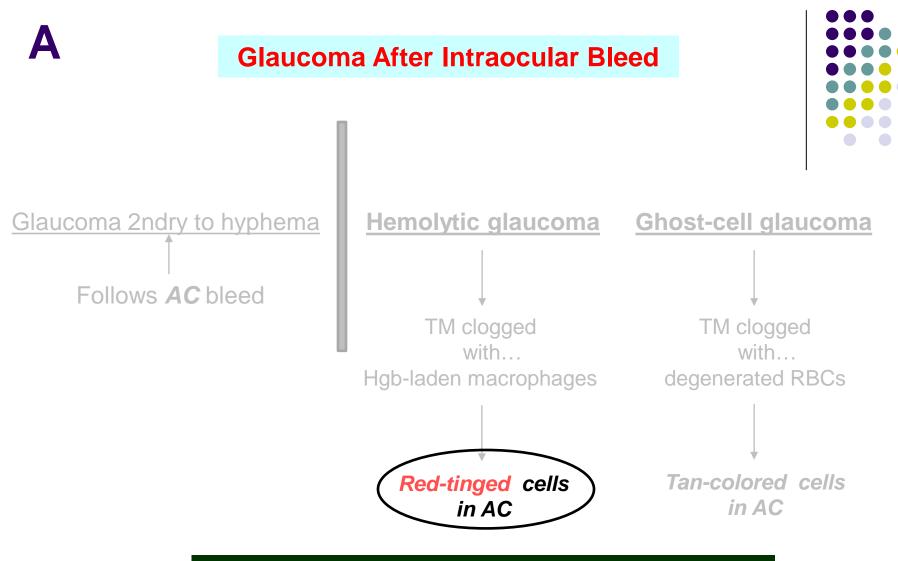
What does examination of the AC reveal?

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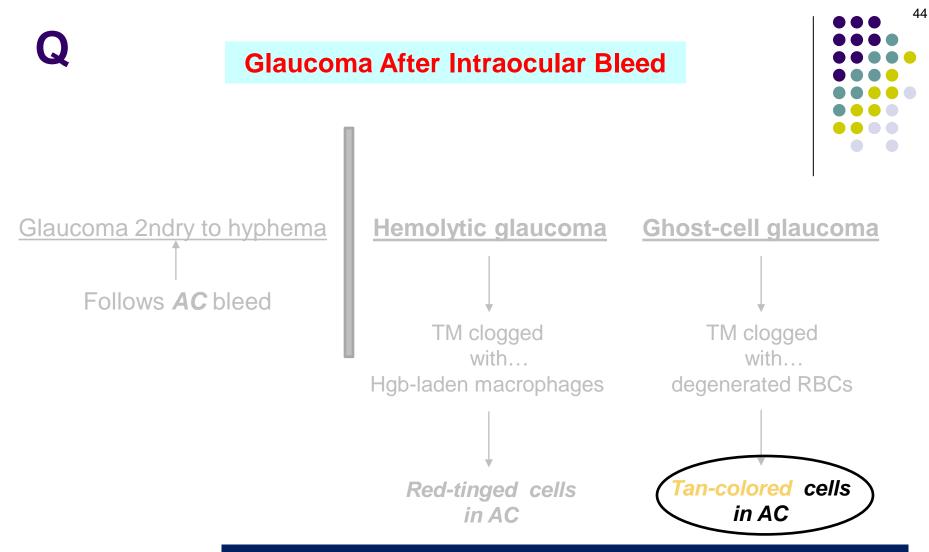
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Would these 'red-tinged cells' be Hgb-laden macrophages?

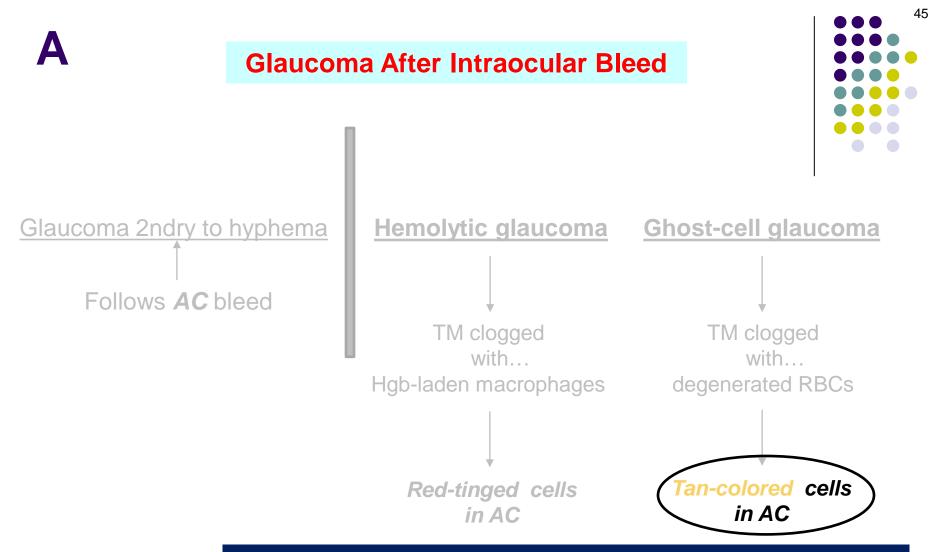


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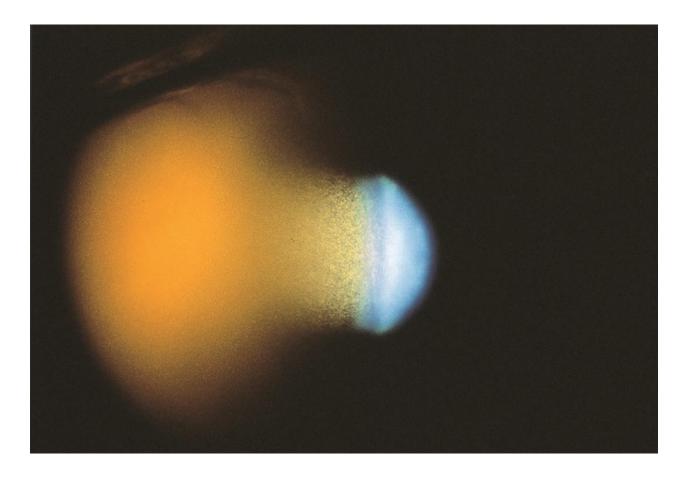
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Would these 'tan-colored cells' be the ghost cells after which the condition was named?

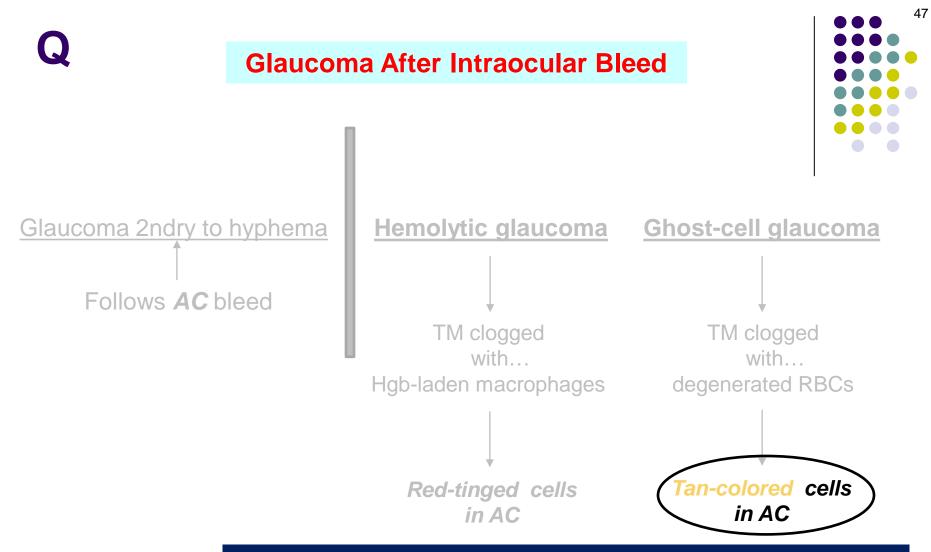


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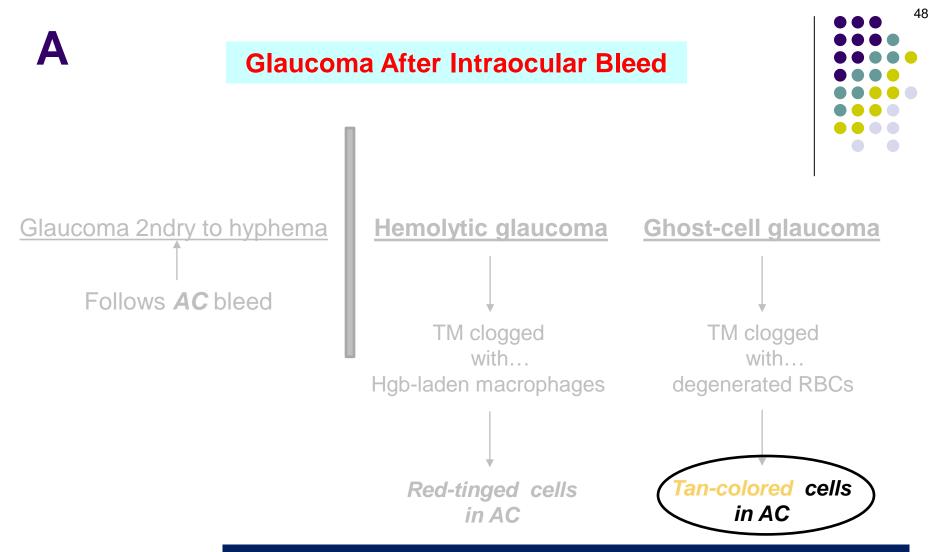
Ghost-cell glaucoma. Copious tan-colored cells in the AC.





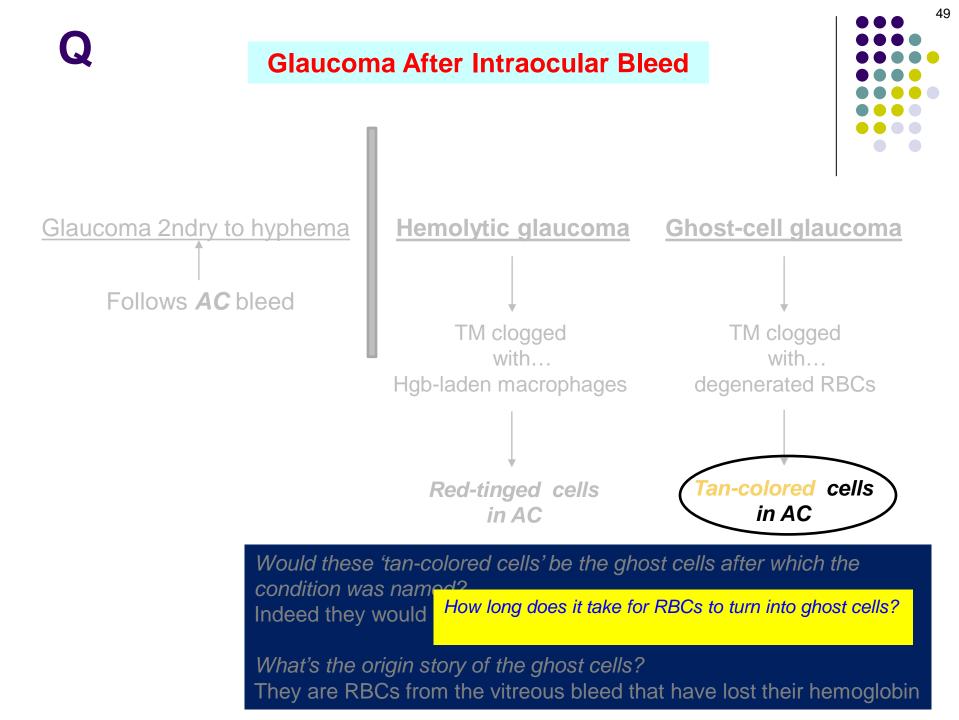
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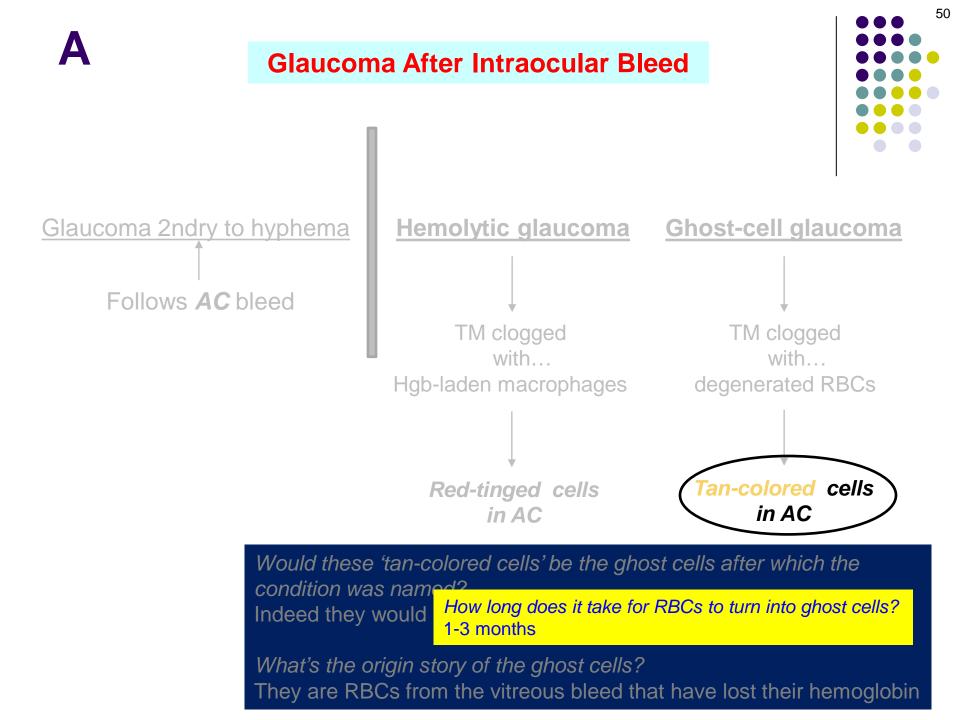
What's the origin story of the ghost cells?

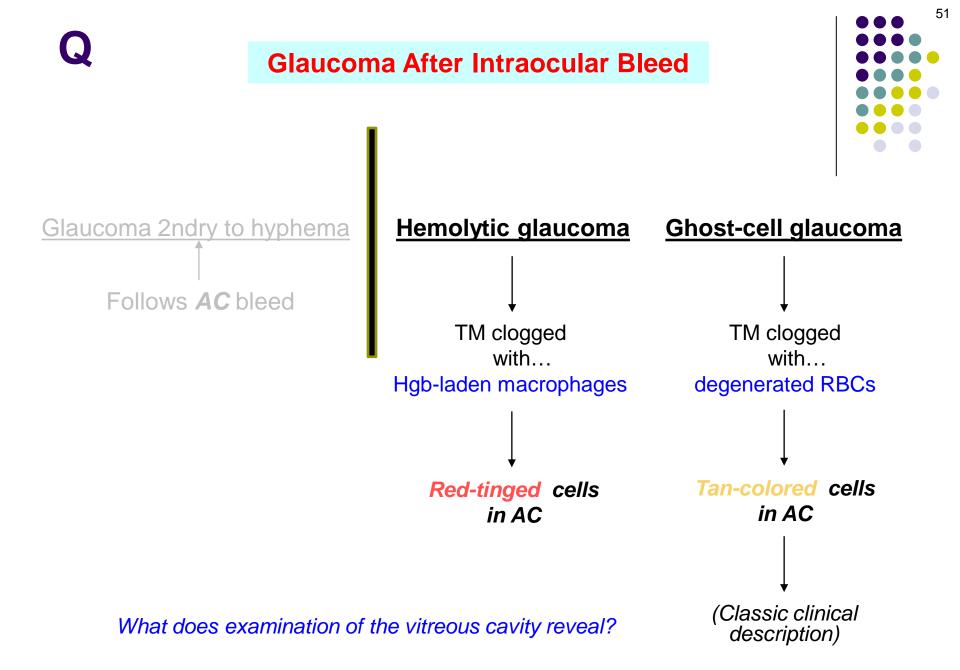


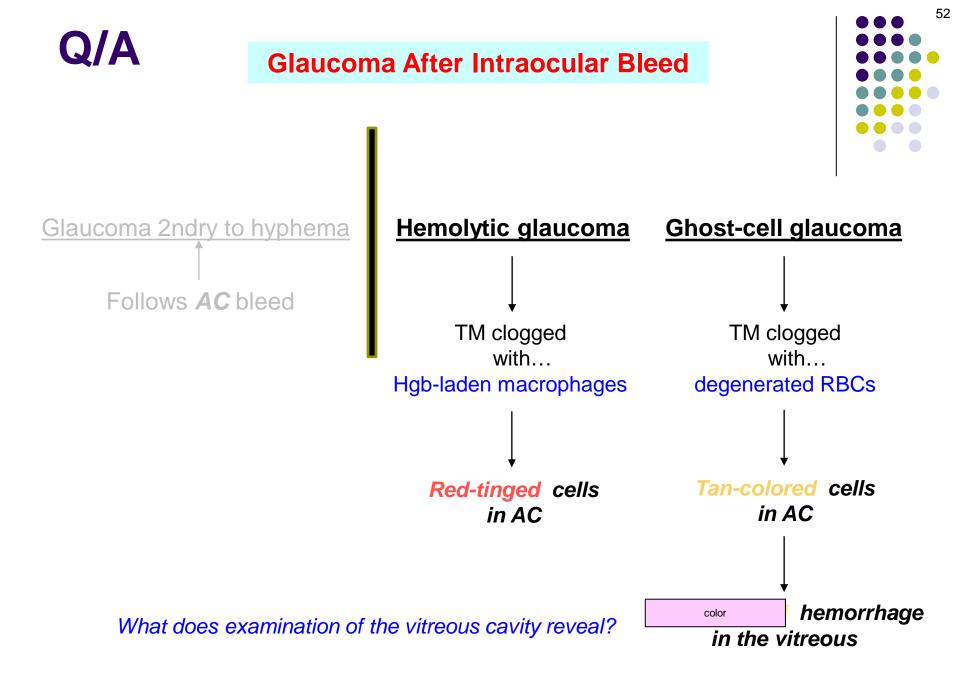
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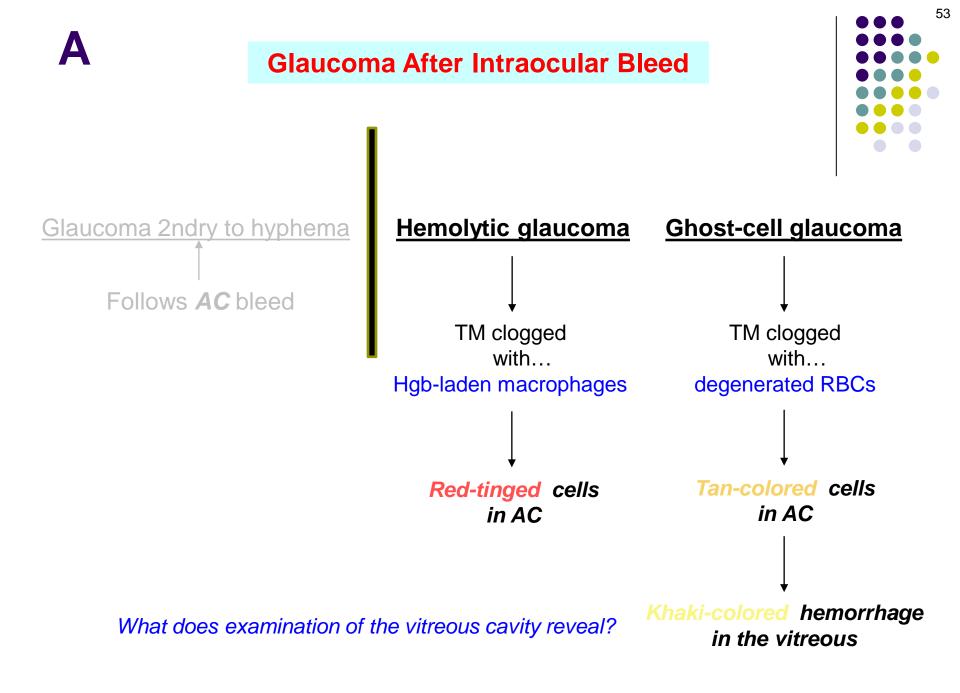
What's the origin story of the ghost cells? They are RBCs from the vitreous bleed that have lost their hemoglobin

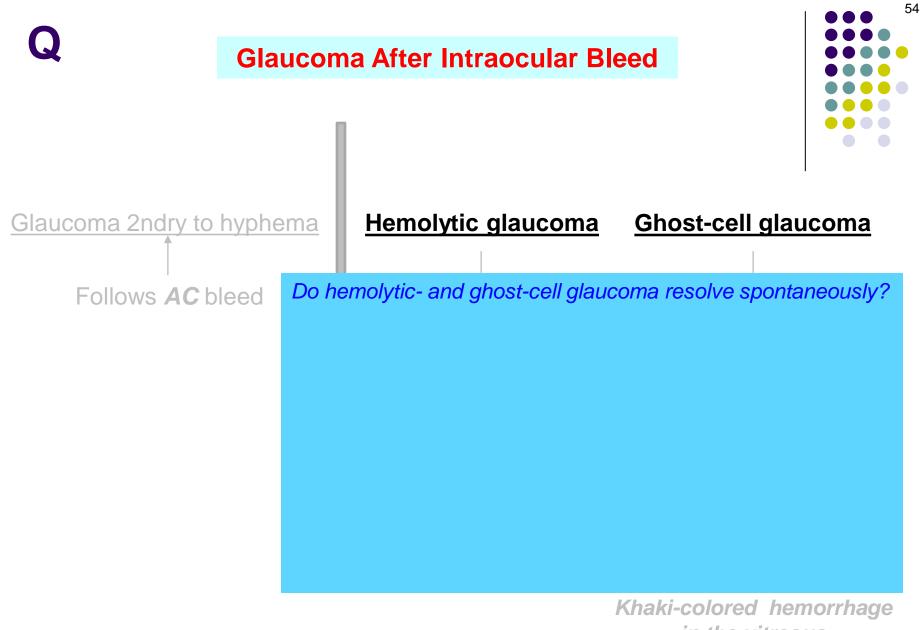




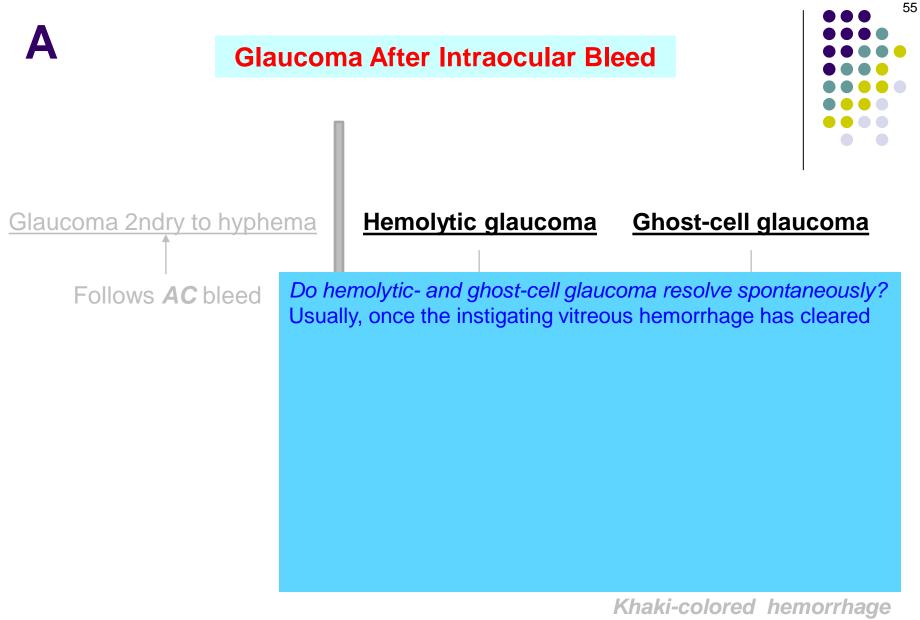




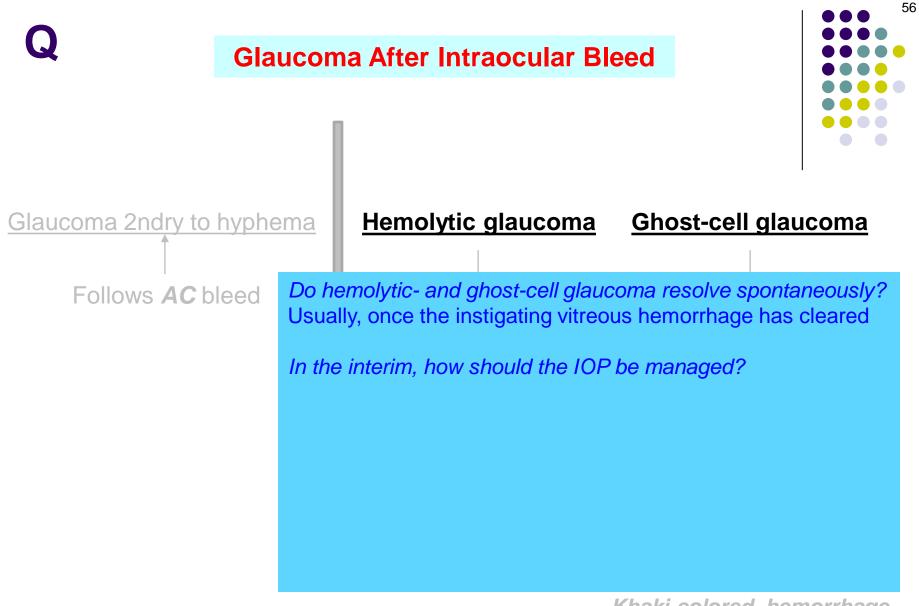




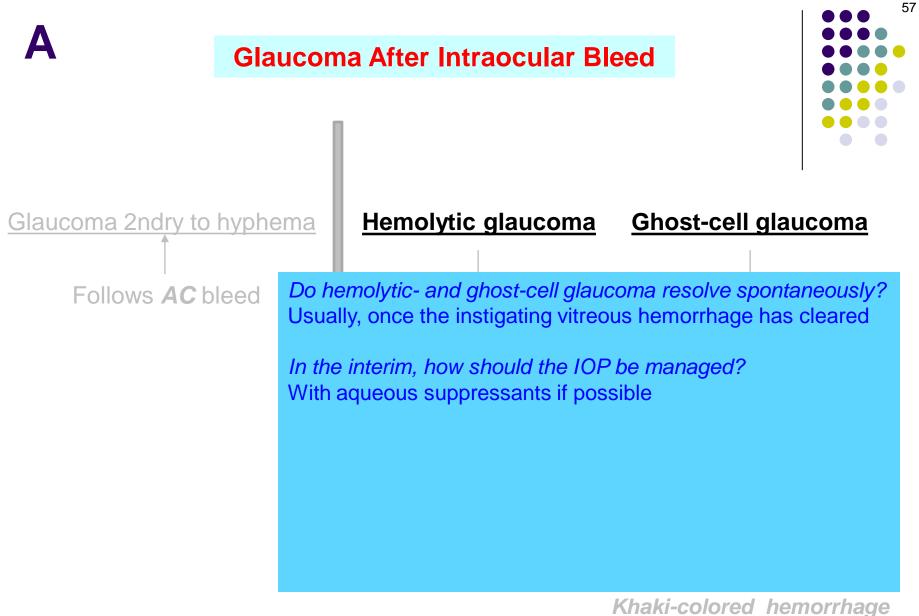
in the vitreous



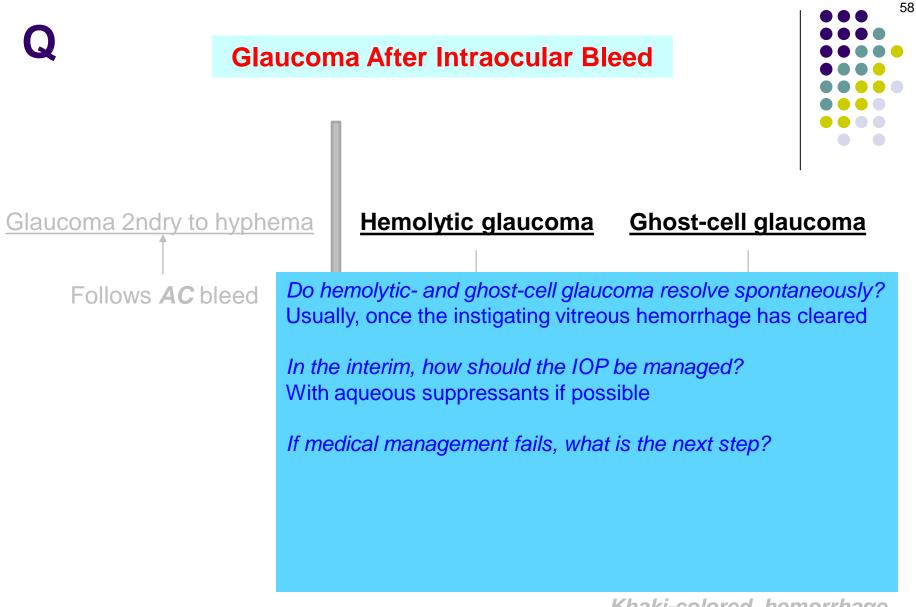
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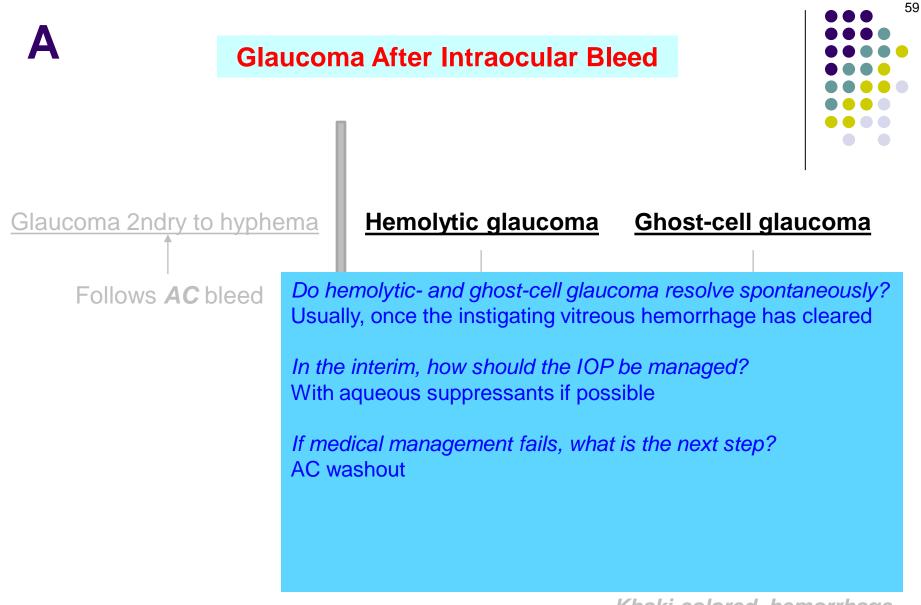
Khaki-colored hemorrhage in the vitreous



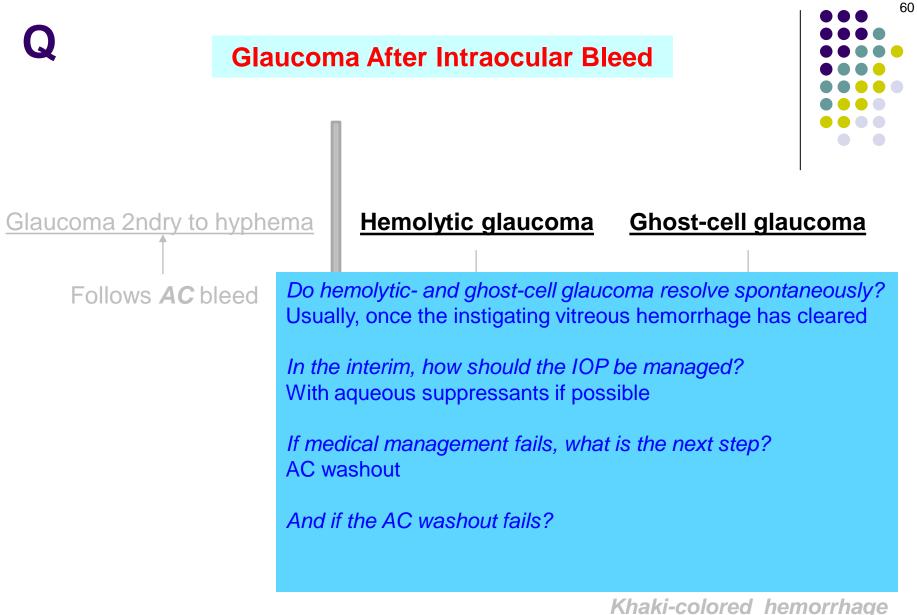
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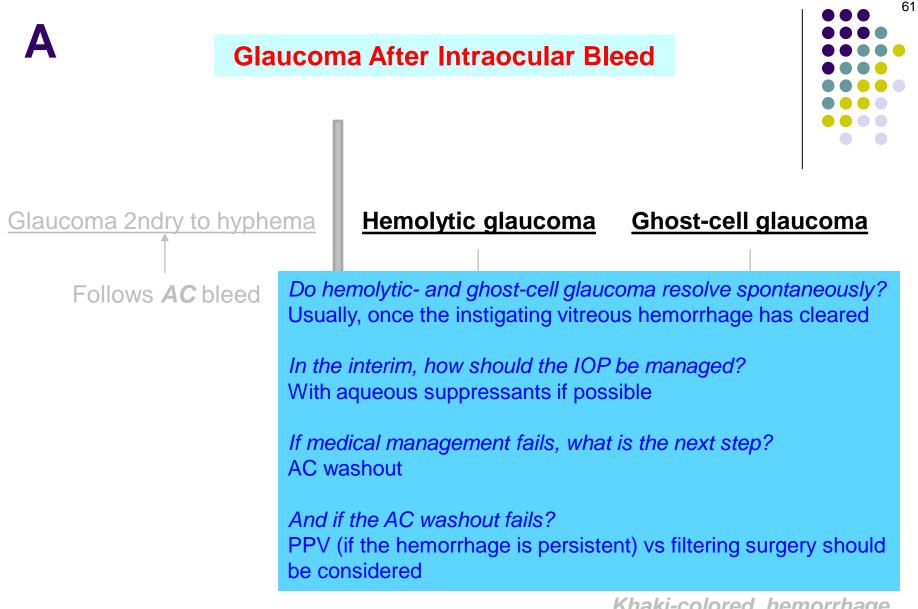
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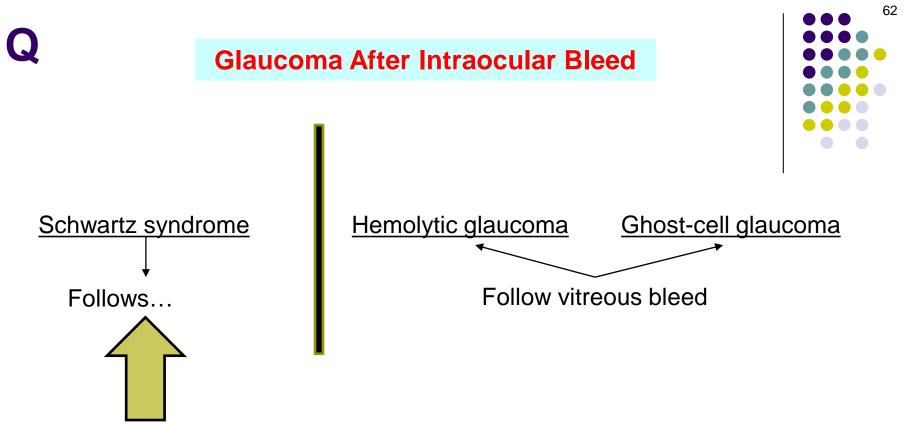
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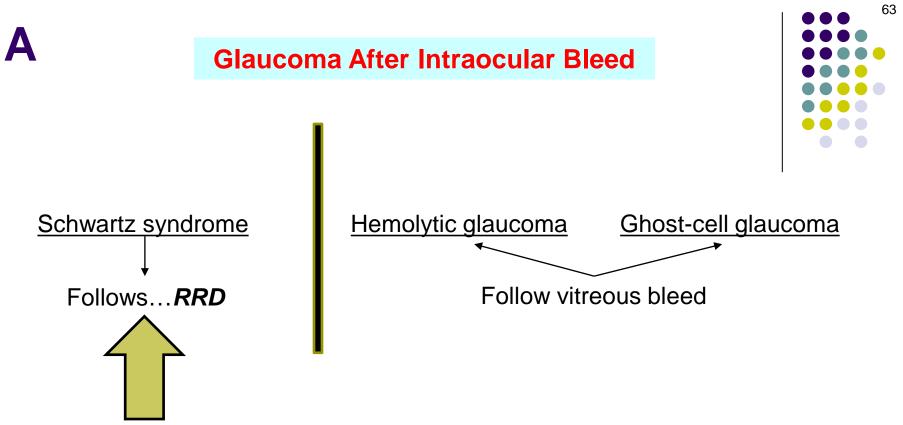


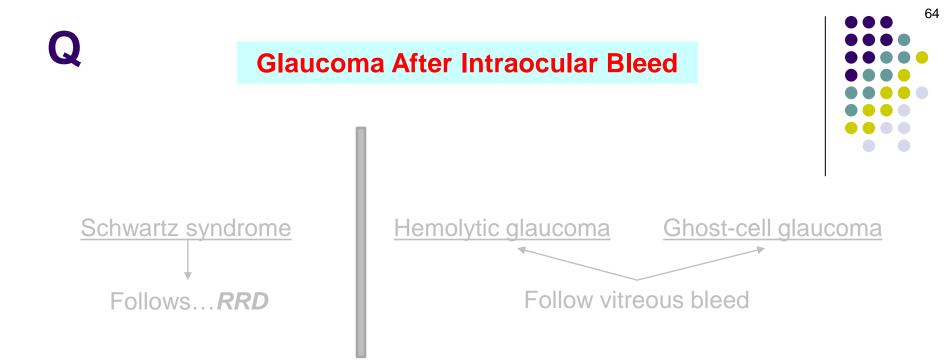
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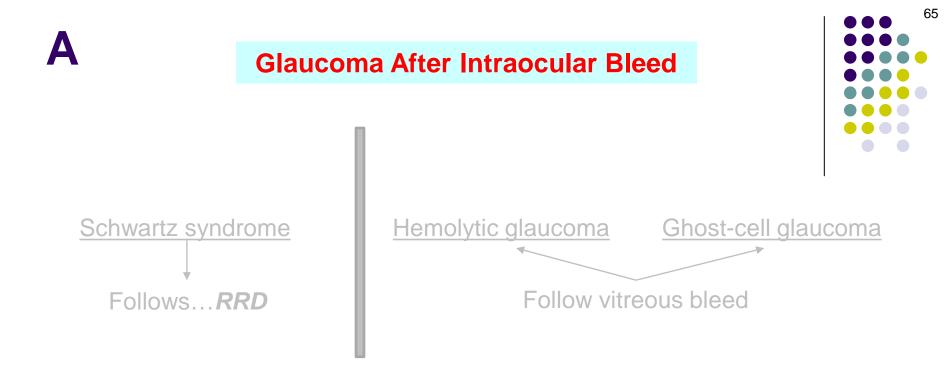
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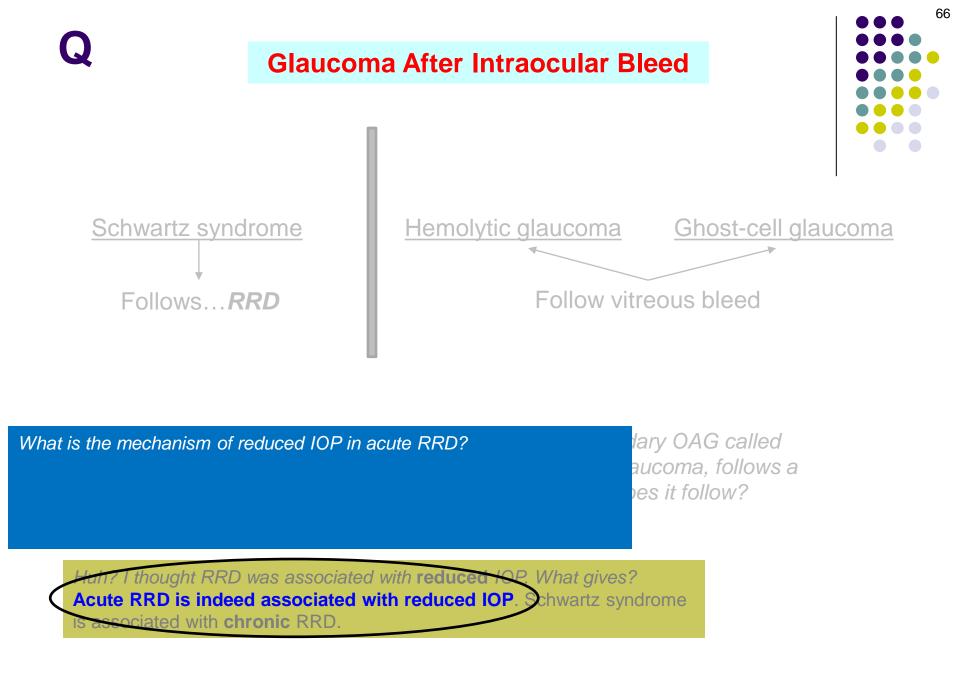


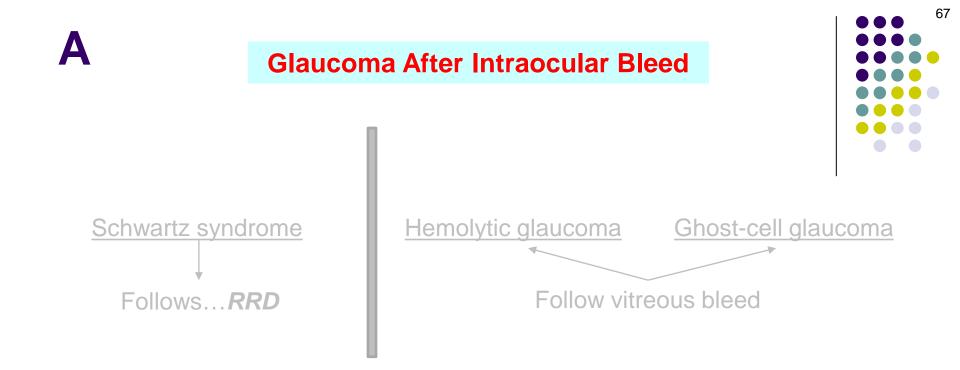


Huh? I thought RRD was associated with reduced IOP. What gives?



*Huh? I thought RRD was associated with* **reduced** *IOP. What gives?* **Acute** RRD is indeed associated with reduced IOP. Schwartz syndrome is associated with **chronic** RRD.

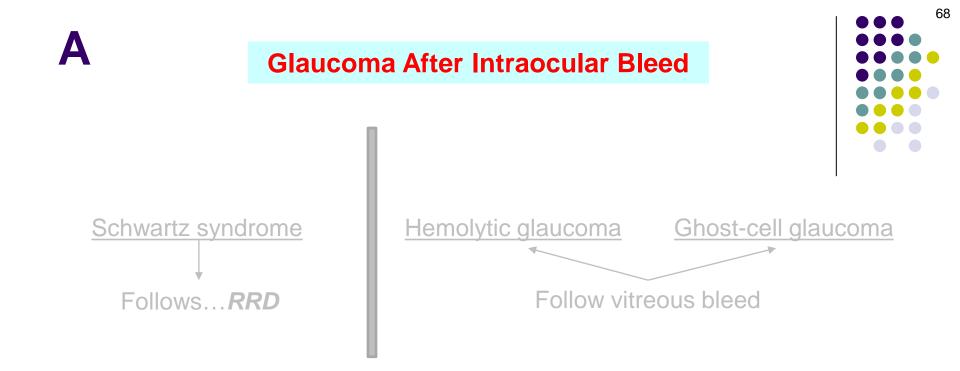




What is the mechanism of reduced IOP in acute RRD? Recall that one function of the RPE is to deturgesce the subretinal space by actively pumping fluid out of it. RRD allows intraocular fluid to pass into the subretinal space, where the RPE attempts to remove it.

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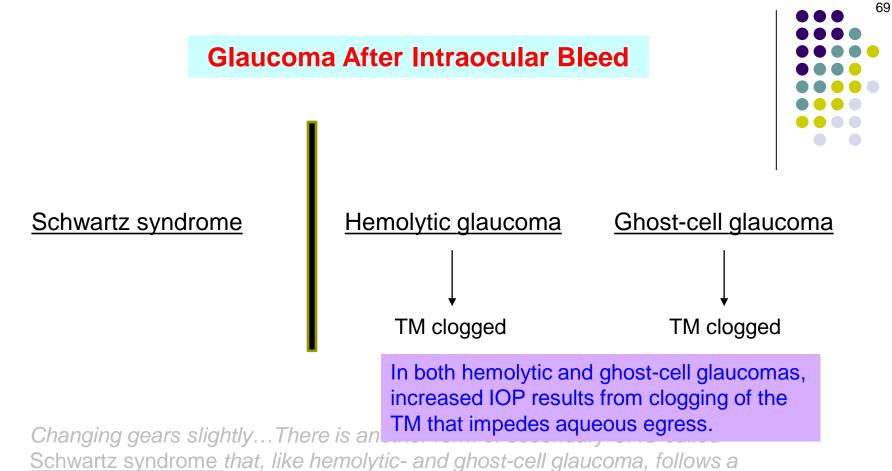
Acute RRD is indeed associated with reduced HOP. What gives? Acute RRD is indeed associated with reduced IOP. Schwartz syndrome is associated with chronic RRD.



What is the mechanism of reduced IOP in acute RRD? Recall that one function of the RPE is to deturgesce the subretinal space by actively pumping fluid out of it. RRD allows intraocular fluid to pass into the subretinal space, where the RPE attempts to remove it. If a significant enough portion of this fluid is removed, IOP will go down.

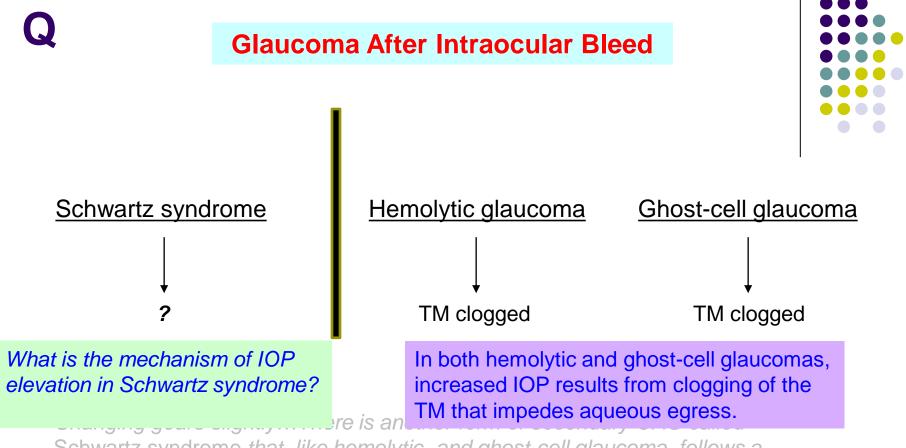
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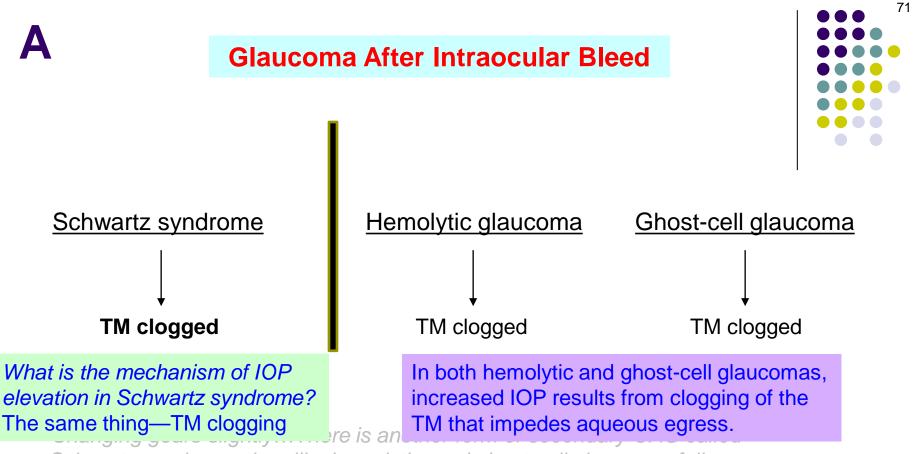
posterior-segment event—but **not** a bleed. What event does it follow?

Rhegmatogenous retinal detachment (RRD)

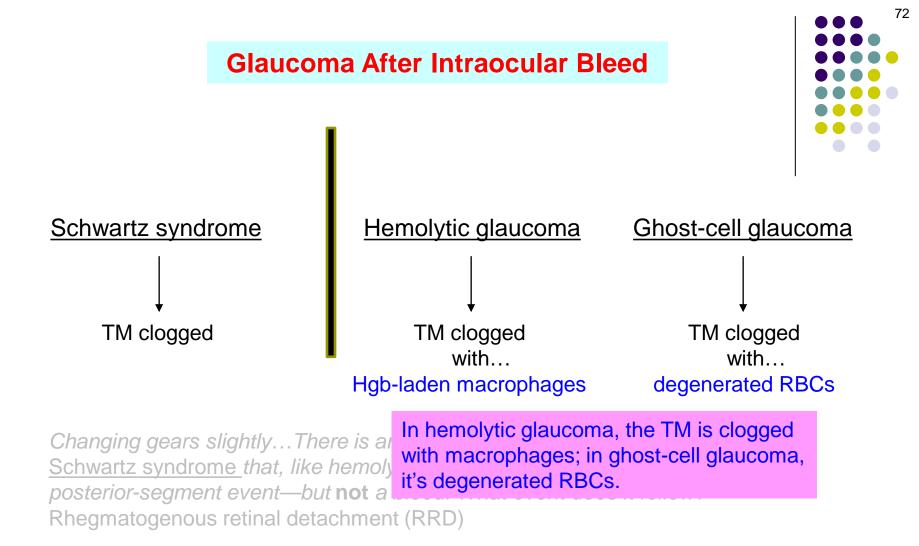


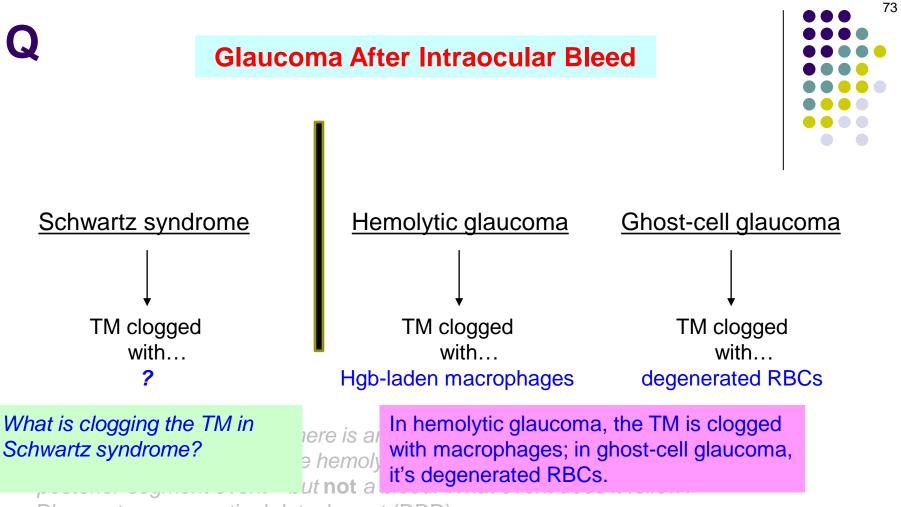
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<u>Schwartz syndrome</u> that, like hemolytic- and ghost-cell glaucoma, follows a posterior-segment event—but **not** a bleed. What event does it follow? Rhegmatogenous retinal detachment (RRD)

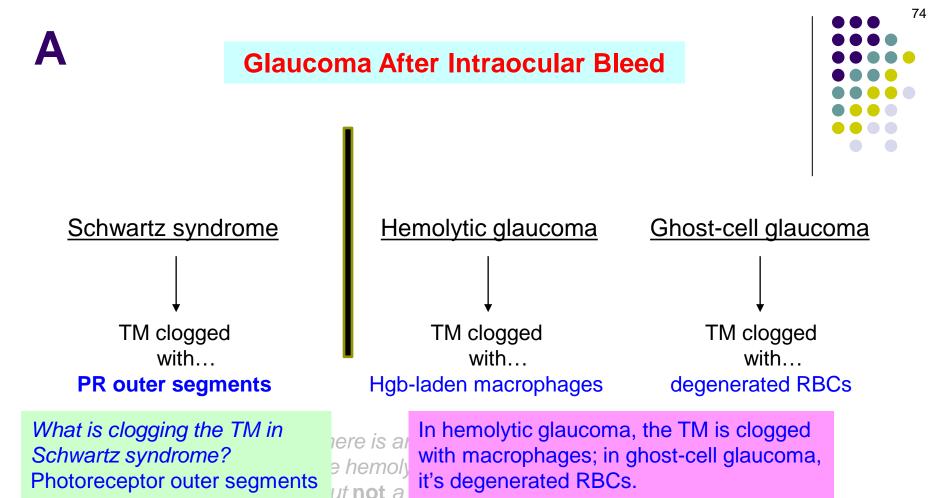


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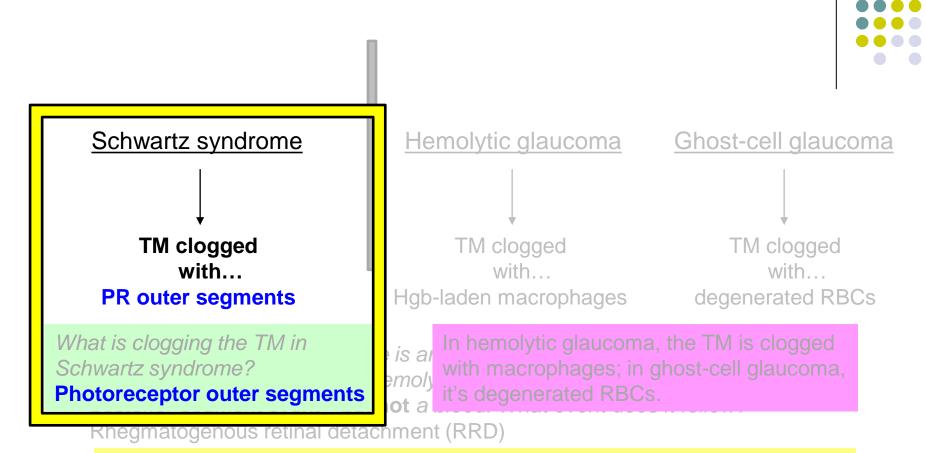




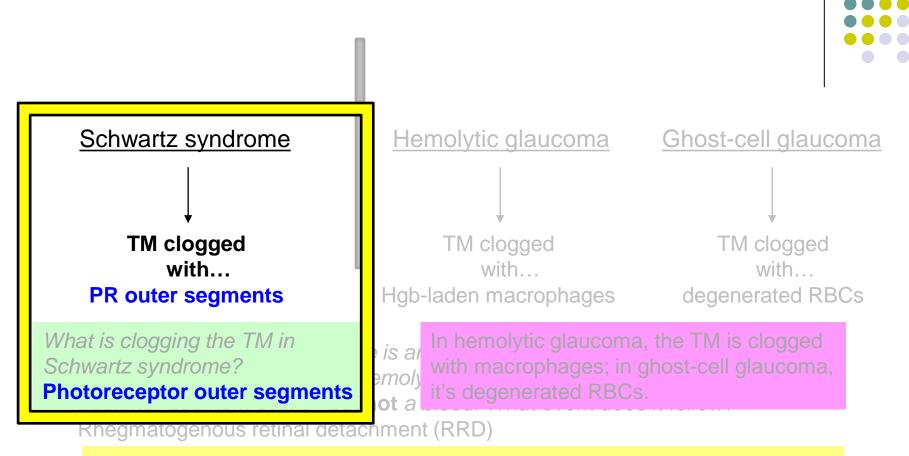
Rhegmatogenous retinal detachment (RRD)



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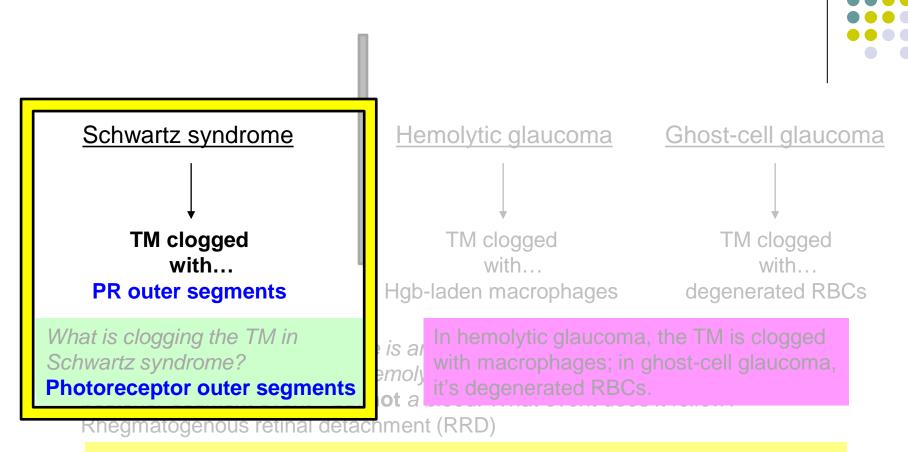
To sum it up: In Schwartz syndrome, chronic RRD allows enough time for liberated PR outer segs to migrate into the AC, where their accumulation in the angle ends up clogging the TM and elevating IOP.



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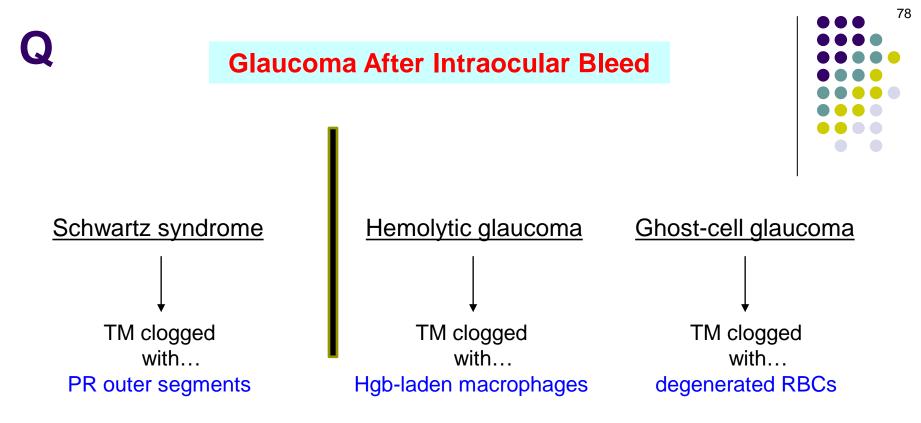
All these PR outer segs floating around the AC—can they be mistaken for inflammatory cells?



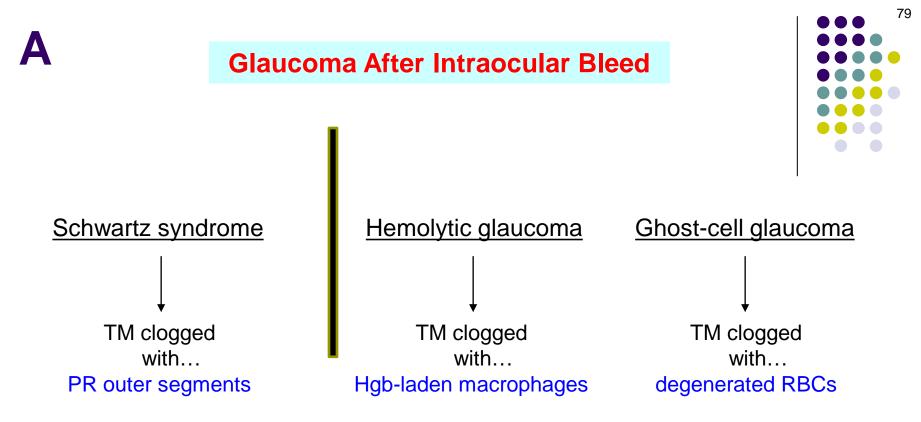


To sum it up: In Schwartz syndrome, chronic RRD allows enough time for liberated **PR outer segs to migrate into the AC**, where their accumulation in the angle ends up clogging the TM and elevating IOP.

> All these PR outer segs floating around the AC—can they be mistaken for inflammatory cells? Yes, uveitic glaucoma is a common misdiagnosis in Schwartz syndrome



What's the best way to manage Schwartz syndrome?



What's the best way to manage Schwartz syndrome? Repair the RRD