

# The Pathology of Glaucoma

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AMERICAN ASSOCIATION  
OF NEUROPATHOLOGISTS

# Disclosures

- **I have no relevant financial relationships to disclose**



# Learning objectives

- **Glaucoma: definition, history, pathophysiology, classification**
- **Identify the normal anatomy and histology of the anterior chamber angle, retina, and optic nerve**
- **Identify the histologic changes involving the anterior chamber angle, retina, and optic nerve associated with glaucoma**



## Why glaucoma teaching rounds?

- **The pathology of glaucoma is perhaps not a common lecture/teaching topic during residency/fellowship**
- **The retina and optic nerve are neuroglial tissues and of interest to us as neuropathologists**





# The Pathology of Glaucoma: Outline

- **Introduction**
  - Definition, History, Pathophysiology
- **Histology and Anatomy**
  - Retina: absent ganglion cells
  - Optic nerve: cupping and atrophy
  - Anterior chamber angle
- **Classification and Pathology**
  - Open angle, closed angle; primary, secondary

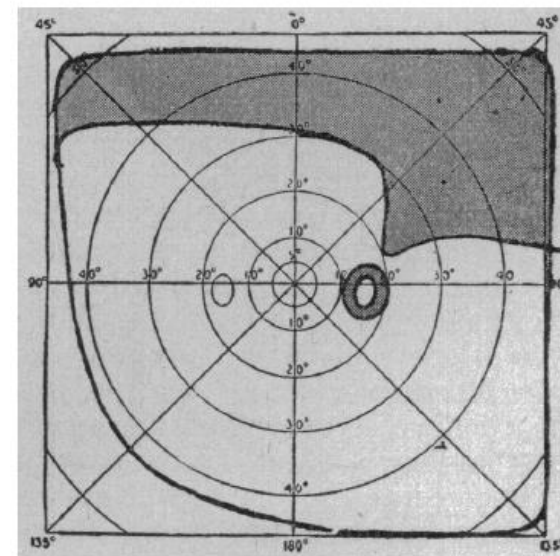




## GLAUCOMA\*

BY WILLIAM D. ROWLAND, M.D.†

**G**LAUCOMA, derived from a Greek word meaning *a sea-green*, is defined by Gould as “a disease of the eye characterized by heightened intraocular tension, resulting in hardness of the globe, excavation of the papilla or optic disc, a restriction of the field of vision, corneal anesthesia, colored halo about lights, and lessening of visual power that may proceed to blindness. The etiology is obscure.”



No. 1, Mr. S, right.

N Engl J Med 1931; 204:653-656



# Introduction to glaucoma

- **Glaucoma is the 2<sup>nd</sup> commonest cause of blindness worldwide**
- **Cataracts are the leading cause of blindness worldwide**
- **Polls: cancer and blindness are our 2 greatest medical fears**



# Introduction to glaucoma

- **Unlike cataracts, vision loss in glaucoma is largely irreversible**
- **Glaucoma affects 80 million individuals**
- **Expected to rise to >100 million by 2040**
- **> 11 million are blind from glaucoma**



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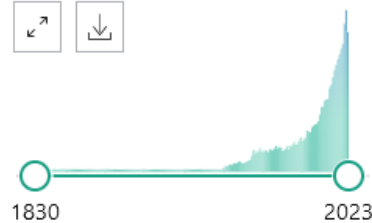
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- Associated data

ARTICLE TYPE

- Books and Documents
- Clinical Trial
- Meta-Analysis

**Glaucoma.**

1 Kang JM, Tanna AP.

Cite Med Clin North Am. 2021 May;105(3):493-510. doi: 10.1016/j.mcna.2021.01.004. Epub 2021 Apr 2. PMID: 33926643 [Review](#).

Share **Glaucoma** is the leading cause of irreversible blindness worldwide. The global prevalence of **glaucoma** in people aged 40 to 80 years is estimated to be 3.5%. With the growing number and proportion of older persons in the population, it is projected that 111.8 million ...

**The pathophysiology and treatment of glaucoma: a review.**

2 Weinreb RN, Aung T, Medeiros FA.

Cite JAMA. 2014 May 14;311(18):1901-11. doi: 10.1001/jama.2014.3192. PMID: 24825645 [Free PMC article](#). [Review](#).

Share **IMPORTANCE: Glaucoma** is a worldwide leading cause of irreversible vision loss. ...**OBJECTIVE:** To describe current evidence regarding the pathophysiology and treatment of open-angle **glaucoma** and angle-closure **glaucoma**. **EVIDENCE REVIEW:** A literature search was c ...

**Targets of Neuroprotection in Glaucoma.**

3 He S, Stankowska DL, Ellis DZ, Krishnamoorthy RR, Yorio T.

Cite J Ocul Pharmacol Ther. 2018 Jan/Feb;34(1-2):85-106. doi: 10.1089/jop.2017.0041. Epub 2017 Aug 18. PMID: 28820649 [Free PMC article](#). [Review](#).

Share Progressive neurodegeneration of the optic nerve and the loss of retinal ganglion cells is a hallmark of **glaucoma**, the leading cause of irreversible blindness worldwide, with primary open-angle **glaucoma** (POAG) being the most frequent form of **glaucoma** in the W ...



# Introduction to glaucoma

- **First known record of glaucoma mentioned in Hippocrates' *Aphorisms***
  - Published in 400 BC
  - *Ars longa vita brevis*
  - Referred to as “glaucois”
  - Dimness of vision, eventual blindness

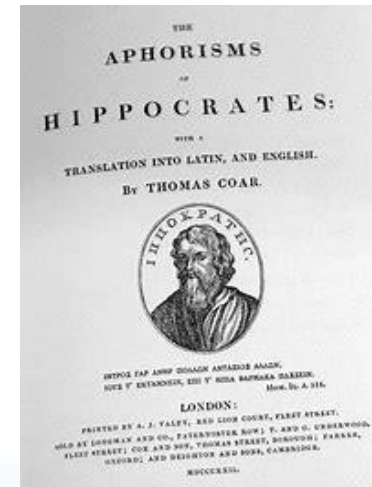


Table 1. Early Historical Events in the Evolution of Glaucoma Diagnosis

Date	Event
400s BC	Hippocrates' <i>Aphorisms</i> includes the first written record of glaucoma.
300s BC	Aristotle proposes that the color of an eye is dependent the amount of water within it.
100s AD	Galen defines glaucoma as a discoloration of the pupils due to fluid shifts in the eye; he draws the optic nerve as a hollow structure.
500s	Aetius describes 2 categories of glaucoma: a defect of the lens and a defect of the pupil.
1510s	da Vinci notes the cornea and aqueous bend light and the temporal VF extends 90° from fixation.
1626	Banister suggests the relevance of eye pressure to glaucoma.
1668	Mariotte describes the blind spot.
1673	van Leeuwenhoek draws first illustration of a peripheral nerve.
1752	Daviel presents results of more than 200 cataract extractions.
1755	Zinn contests the notion that the optic nerve is a hollow structure.
1807	Young specifies the dimensions of the VF.
1826	St Yves describes clinical features of advanced glaucoma in <i>Nouveau traité des Maladies des Yeux</i> .
1830	Mackenzie delineates 6 stages of glaucoma in <i>Practical Treatise on the Diseases of the Eye</i> .
1851	von Helmholtz invents the ophthalmoscope.
1851	Mueller describes depression of the optic disc in glaucoma.
1854	Jaeger illustrates swelling of the optic disc in glaucoma.
1855	Weber describes the glaucomatous optic disc as "cupped."
1856	von Graefe proposes incorporation of VF assessment into clinical practice.
1860	Schwalbe discovers anatomic connection between anterior chamber and ciliary veins.
1867	Weber invents the first applanation tonometer.
1870	Leber describes the aqueous pathway.
1879–1890	Priestley Smith suggests that glaucoma damage is due to vascular and metabolic changes in addition to mechanical forces; he later proposes that narrowing of the angle predisposes to glaucoma.
1907	Trantas promotes gonioscopy as routine diagnostic technique.
1920	Seidel refines Leber's filtration theory to account for colloid osmotic pressure of plasma proteins.
1945	Hans Goldmann invents the hemispheric bowl perimeter.
1950	Hans Goldmann introduces the Goldmann tonometer.

VF = visual field.





## THE DIAGNOSIS OF GLAUCOMA

BY DAVID W. WELLS, M.D., BOSTON

THERE is no brighter page in the history of ophthalmology than the successful treatment of glaucoma, if diagnosed in its incipiency; but there is nothing more pathetic than the discovery of the neglected case.

This neglect happens so frequently notwithstanding the many warnings sounded by ophthalmologists, that it is evident that the general medical profession is not yet sufficiently impressed with the importance of the subject.





# Glaucoma pathophysiology

- **Intraocular pressure plays a major role in the development of glaucomatous optic neuropathy and is considered the most significant risk factor.**
- **Disruption in the production or outflow of aqueous humor fluid may lead to increased intraocular pressure**



## How does elevated IOP damage retinal ganglion cells?

- **Ischemia and/or blockage of axoplasmic flow** caused by mechanical compression of axons in the pores of the **lamina cribrosa**, which are distorted by high levels of IOP.
- Blockage of axoplasmic flow may **deprive cells of brain-derived neurotrophic factor** whose absence triggers programmed cell death.
- **Glial cell activation and neuroinflammatory processes** are thought to be important mediators of retinal ganglion cell damage.



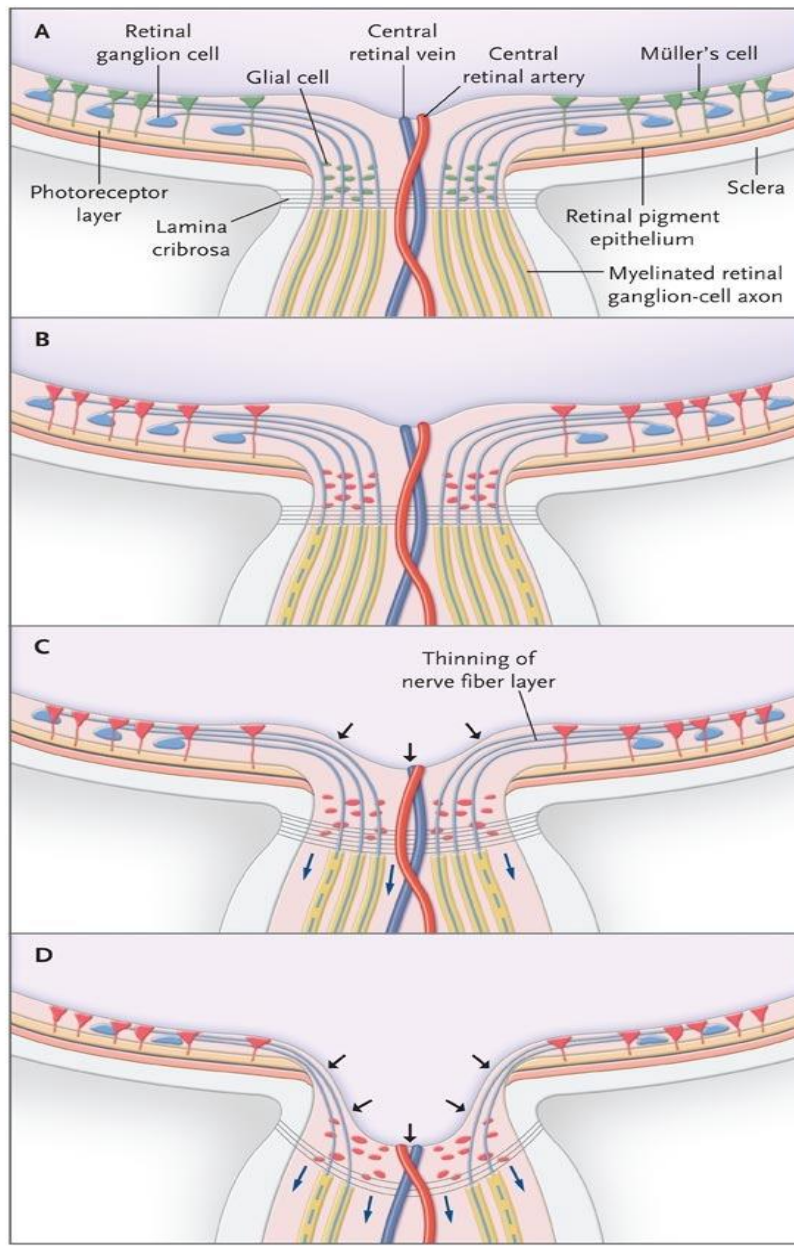
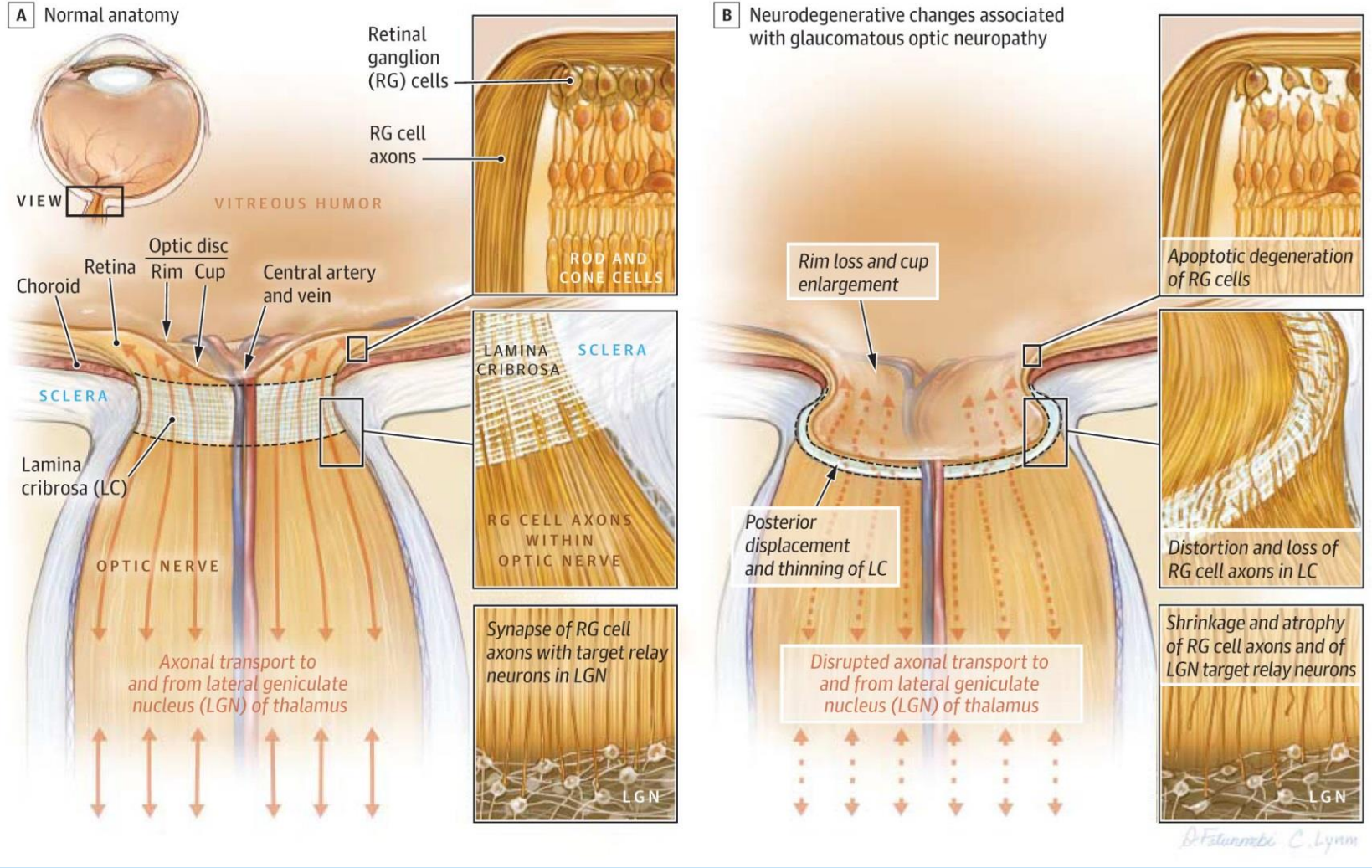


Figure 4. The Optic-Nerve Head and Proposed Events Leading to Retinal Ganglion-Cell Death in Glaucoma. In the normal optic-nerve head and retina (Panel A), retinal ganglion-cell axons exit the eye through the lamina cribrosa, becoming myelinated only in the postlaminal region. Glia in the retina (e.g., Müller's cells) and optic-nerve head (e.g., astrocytes and microglia) are quiescent (green). **Increasingly elevated intraocular pressure puts stress on retinal ganglion cells, and glial cells become reactive** (Panel B, red). Elevated intraocular pressure also leads to the production of a variety of substances, including **tumor necrosis factor  $\alpha$** , which in turn **damage retinal ganglion-cell axons** (dashed lines) at the lamina cribrosa. At this point there is no clinically detectable change in the cupping of the optic-nerve head. **Damage to retinal ganglion-cell axons is followed by cell (soma) death through apoptosis** (Panel C). **Loss of retinal ganglion cells and axon fibers results in thinning of the nerve-fiber layer.** The lamina cribrosa itself undergoes remodeling, becoming thicker while bowing posteriorly (blue arrows), with increased cupping of the optic-nerve head (black arrows). In the advanced stage of glaucoma (Panel D), **apoptosis and neuroinflammatory processes result in cell death and loss of most retinal ganglion cells and axons.** The prelaminar tissue is substantially attenuated, and the **lamina cribrosa becomes thinner and bowed more posteriorly** (blue arrows), resulting in pronounced cupping of the optic-nerve head (black arrows).

Kwon et al, N Engl J Med 2009; 360:1113-1124



Weinreb RN, Aung T, Medeiros FA. The pathophysiology and treatment of glaucoma: a review. JAMA 2014;311:1901-11.



# Glaucoma facts

- **There is no cure for glaucoma**
- **Lost vision cannot be regained**
- **Glaucoma is a chronic condition and must be monitored for life**
- **There may be no warning symptoms:**
  - **‘silent blinder’ similar to hypertension - ‘silent killer’**
- **Everyone is at risk, from birth to elderly**
- **Knowledge, information, prevention, treatment**







## 4 Things You can Learn About Glaucoma from Bono



Leer en Español: [4 cosas que podemos aprender de Bono acerca del glaucoma](#)

Written By: [Linda Apeles](#)

Reviewed By: [J. Kevin McKinney, M.D.](#)

Jan. 09, 2015

One of the biggest glaucoma-related news stories of 2014 was [Bono's revelation that he has the condition](#). While his comments about it have been brief, there are important tips the public can learn about glaucoma following the rockstar's announcement.

### 1. Having glaucoma doesn't mean you have to go blind.

When Bono announced he had glaucoma, he revealed that he has had the condition for many years. He serves as a great example of how many people with glaucoma can keep their sight and still lead very active lives if treated early enough. In fact, the probability of [blindness due to glaucoma has decreased by nearly half since 1980](#). Researchers believe that advances in diagnosis and therapy are likely causes for the decrease.

### 2. Glaucoma treatments work!

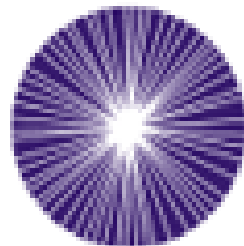
It's no wonder the public never suspected that Bono had an eye disease – that's how effective [glaucoma treatments, such as medicated eye drops and minimally invasive surgery](#), can be. "I have good treatments and I am going to be fine," Bono said at the time of the announcement.

### 3. The earlier you get diagnosed, the better.

The key to preventing vision loss from glaucoma is early diagnosis. While the details of his treatment have not been shared with the public, 54 year-old Bono said he has had the disease for over 20 years, so it's likely he was diagnosed at an early stage. Blindness from glaucoma can often be prevented with early treatment.

### 4. Glaucoma may have no obvious symptoms in its early stages.

As you get older, it is especially important to have regular medical eye exams. The only sure way to diagnose glaucoma is with a complete eye exam. A screening that only checks eye pressure is not enough to find glaucoma. The American Academy of Ophthalmology recommends that all adults (even if they have no signs of symptoms of eye problems) get a baseline [eye examination](#) at least by age 40, the time when early signs of disease or changes in vision may occur. A baseline exam can help identify signs of eye disease at an early stage when many treatments can have the greatest impact on preserving vision. Of course, if you have any problems with your vision or eye comfort before age 40, don't wait; make an appointment with an ophthalmologist right away.



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I do not have glaucoma, but I do protect my eyes



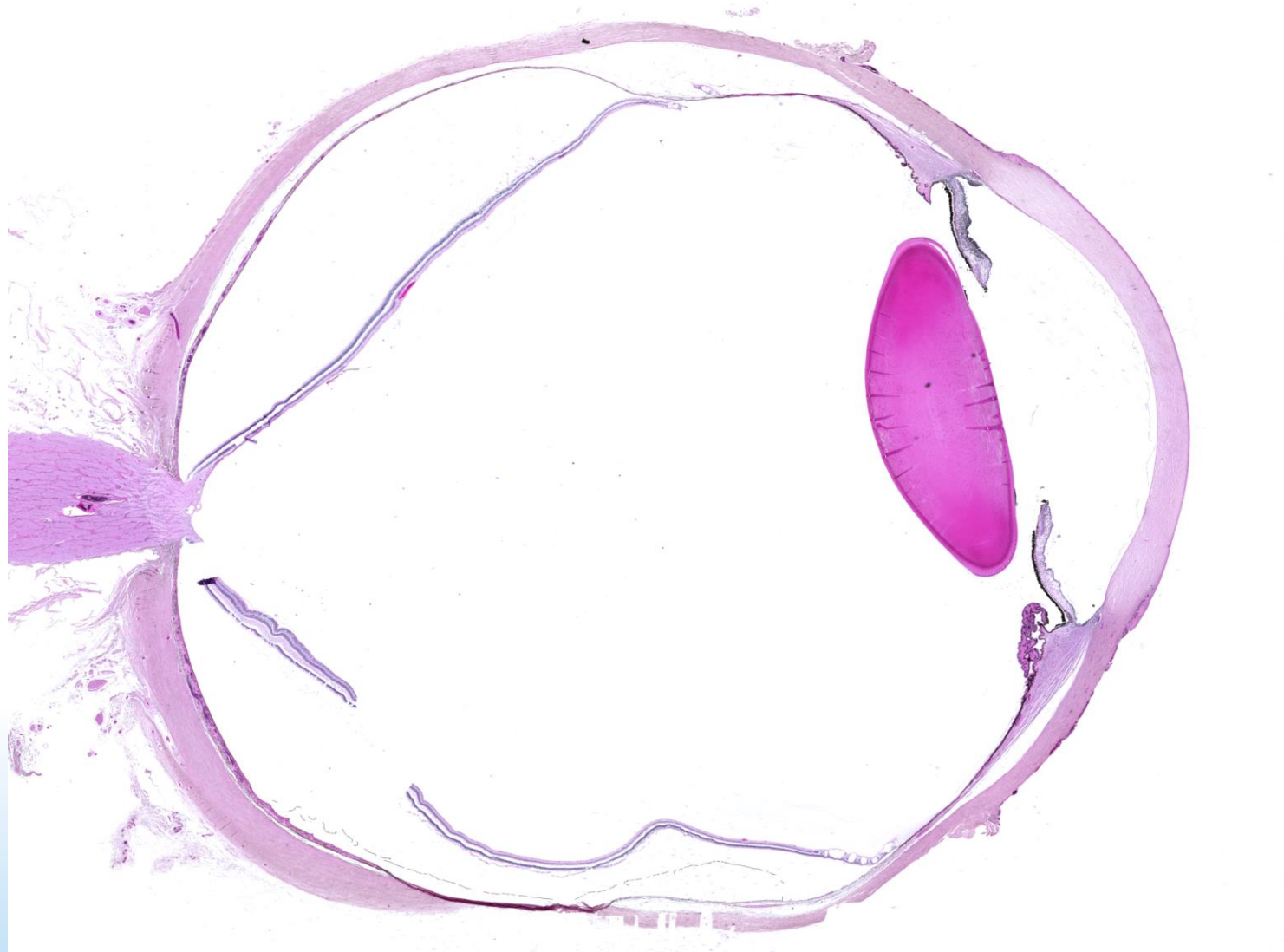
# The Pathology of Glaucoma: Outline

- **Introduction**
  - Definition, History, Pathophysiology
- **Histology and Anatomy**
  - Retina: absent ganglion cells
  - Optic nerve: cupping and atrophy
  - Anterior chamber angle: anatomy, histology, landmarks
- **Classification**
  - Open angle, closed angle; primary, secondary



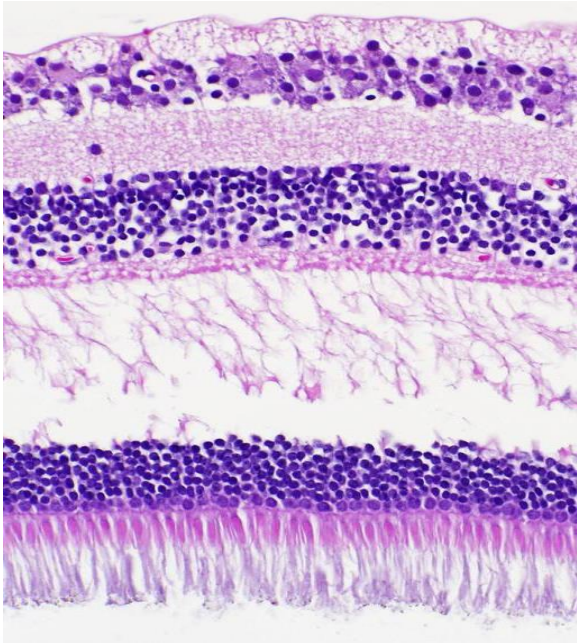


# Normal globe



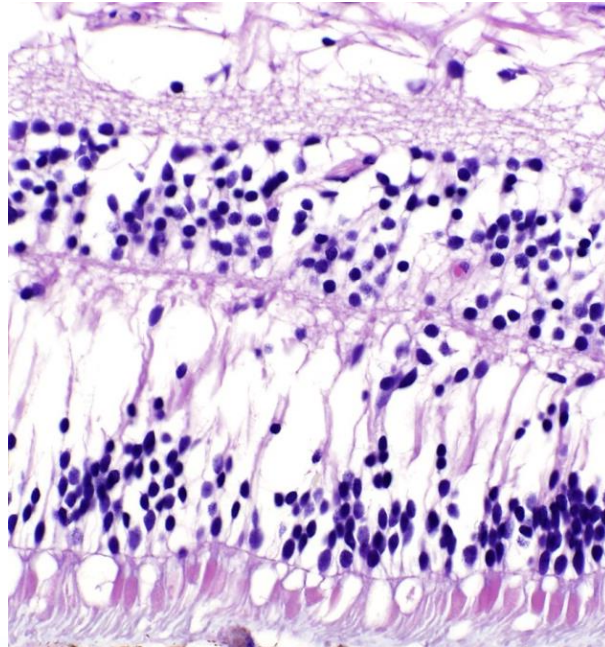
# Which image of the retina is diagnostic for glaucoma?

Vitreous



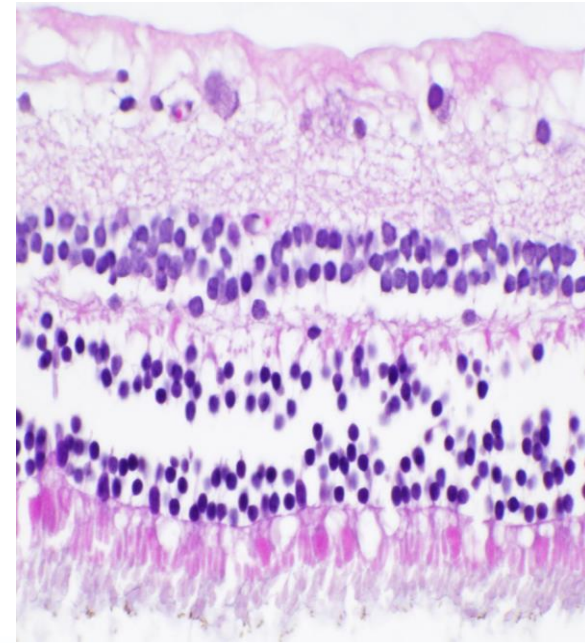
Choroid/sclera

Vitreous



Choroid/sclera

Vitreous

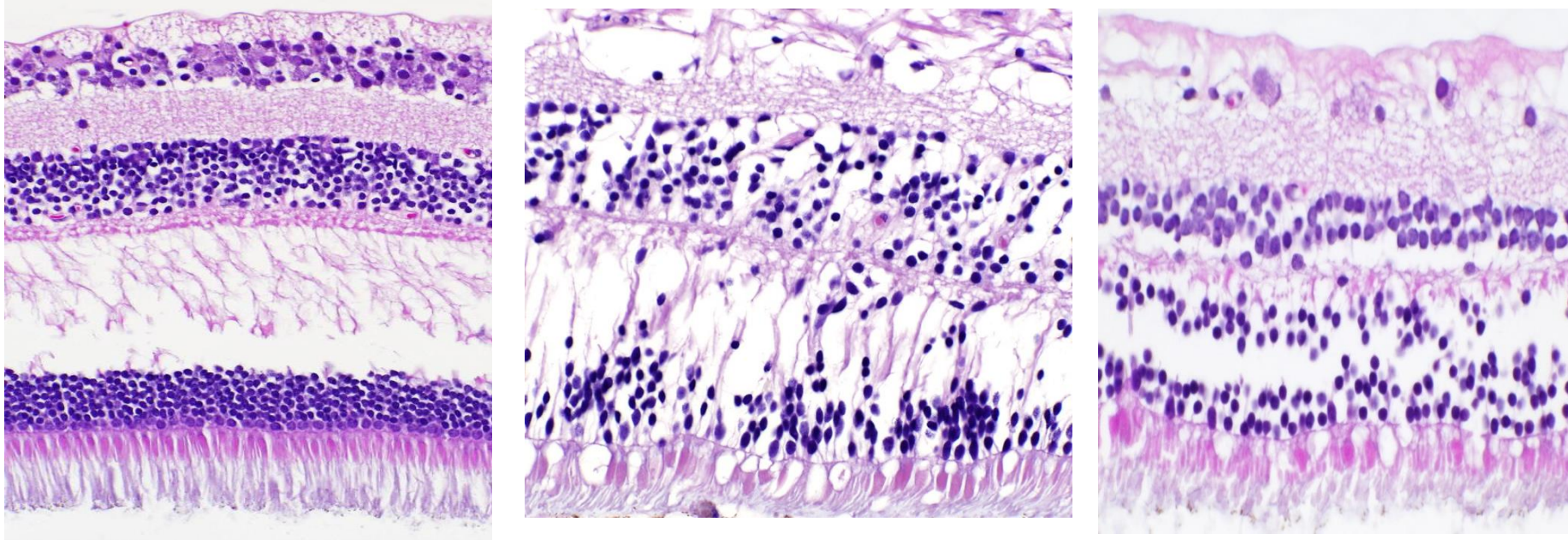


Choroid/sclera





# Answer: the image in the middle



**Left: normal macula (multilayer of ganglion cells), intact plexiform and nuclear layers.**

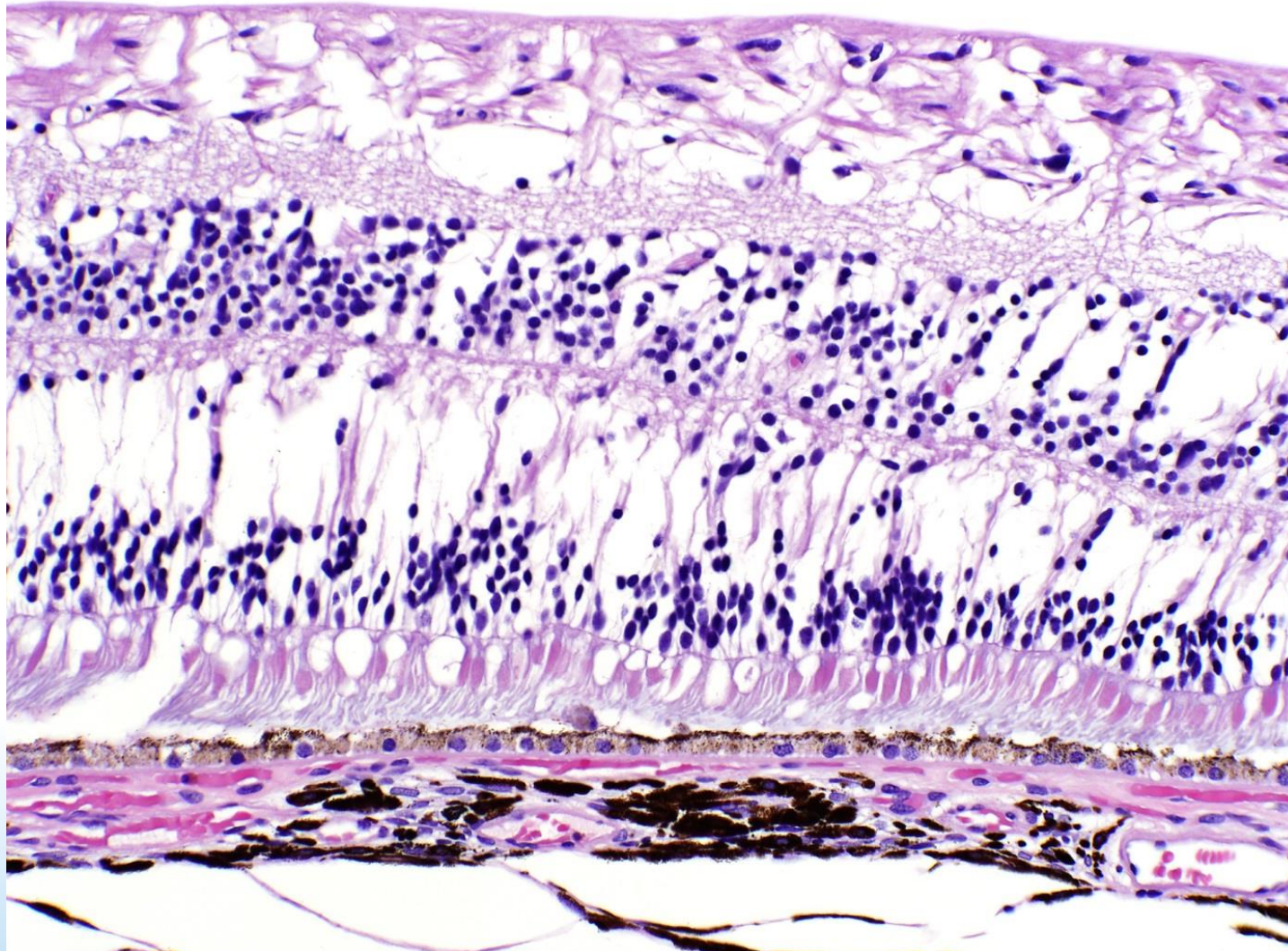
**Middle: shows loss of ganglion cells and atrophy of the nerve fiber layer**

**Right: normal peripheral retina with ganglion cells**





# Retina with absent ganglion cells in the nerve fiber layer



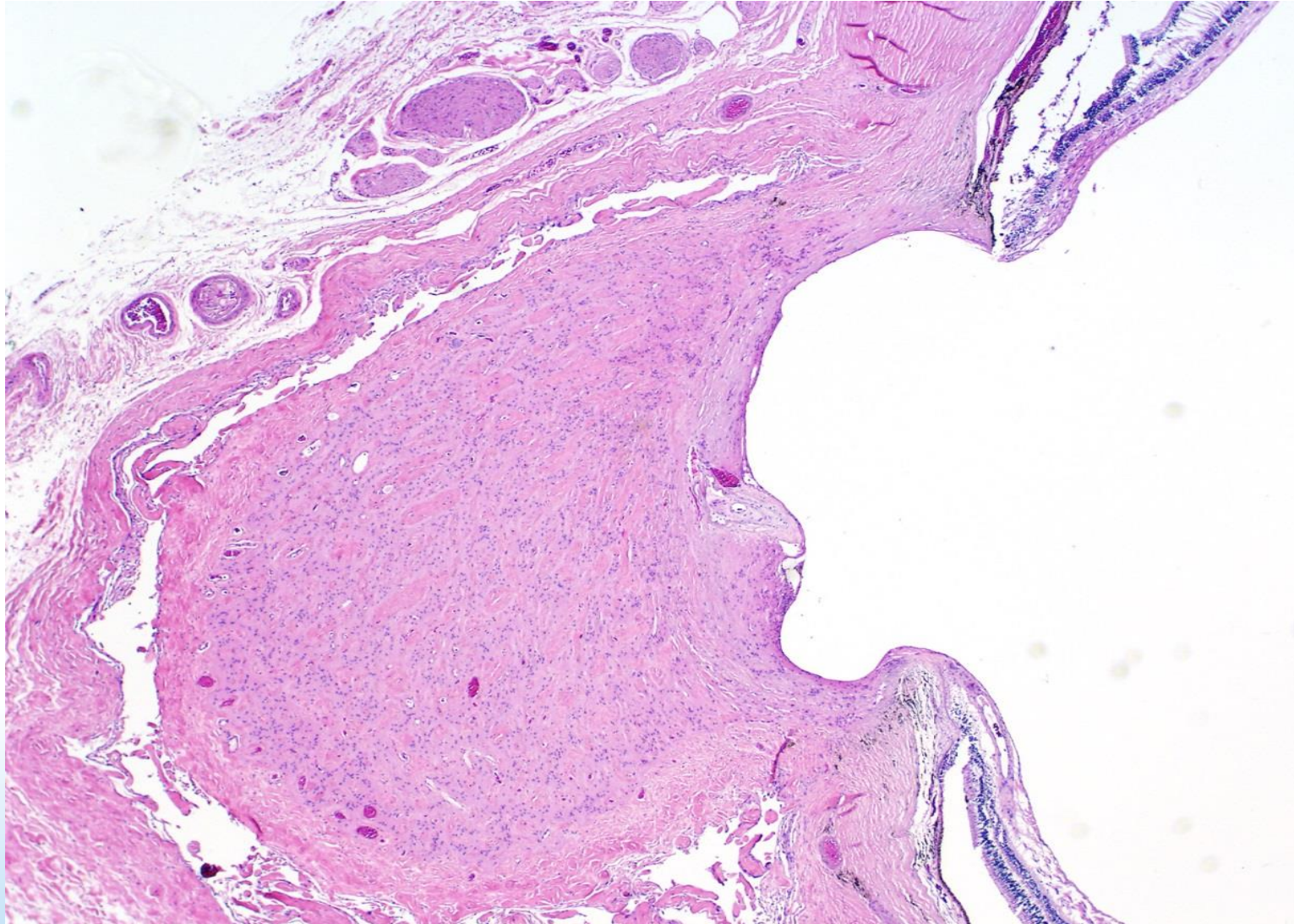
# Optic nerve cupping

- **Cupping of the optic disc distinguishes glaucomatous optic atrophy from primary optic atrophy**
- **Cupping also suggests that elevated intraocular pressure (IOP) is a major risk factor in the pathogenesis of glaucomatous optic atrophy**
- **Schnabel degeneration**
- **Lamina cribrosa**

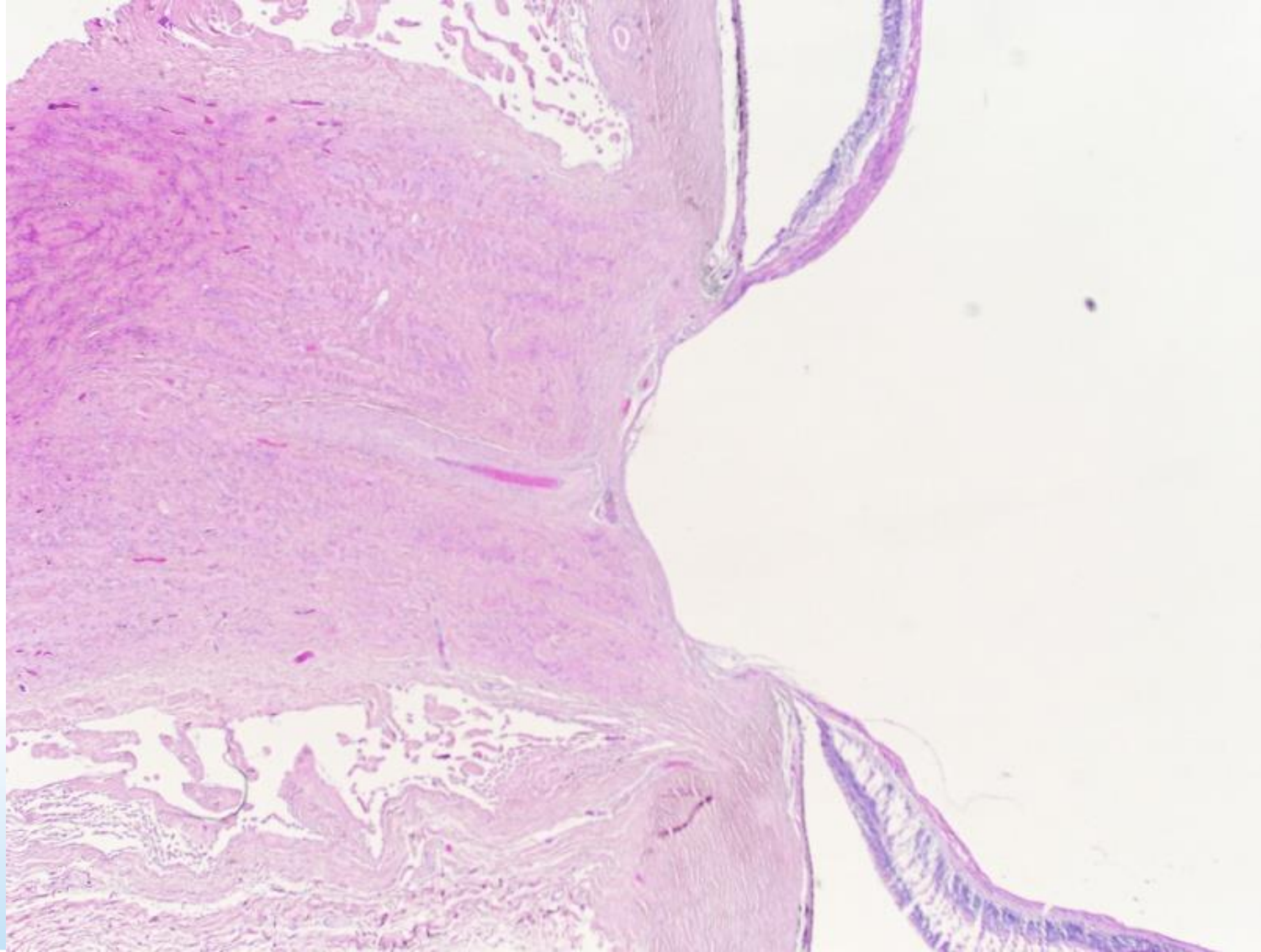




# Optic nerve cupping/excavation



# Optic nerve cupping





# Papilledema

- **Papilledema is the opposite of cupping, it is prominence of the optic cup due to increased intracranial pressure, e.g., brain tumor**



Normal disc OS

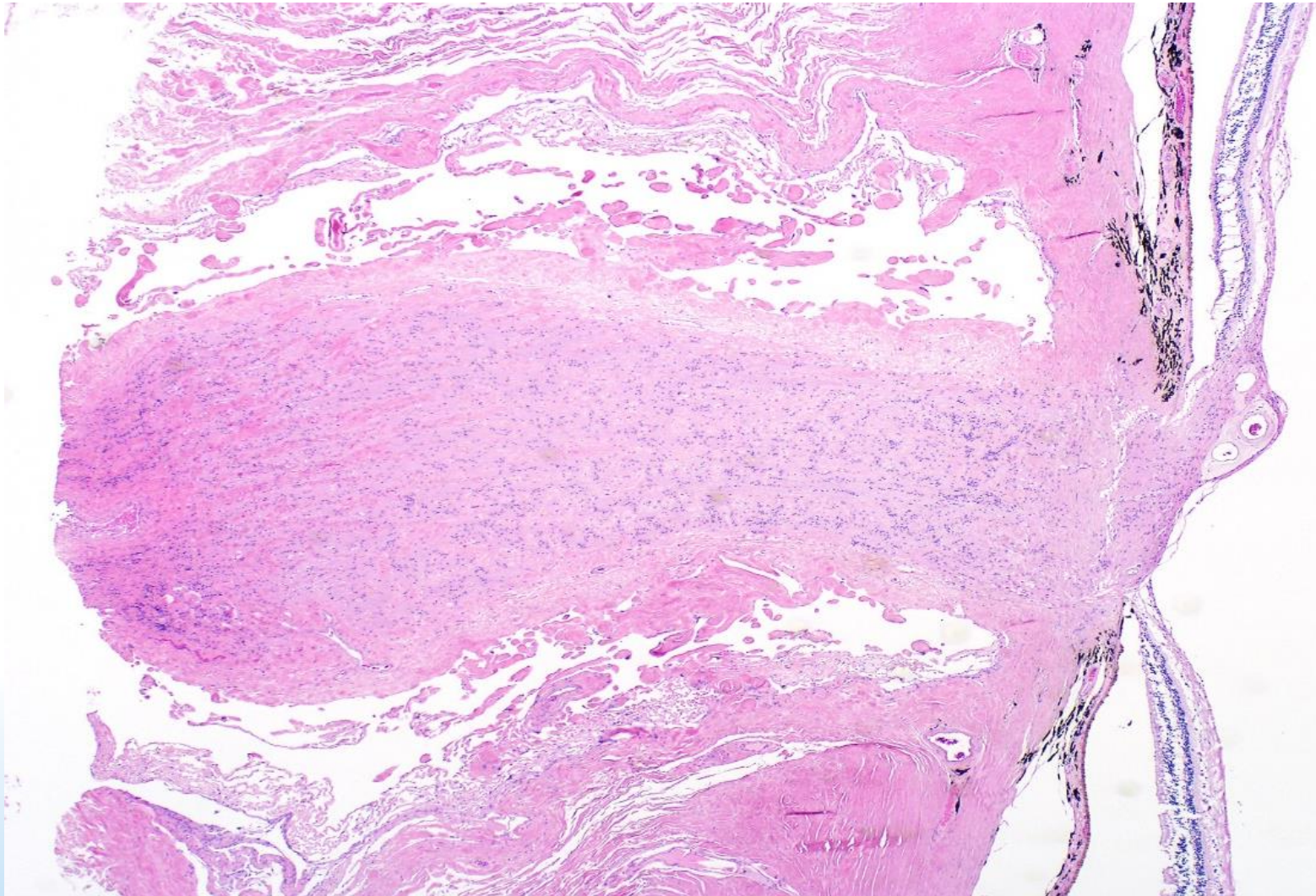


Papilledema - Meningioma





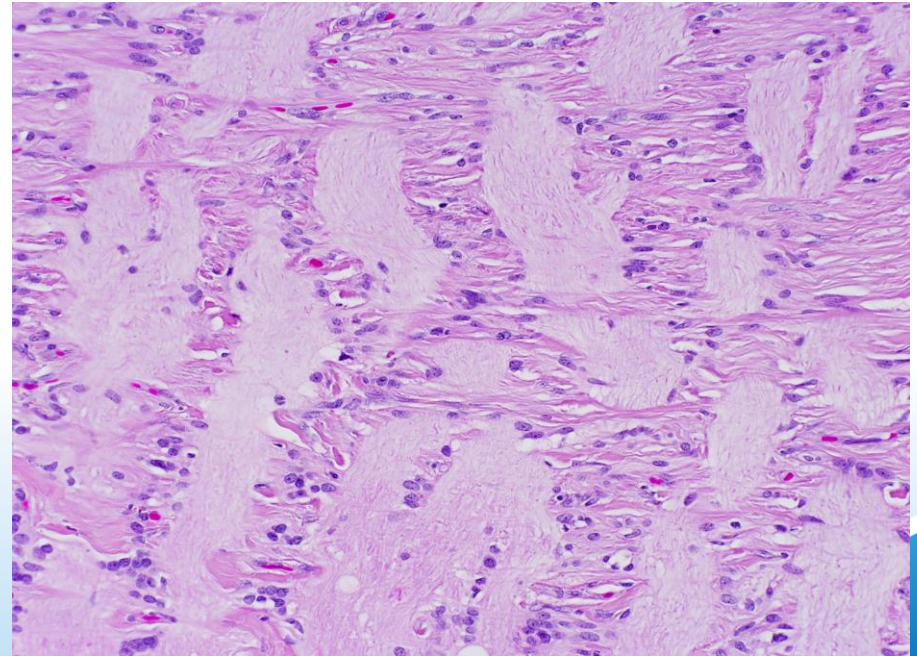
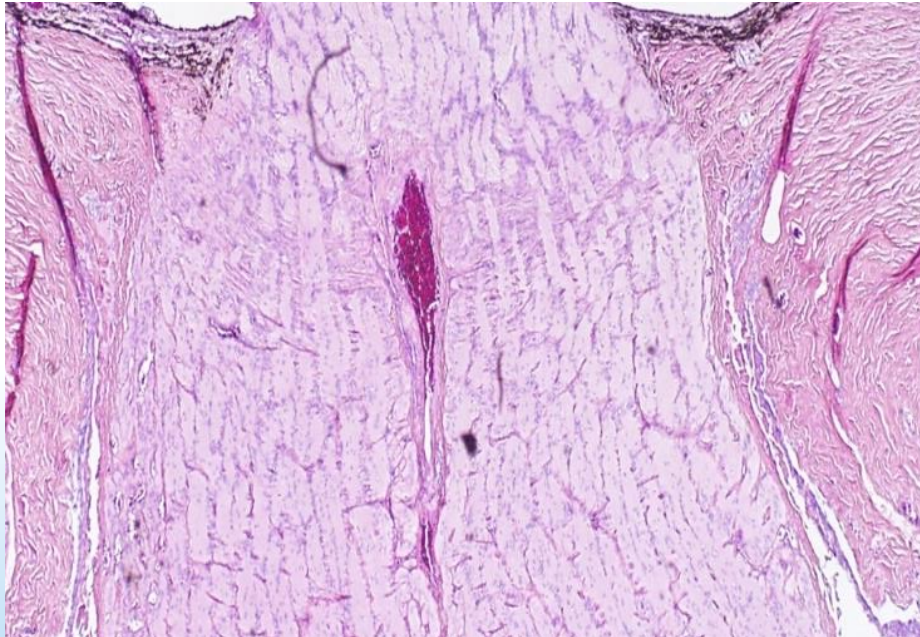
# Optic Nerve Atrophy (without cupping)





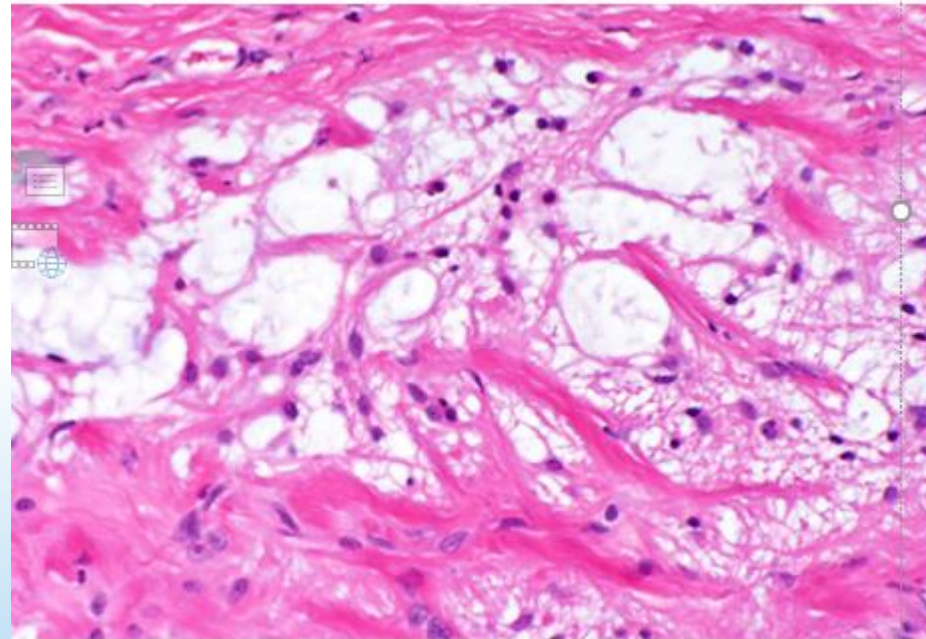
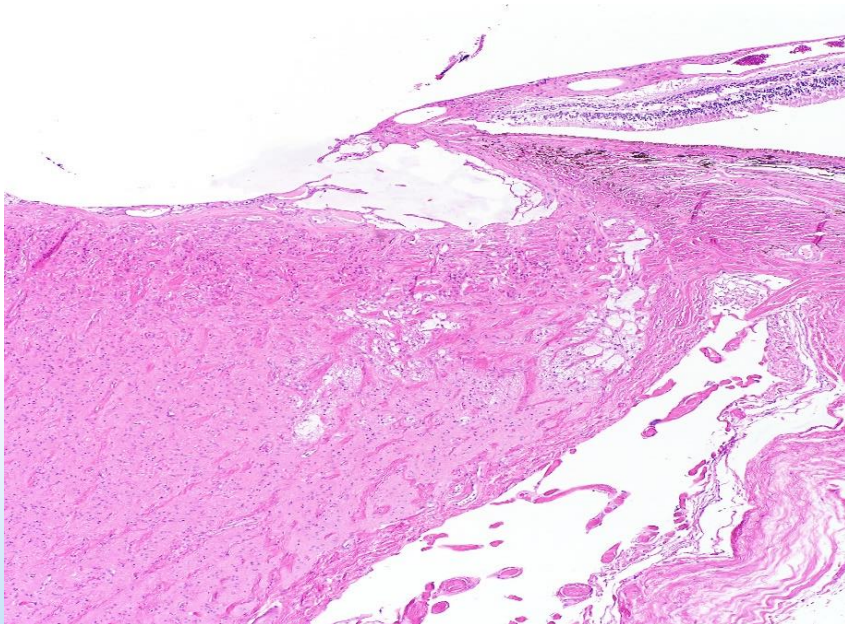
# Lamina cribrosa of the optic nerve head

- **Weakest part of the sclera, through which the optic nerve and the central retinal artery and vein enter the globe**
- **Lamina cribrosa abnormalities may be associated with glaucoma progression, e.g., axon compression and blockage of axonal flow**



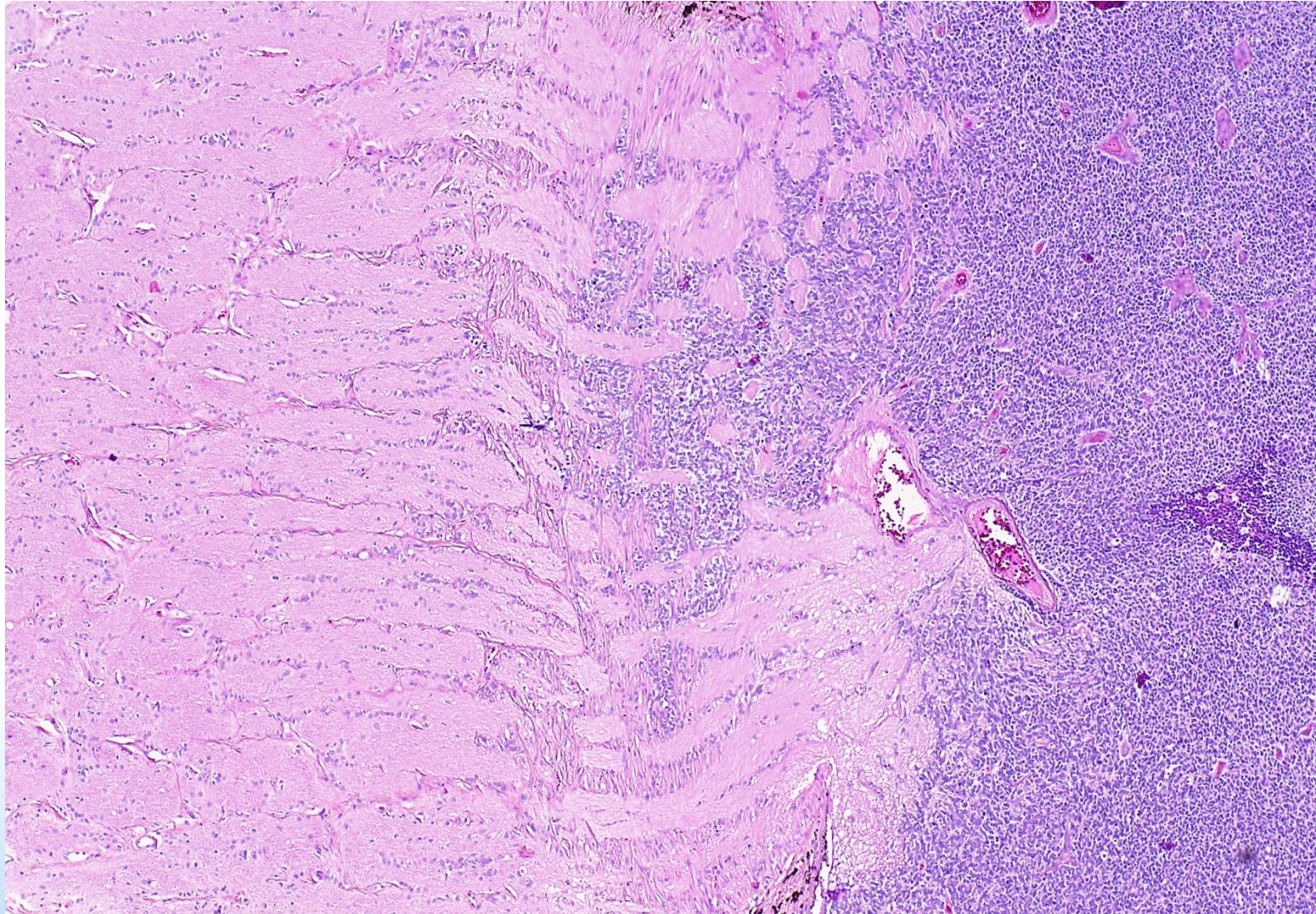
# Schnabel degeneration

- **Hyaluronic acid (acid mucopolysaccharide) accumulation posterior to the lamina cribrosa**
  - Alcian blue, colloidal iron
- **Seen in glaucoma; vascular origin?**
  - Giarelli L, et al., Archives of Pathology & Laboratory Medicine: 2003, 127: 1314-1319.





# Lamina cribrosa involved by retinoblastoma



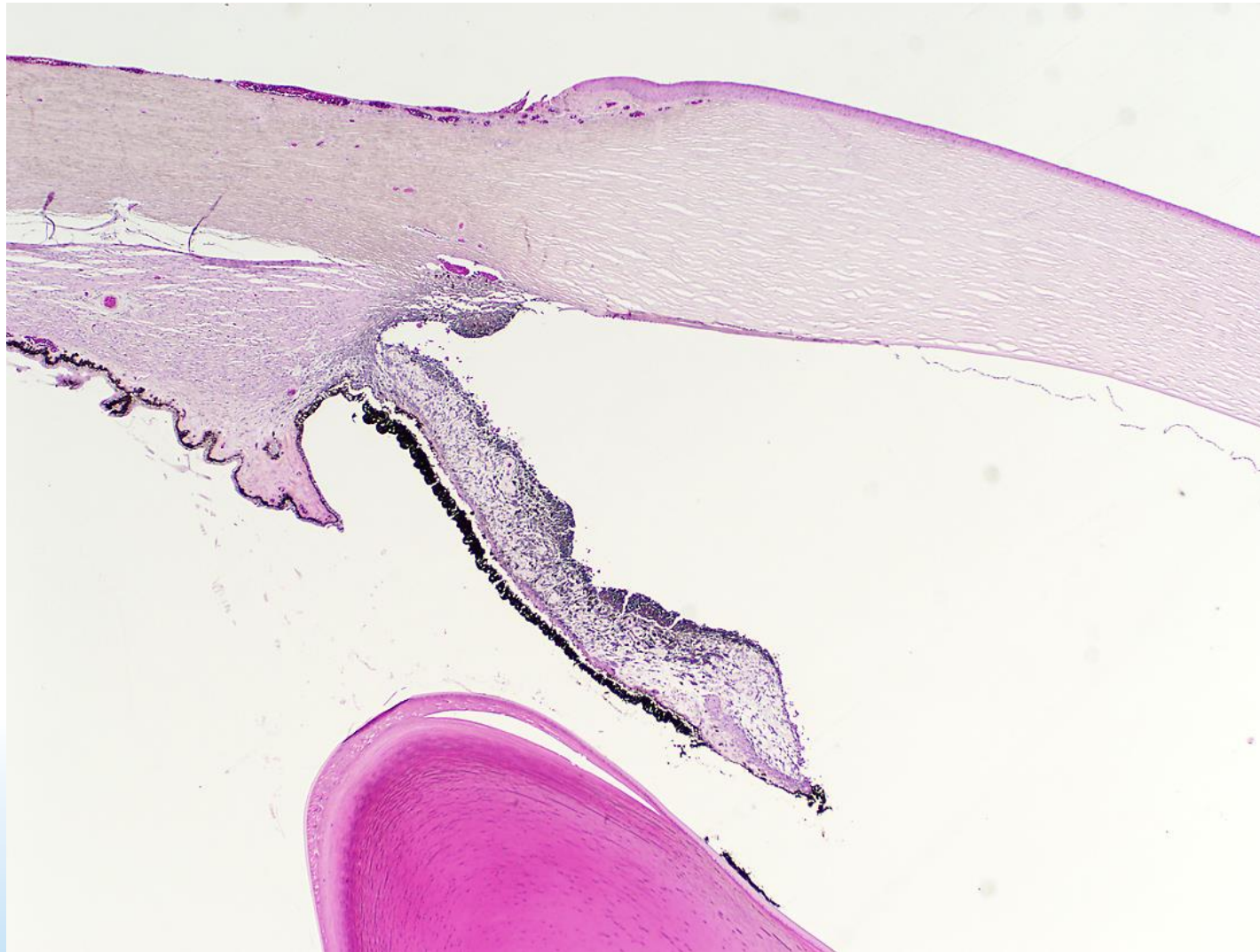


# Anterior chamber angle

- **Anterior: lined by corneal endothelium**
- **Peripheral: lined by the trabecular meshwork and the anterior face of the ciliary body and iris root**
- **Posterior: lined by the anterior surface of the iris and pupillary portion of the lens**

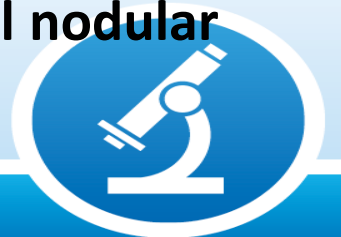


# Normal anterior chamber angle

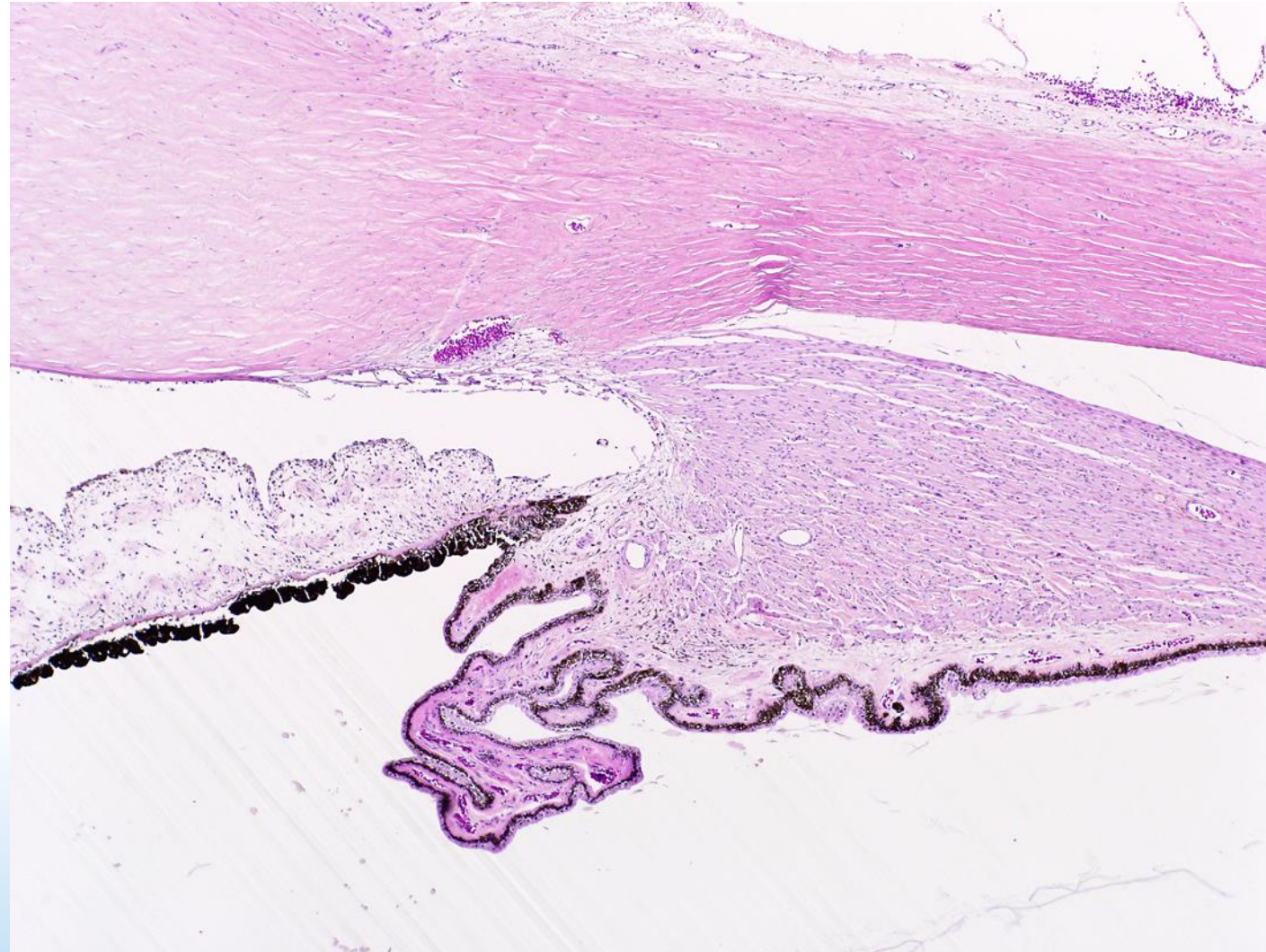


# Anterior chamber angle: Key structures and landmarks

- **Scleral spur**
  - posterior edge of the trabecular meshwork
- **Schlemm canal**
  - lymphatic-like vascular drainage site for aqueous humor
- **Trabecular meshwork**
  - Connective tissue responsible for aqueous fluid outflow
- **Schwalbe line / posterior embryotoxon**
  - Schwalbe line demarcates the termination of Descemet membrane and the anterior border of the trabecular meshwork; posterior embryotoxon is an anatomical nodular prominence of Schwalbe line

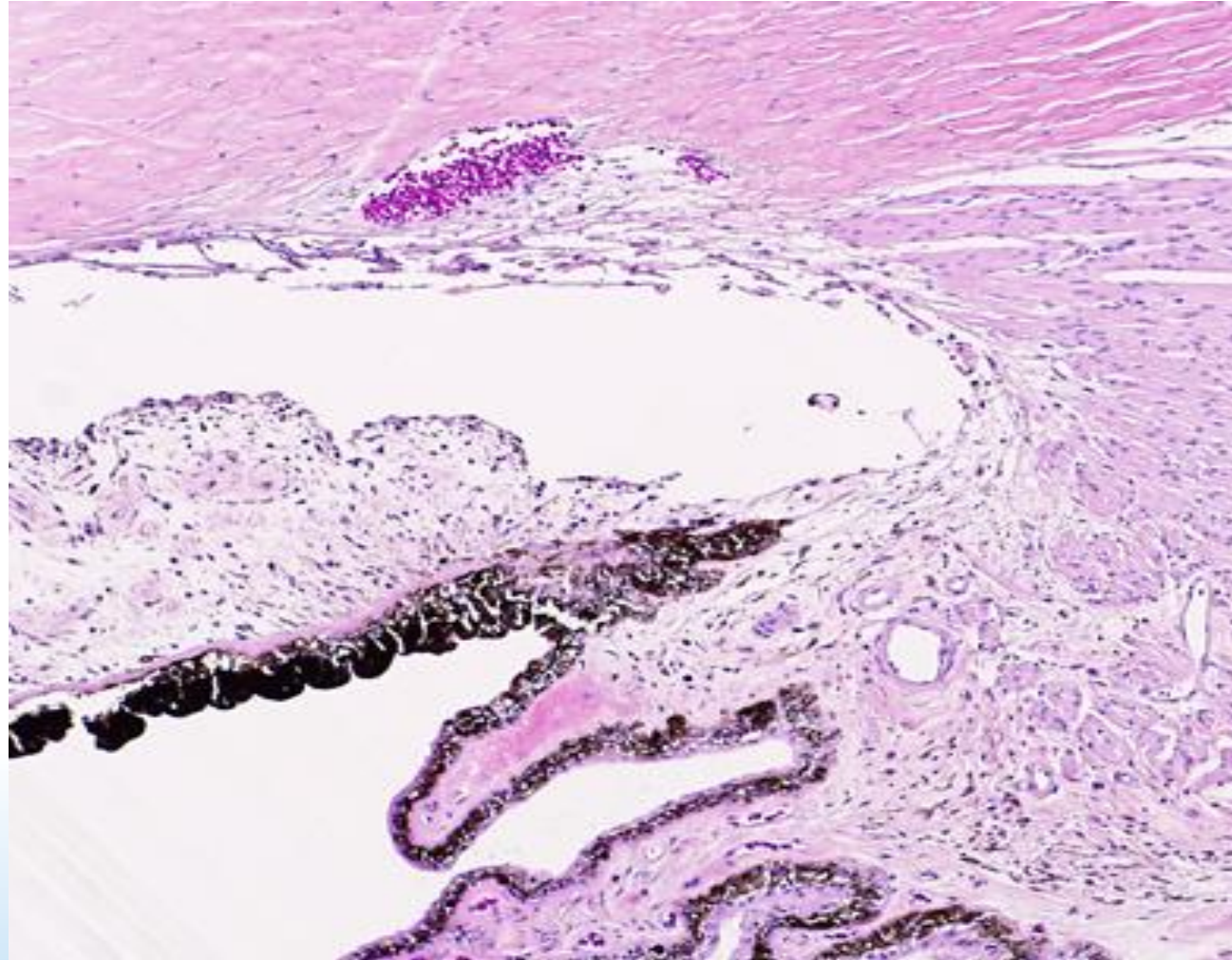


# Normal open anterior chamber angle





# Normal open anterior chamber angle



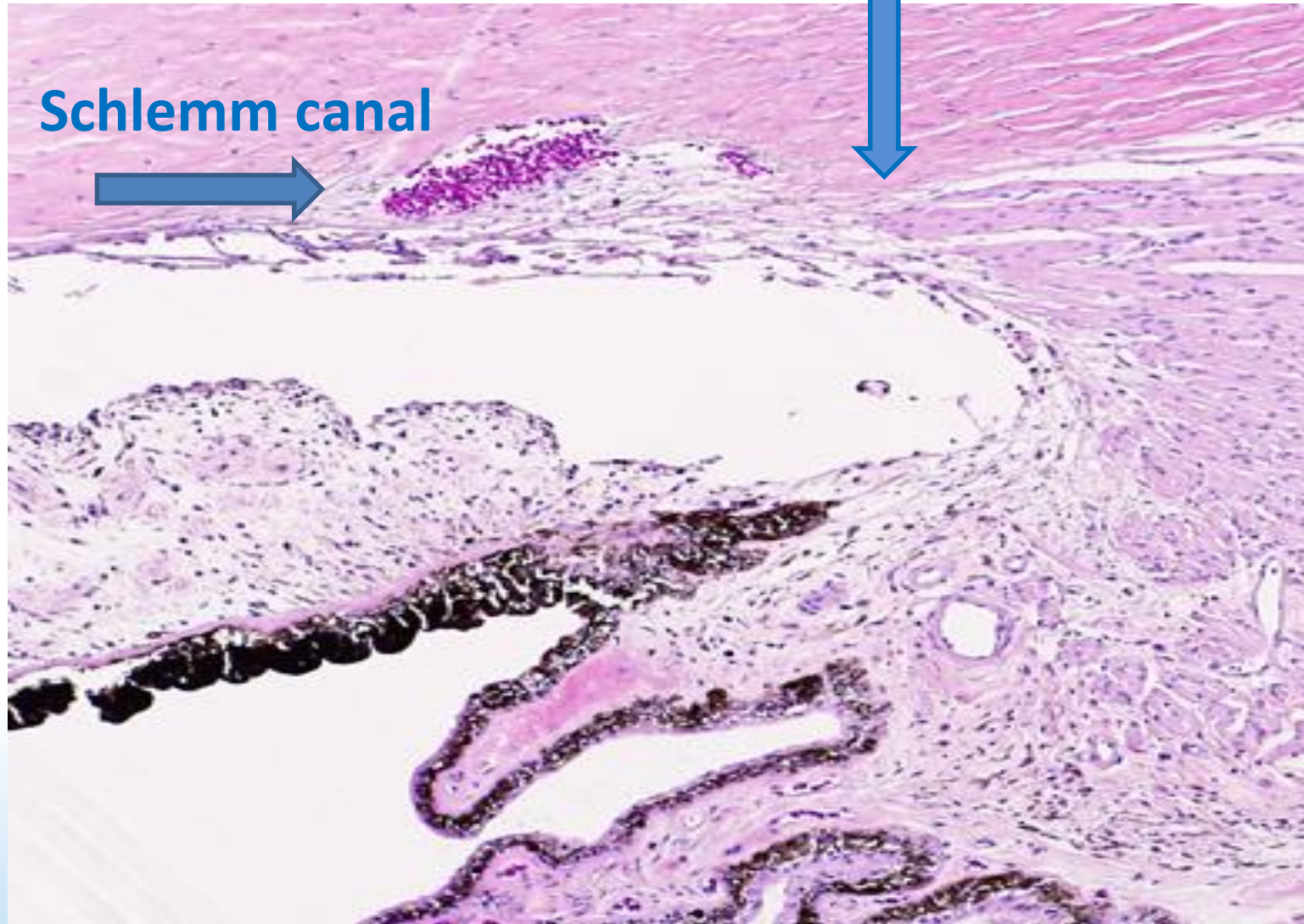
# Scleral spur

- **Attachment site for ciliary body muscle**
- **It is the posterior edge of the trabecular meshwork**
- **The scleral spur is the key clinical landmark in gonioscopy: if visualized, then by definition the angle is open**



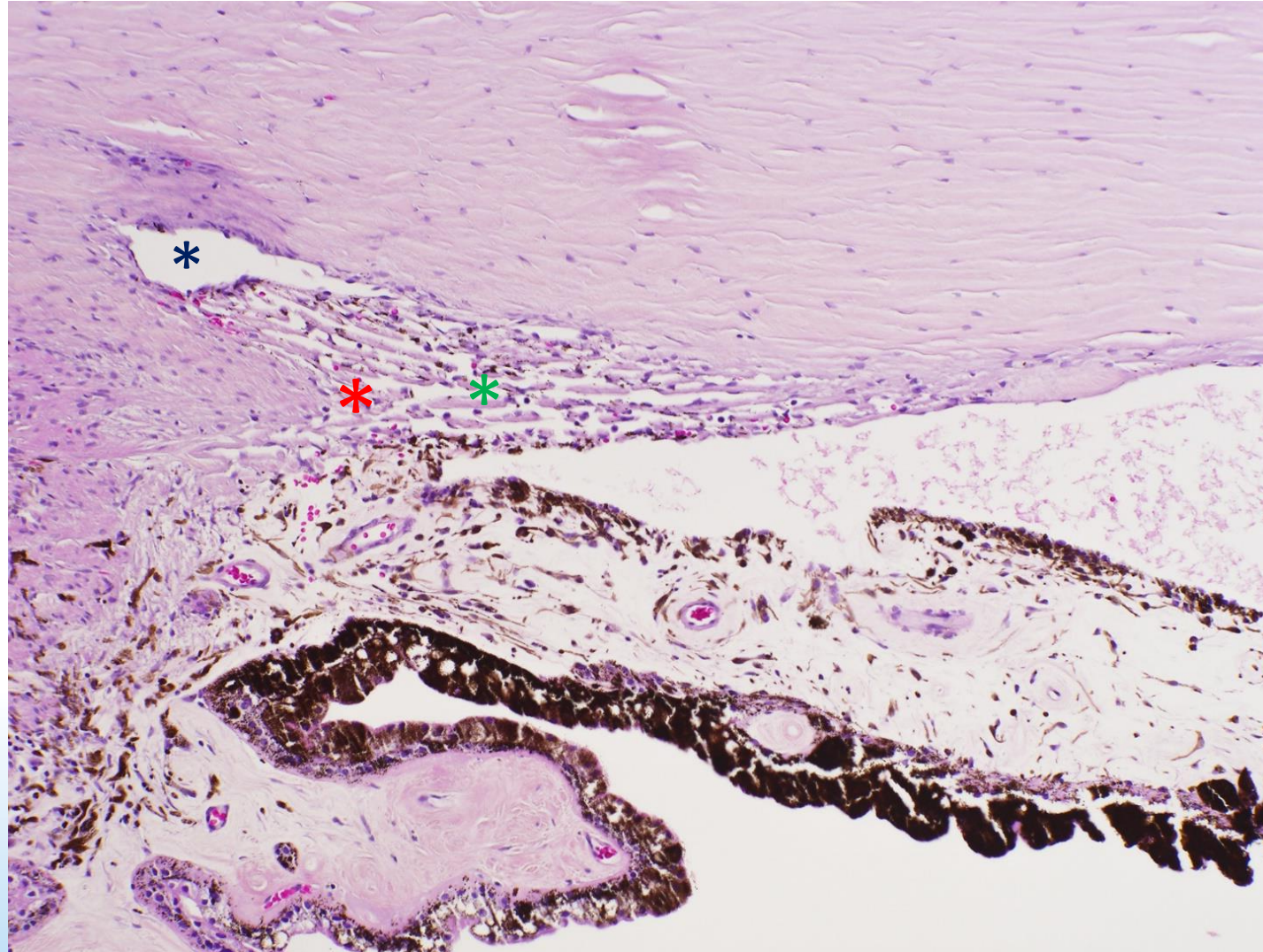
Schlemm canal

Scleral spur



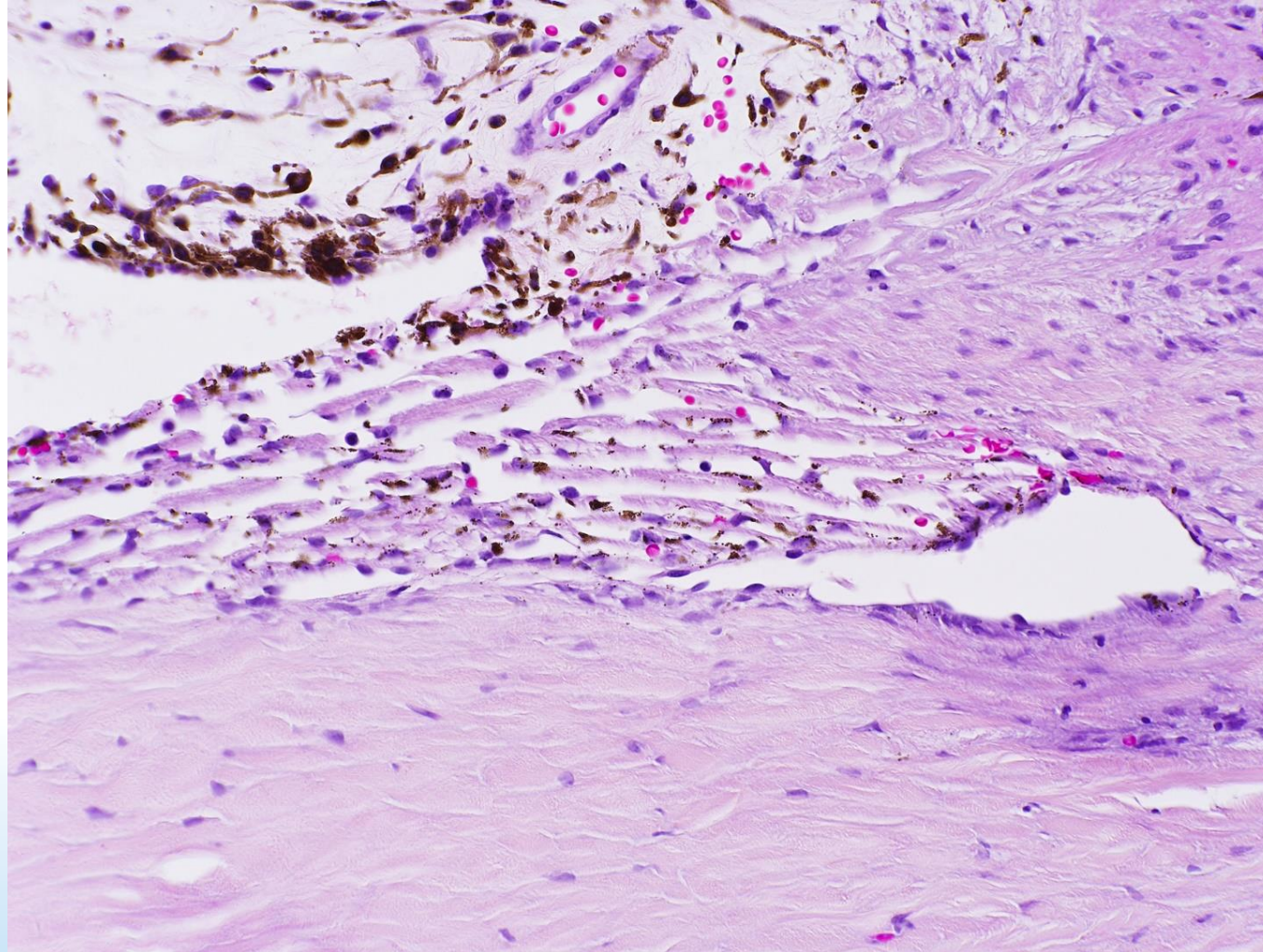


Schlemm canal\*, trabecular meshwork\* (collagen beams)  
anterior ciliary body muscle (scleral spur\*), juxtacanalicular tissue



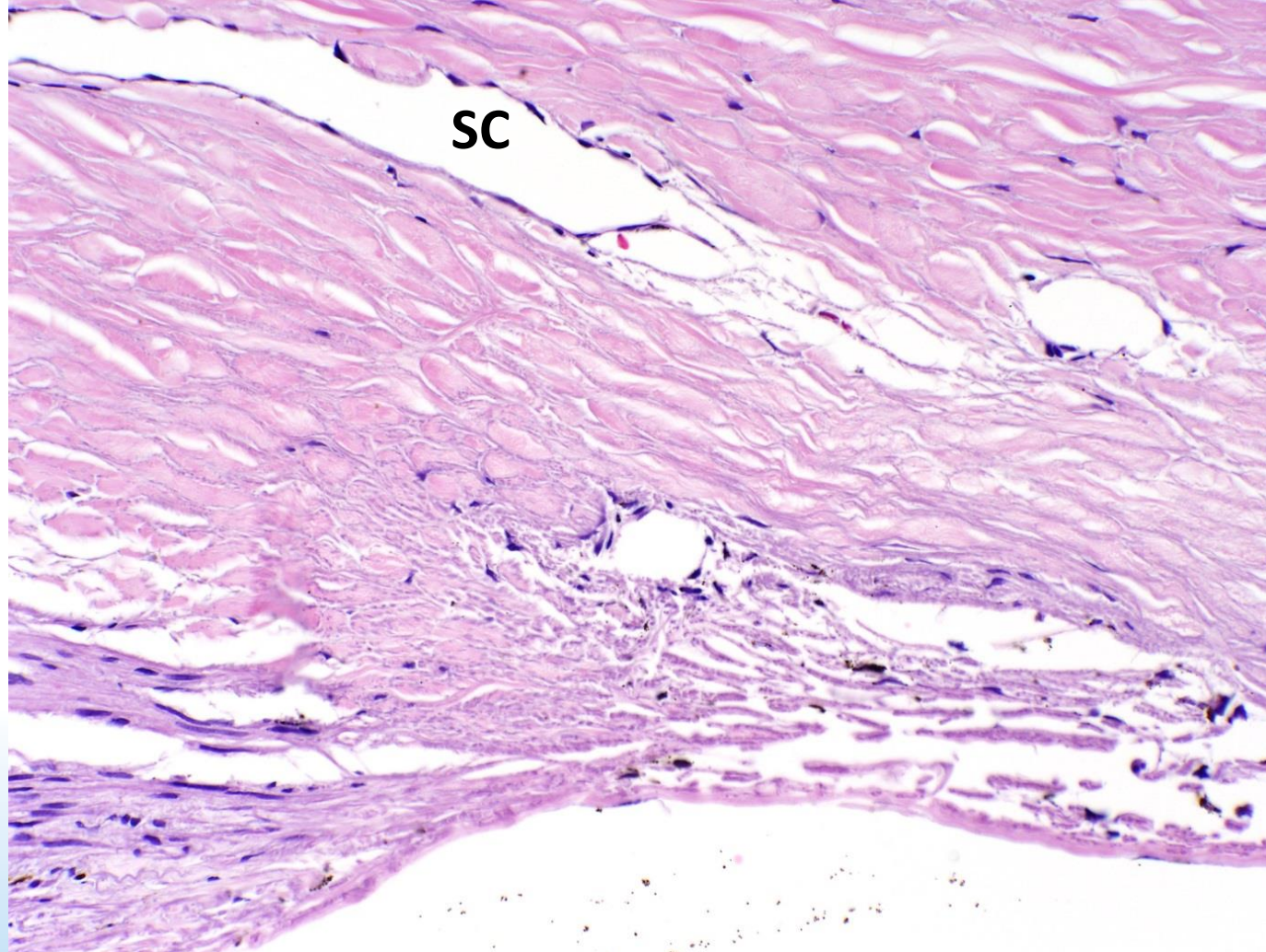


**Schlemm canal, trabecular meshwork (collagen beams)  
anterior ciliary body muscle, juxtacanalicular tissue**





# Trabecular meshwork: collagen beams and elastic tissue



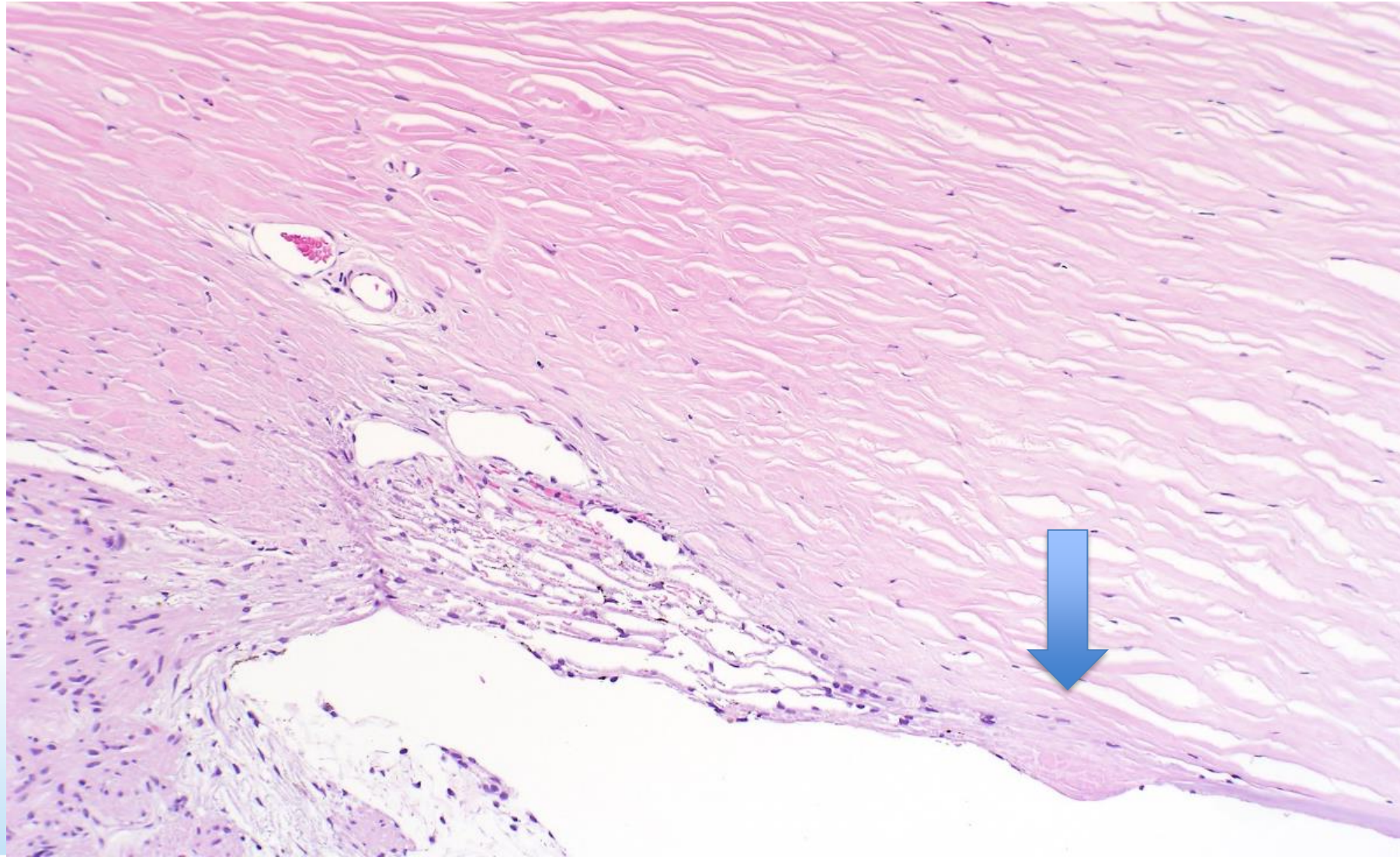
# Schwalbe line // Posterior embryotoxon

- **Schwalbe line: the termination of Descemet membrane; the anterior border of the trabecular meshwork**
- **Posterior embryotoxon (sometimes seen in congenital glaucoma): thickening/nodular prominence of Schwalbe line**
- **Need a fortuitous section to identify it**



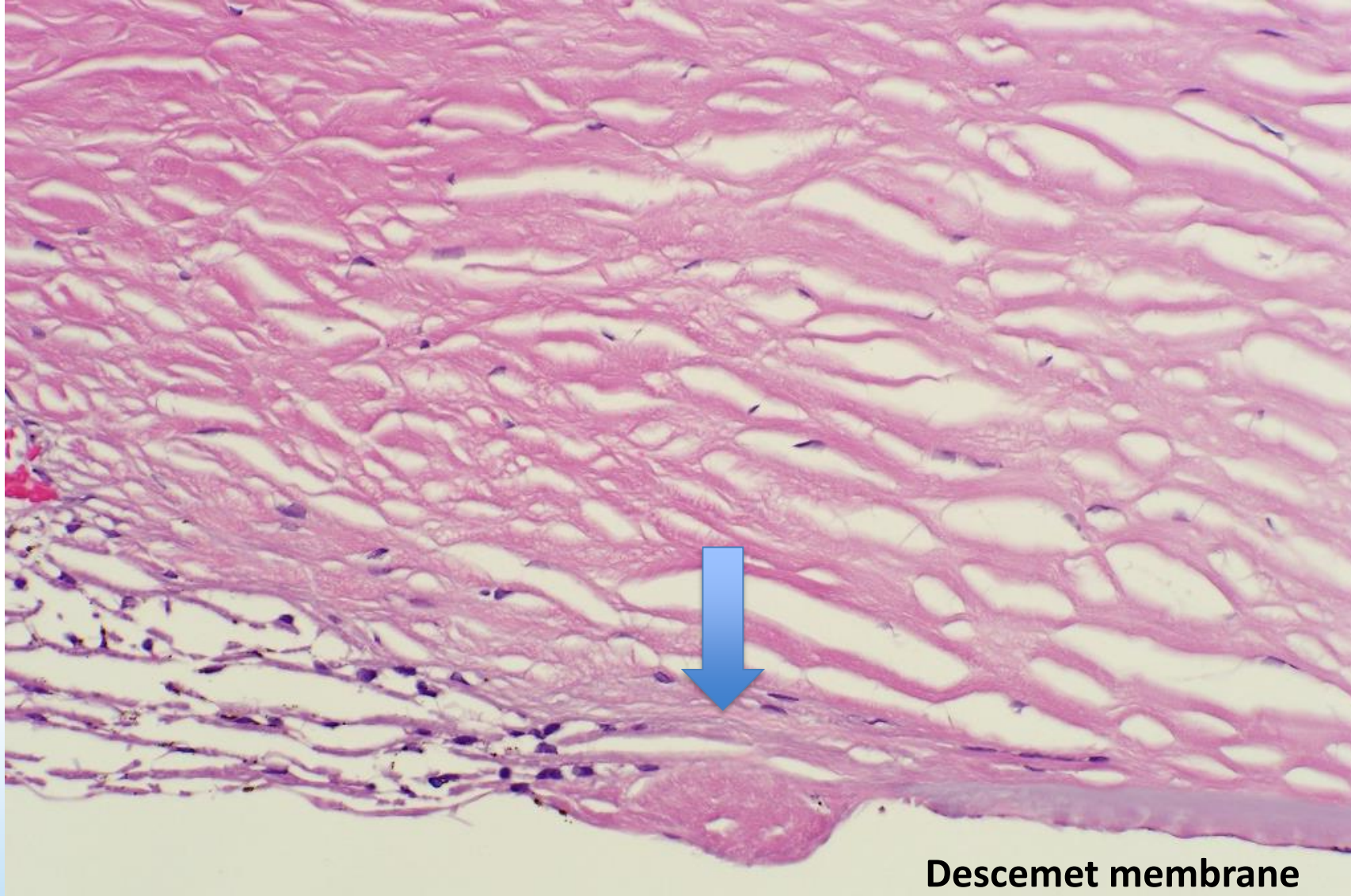


# Posterior embryotoxon





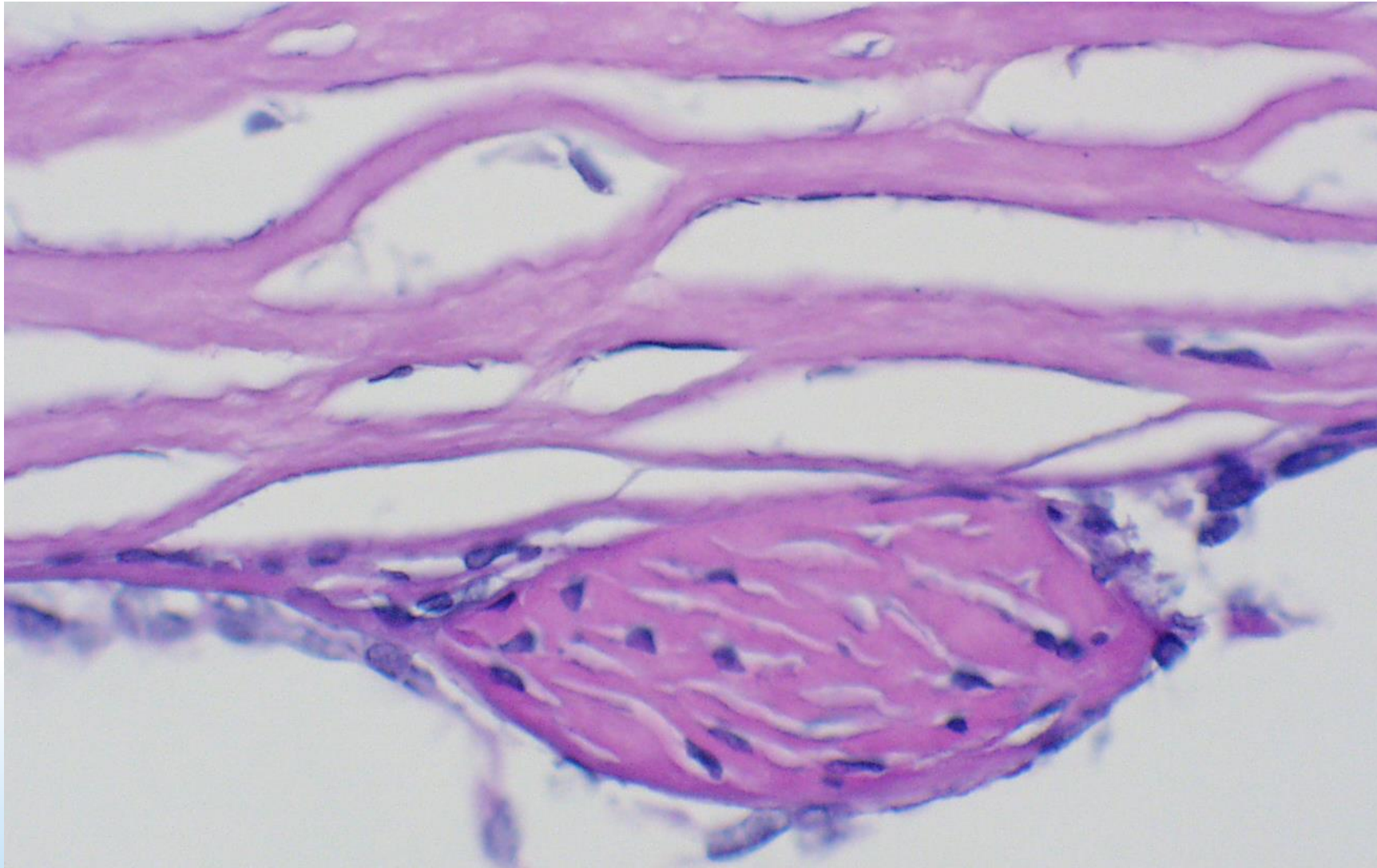
# Posterior embryotoxon



Descemet membrane



# Posterior embryotoxon



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# Classification of glaucoma

- **Primary open-angle glaucoma**
- **Primary closed-angle glaucoma**
  - (acute: red eye) (chronic: exfoliation/pseudoexfoliation)
- **Congenital glaucoma**
- **Normal-tension glaucoma**
  
- **Secondary glaucoma (non-acute, angle open or closed)**



# Primary open-angle glaucoma

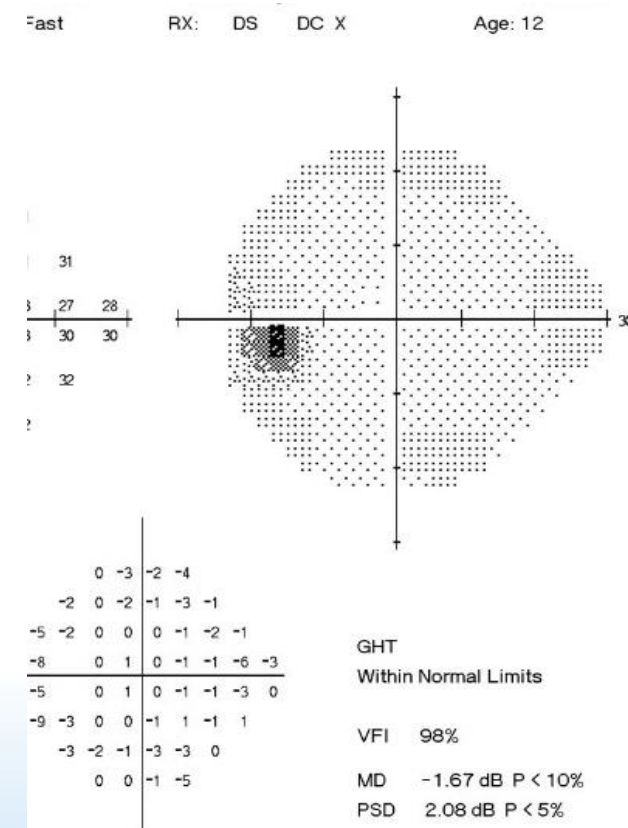
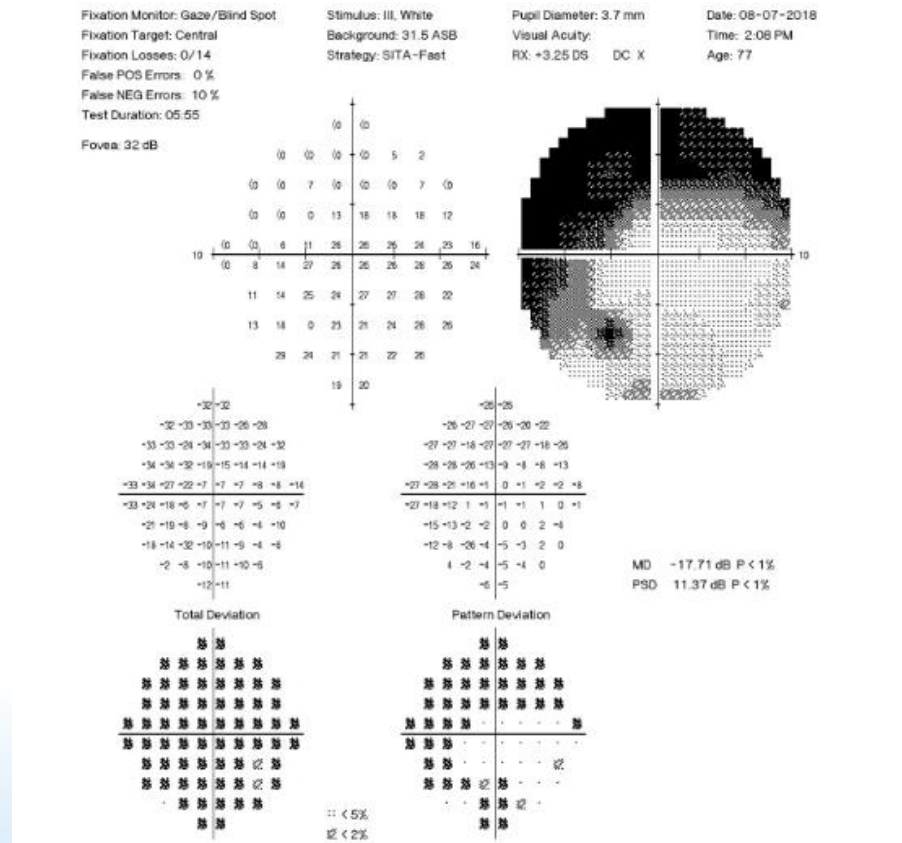
- **Idiopathic**
- **No antecedent disease**
- **Most common type of glaucoma**
- **Affects 1-3% of the population**
- **Older individuals**
  
- **Asymptomatic painless visual loss**
- **Frequently bilateral**
- **Frequent family history**

Kwon et al, N Engl J Med 2009; 360:1113-1124



# Humphrey visual field Superior and inferior arcuate

# Normal (for comparison)



# Primary open-angle glaucoma

- **Complex genetics, linked to 14 genes**
- **Mutations in myocilin (*MYOC*) gene on chromosome 1 found in 3-5% of patients with familial forms of POAG**
- **High eye pressures due to reduced aqueous outflow through the trabecular meshwork and Schlemm canal, with intracellular aggregation of the mutant myocilin protein**
- **Anterior chamber angle is open**
  - vs acute closed-angle glaucoma





# Closed angle glaucoma (primary/acute)

- Adherence/apposition of the peripheral iris to the trabecular meshwork

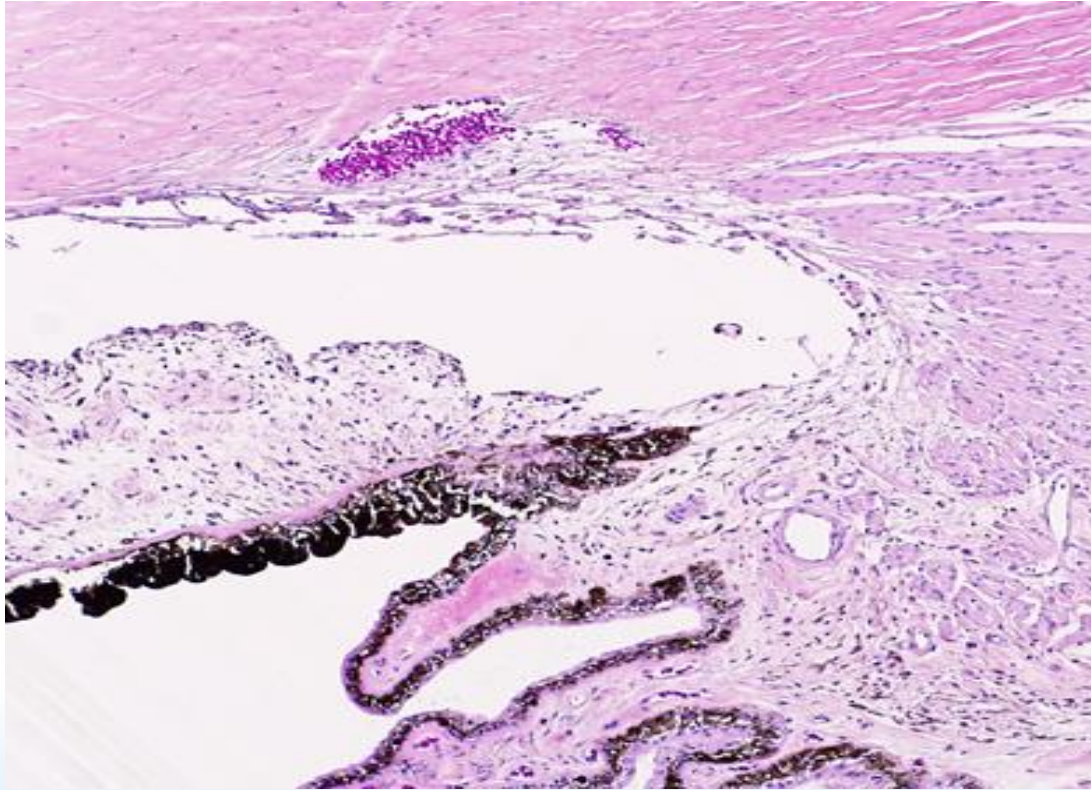


Acute Angle-Closure Glaucoma. Pohl, H and Tarnutzer, AA. N Engl J Med 2018; 378:e14

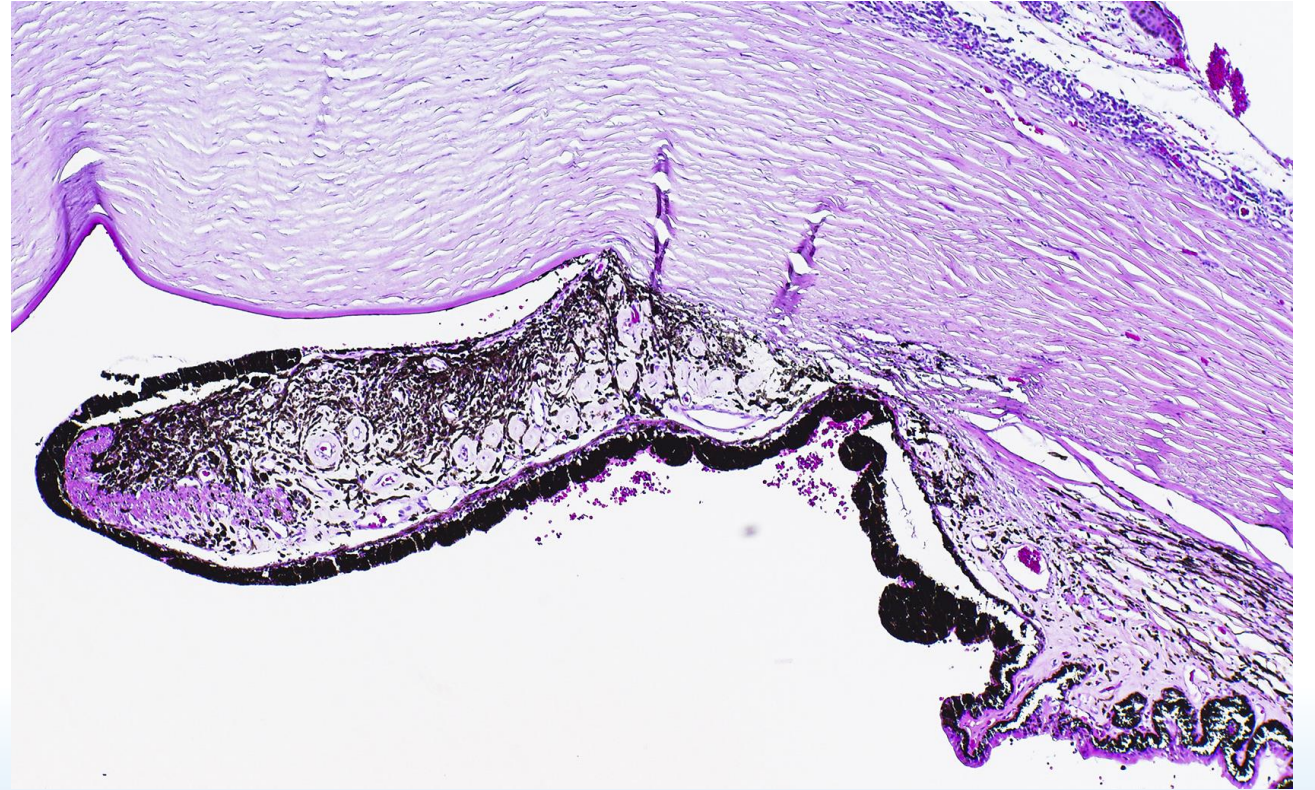




## Open anterior chamber angle



## Closed anterior chamber angle



# 2 scanned slides

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AANP November 2...

1 - AANP Novem...

2 - AANP Novem...



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The image displays a histological slide of a cross-section of an eye, stained with hematoxylin and eosin (H&E). The central feature is the lens, which is surrounded by the lens capsule and the lens fibers. The iris is visible on the right side, showing its characteristic structure. The surrounding tissues, including the ciliary muscles and the choroid, are also visible. The slide is presented in a software interface with a toolbar at the top and a sidebar on the left showing two thumbnails of the same slide.



# Congenital glaucoma

- Buphthalmos (ocular enlargement, ox eye) is a clinical hallmark of congenital glaucoma
- Autosomal recessive cases: mutations in cytochrome *P4501B1* (*CYP1B1*) gene, chromosome 2
- Hypothetical mechanisms:
  - An imperforate mesodermal sheet covering the trabecular meshwork
  - Congenital absence of Schlemm canal
  - Persistence of a fetal angle configuration with mesenchymal tissue in the angle
- Haab striae: healed ruptures in Descemet membrane



# Congenital glaucoma, buphthalmos



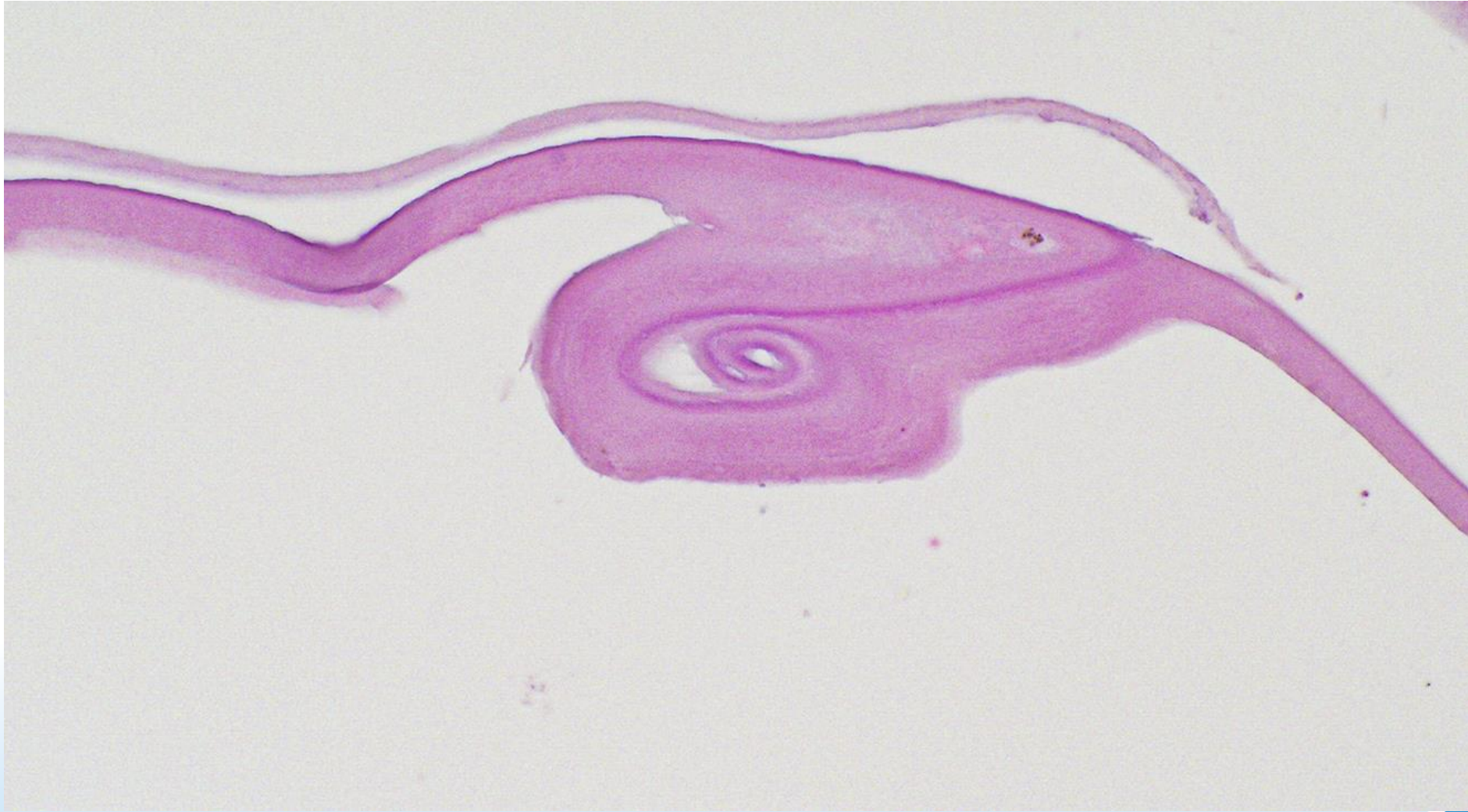
# Haab striae

- **Descemet membrane coils and forms a scroll when it ruptures and heals.**
- **Differential diagnosis:**
  - Haab striae – congenital glaucoma
  - Keratoconus
  - Obstetrics forceps injury

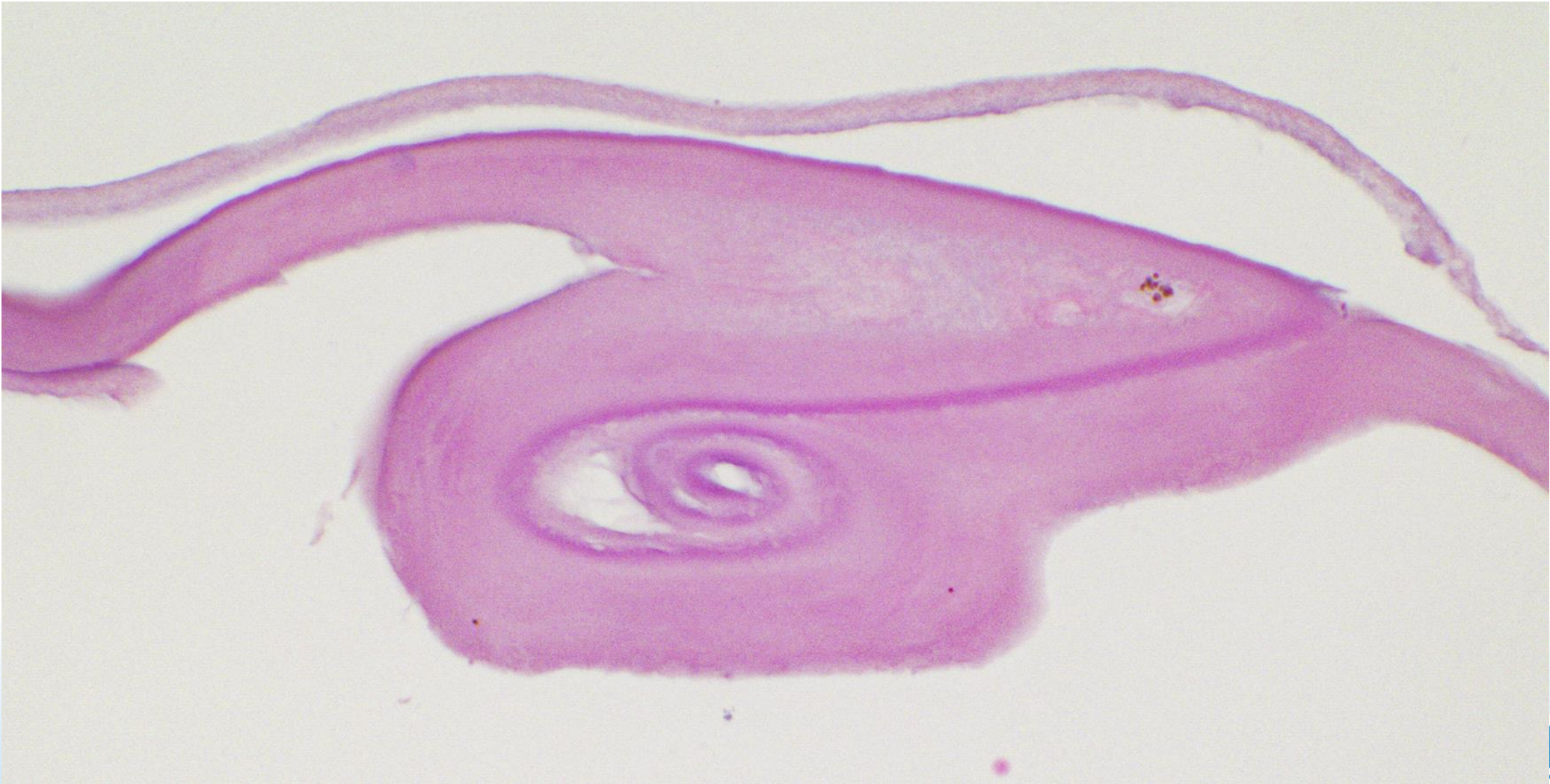




# Haab striae



# Haab striae





# Classification, types of glaucoma

- **Secondary types of glaucoma:**
  - Epithelial downgrowth
  - Irido Corneal Endothelial syndrome (ICE)
  - Neovascular glaucoma (diabetes mellitus)
  
  - Tumor associated melanoma (melanomalytic)
  - Traumatic glaucoma
  - Pigmentary glaucoma
  
  - Pseudoexfoliative glaucoma
  - Orbital pathology: tumors, thyroid ophthalmopathy,
    - can raise IOP by directly compressing the globe
  - Uveitic glaucoma



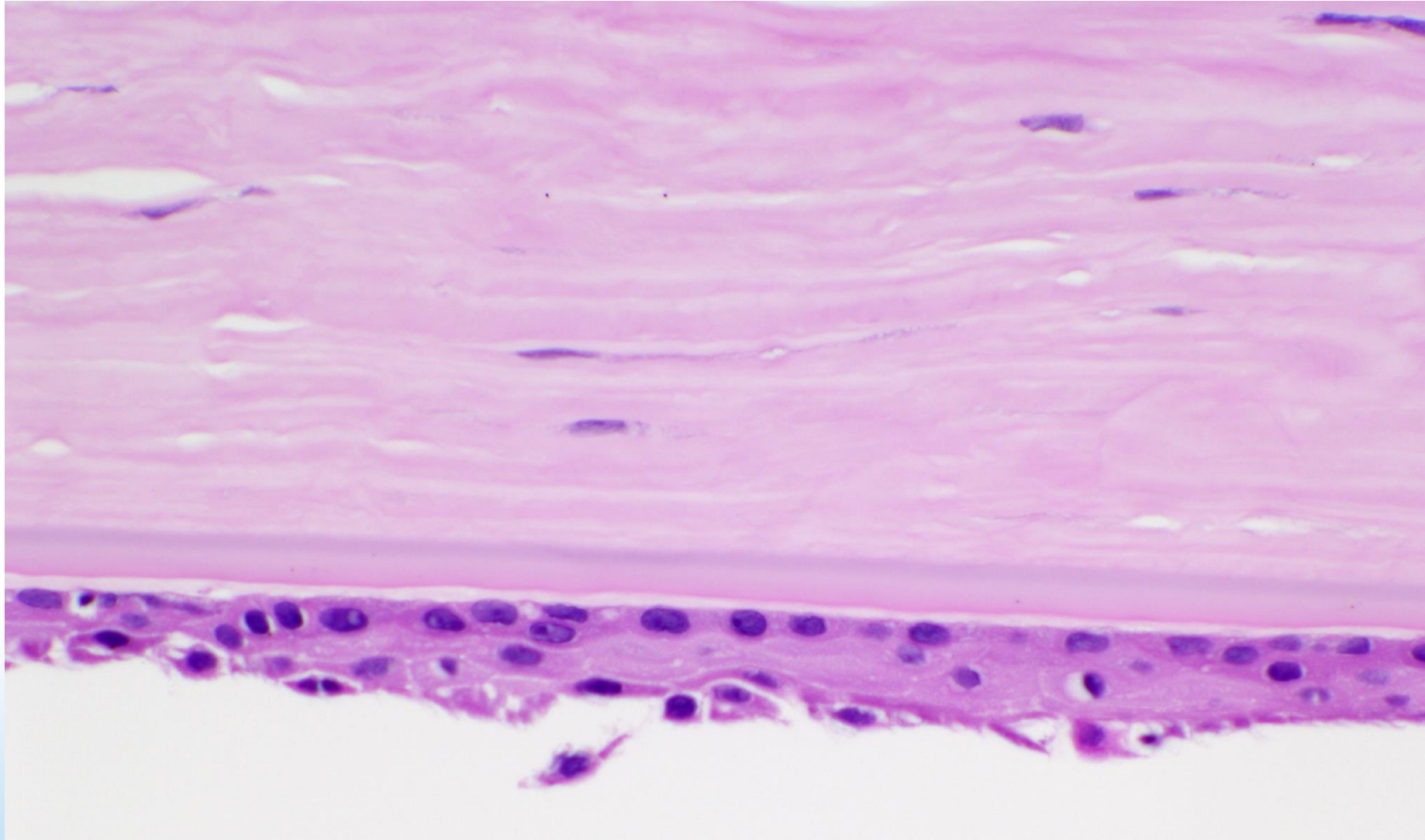
## **Secondary closed-angle glaucoma: Proliferation of cells on anterior chamber structures**

- **Epithelial downgrowth/ingrowth**
  - Ocular surface epithelium lines posterior cornea, trabecular meshwork, anterior surface of the iris after surgical or nonsurgical trauma
- **Proliferation of abnormal corneal endothelial cells**
  - Iridocorneal endothelial (ICE) syndrome – Descemetization of anterior surface of iris
- **Iris neovascularization**
  - Neovascular glaucoma, diabetes

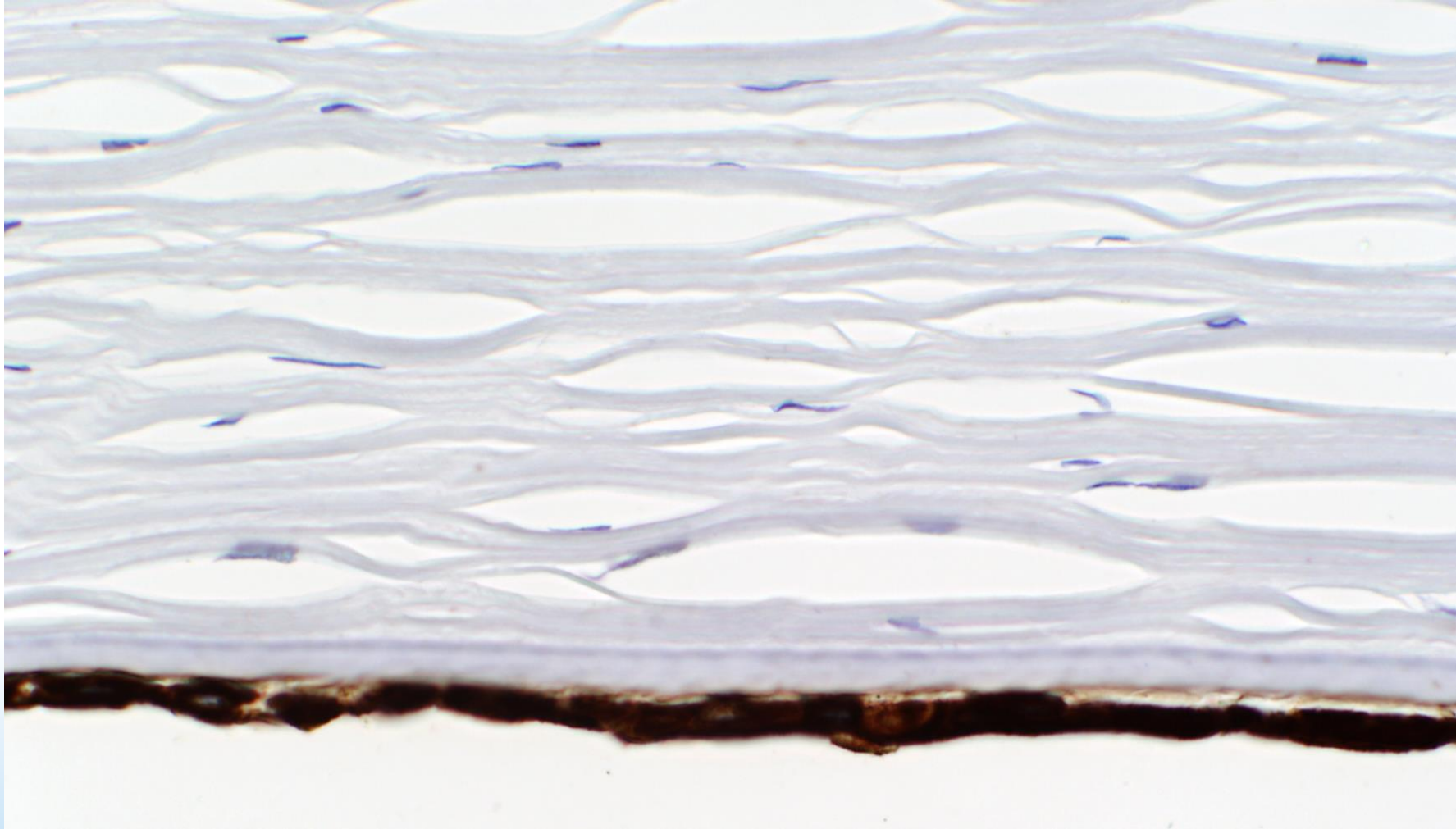




# Epithelial downgrowth / ingrowth

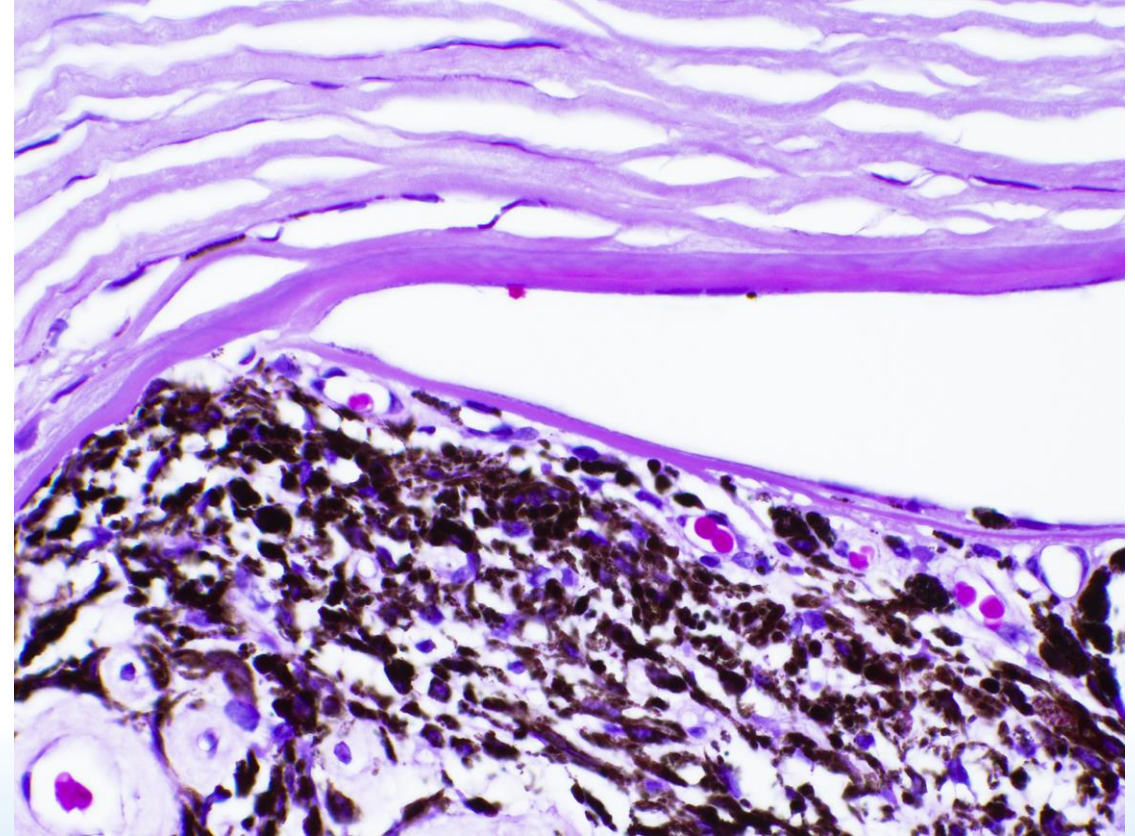


## Epithelial ingrowth/downgrowth: Cytokeratin immunohistochemistry



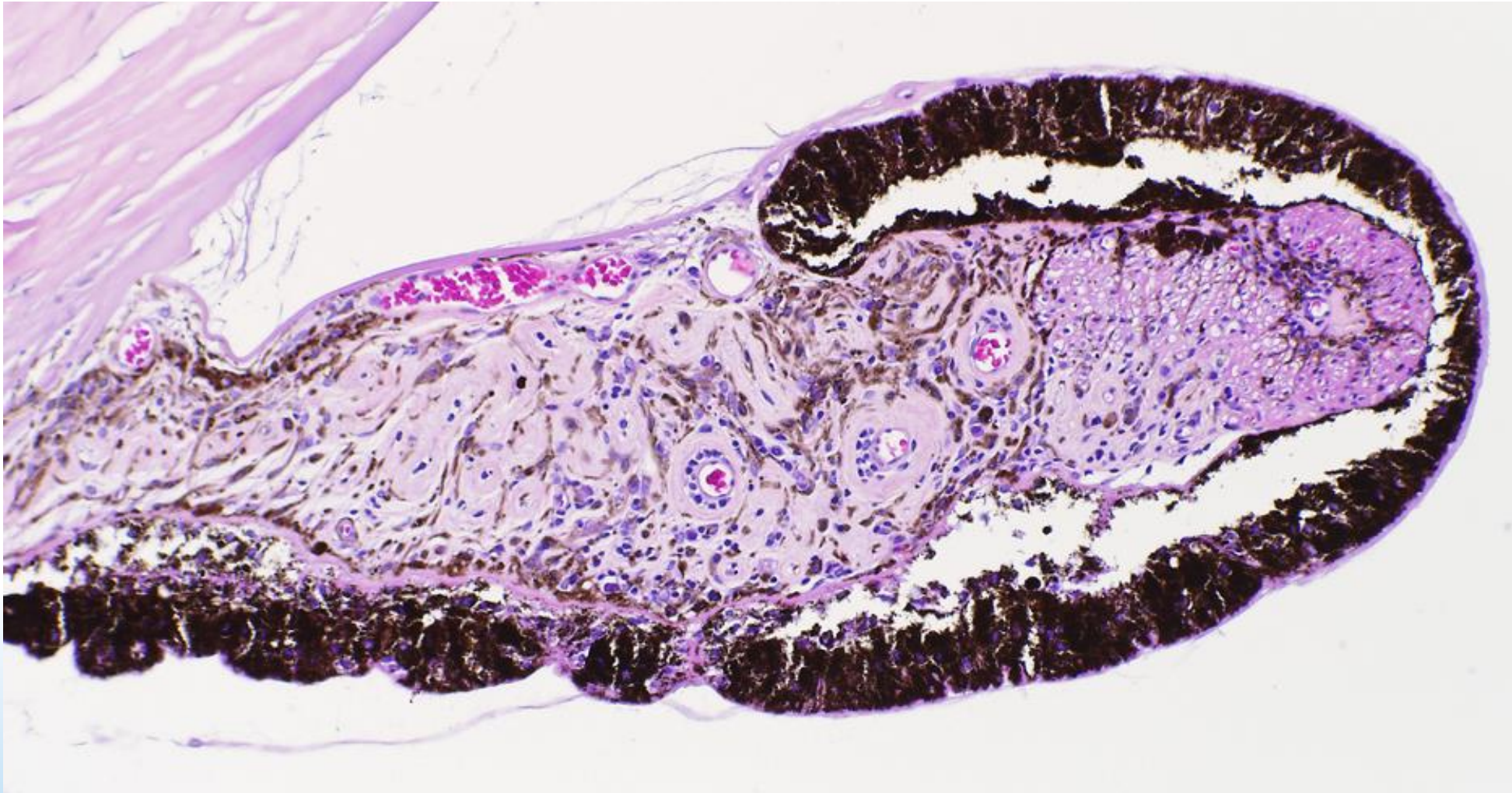


**Irido Corneal Endothelial syndrome: Descemet membrane lines the anterior surface of the iris (Descemetization) + peripheral anterior synechiae = closed angle**





**Irido Corneal Endothelial syndrome: Descemet membrane lines the anterior surface of the iris, peripheral anterior synechiae, ectropion uveae**



# Neovascular glaucoma

- **Abnormal formation of new blood vessels on the surface of the iris and into the angle**
- **Always associated with other abnormalities**
  - Diabetes, trauma, tumors (retinoblastoma)
- **The new blood vessels block aqueous fluid from exiting through the trabecular meshwork**
- **Eye pressure increases**



# Diabetic retinopathy // Neovascular glaucoma



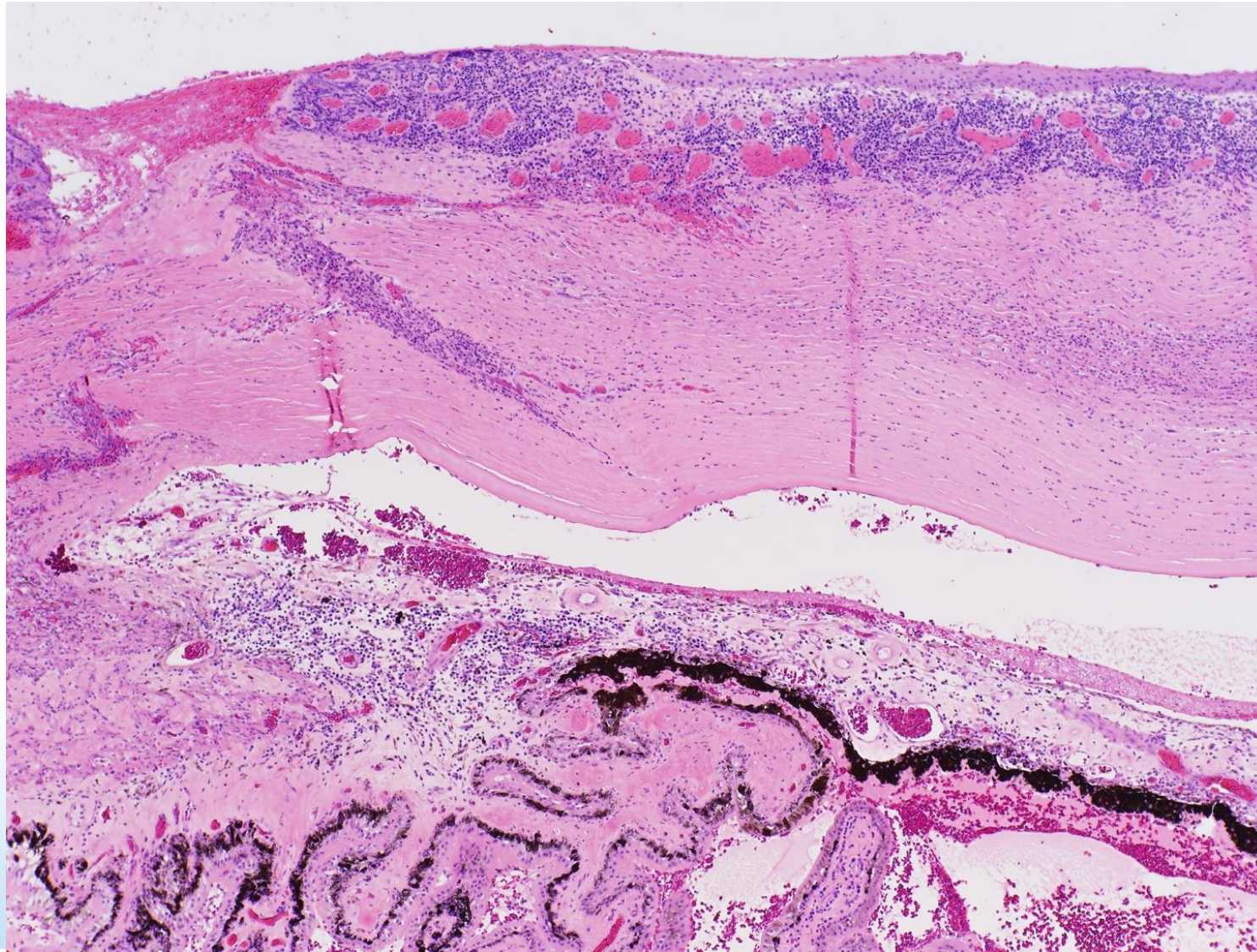


# Iris neovascularization // absolute glaucoma s/p trauma



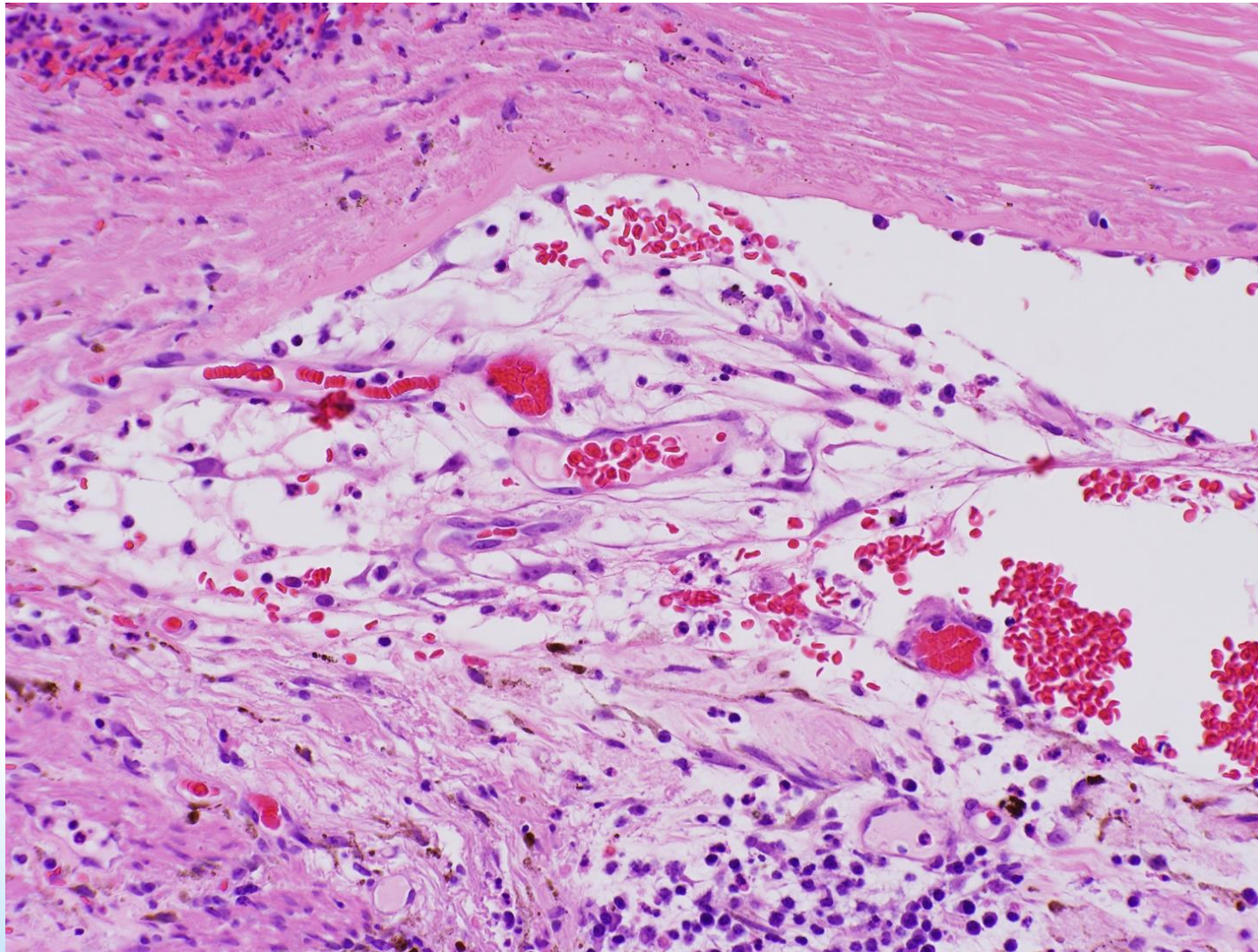


# Neovascular glaucoma

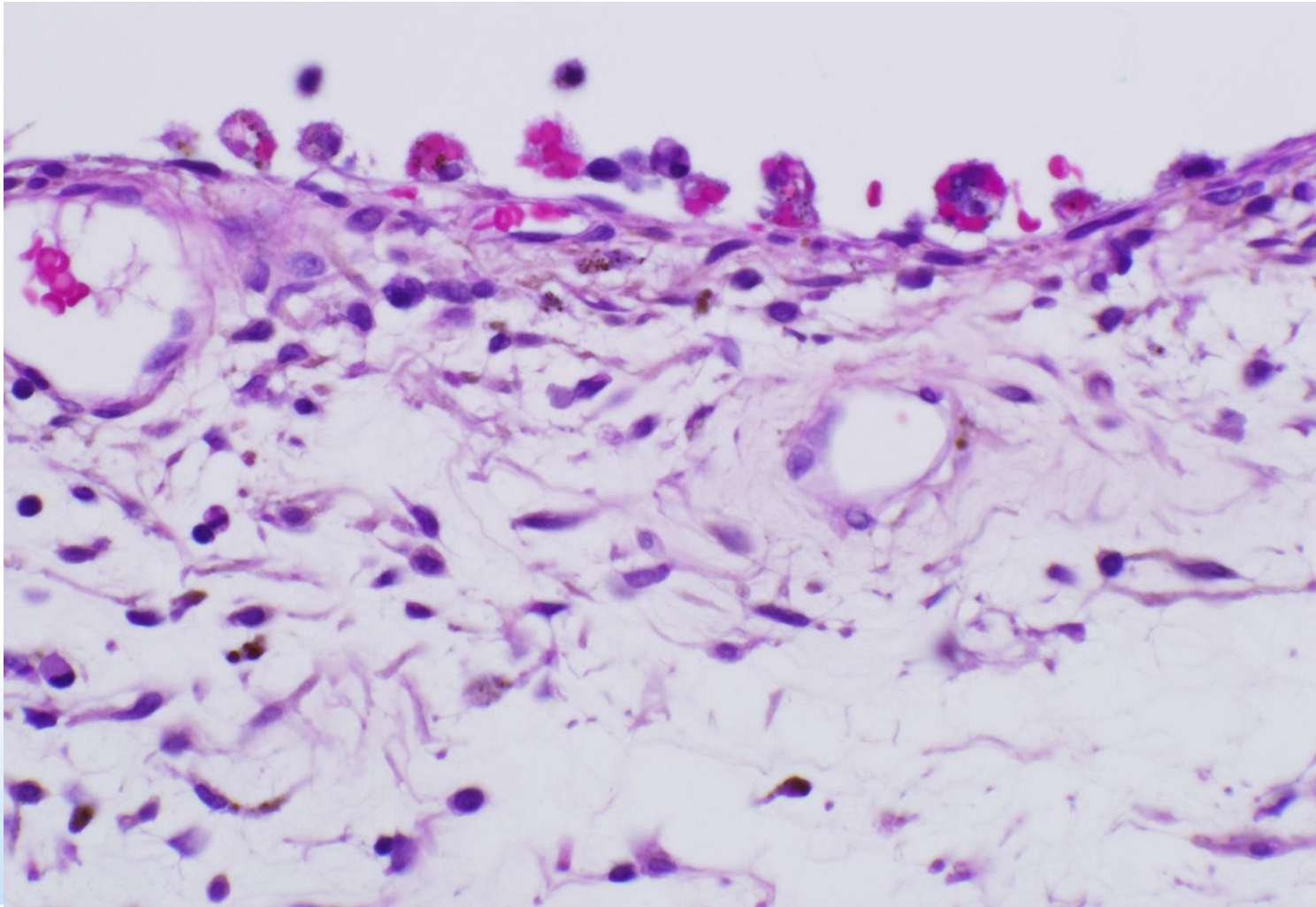




# Neovascular glaucoma

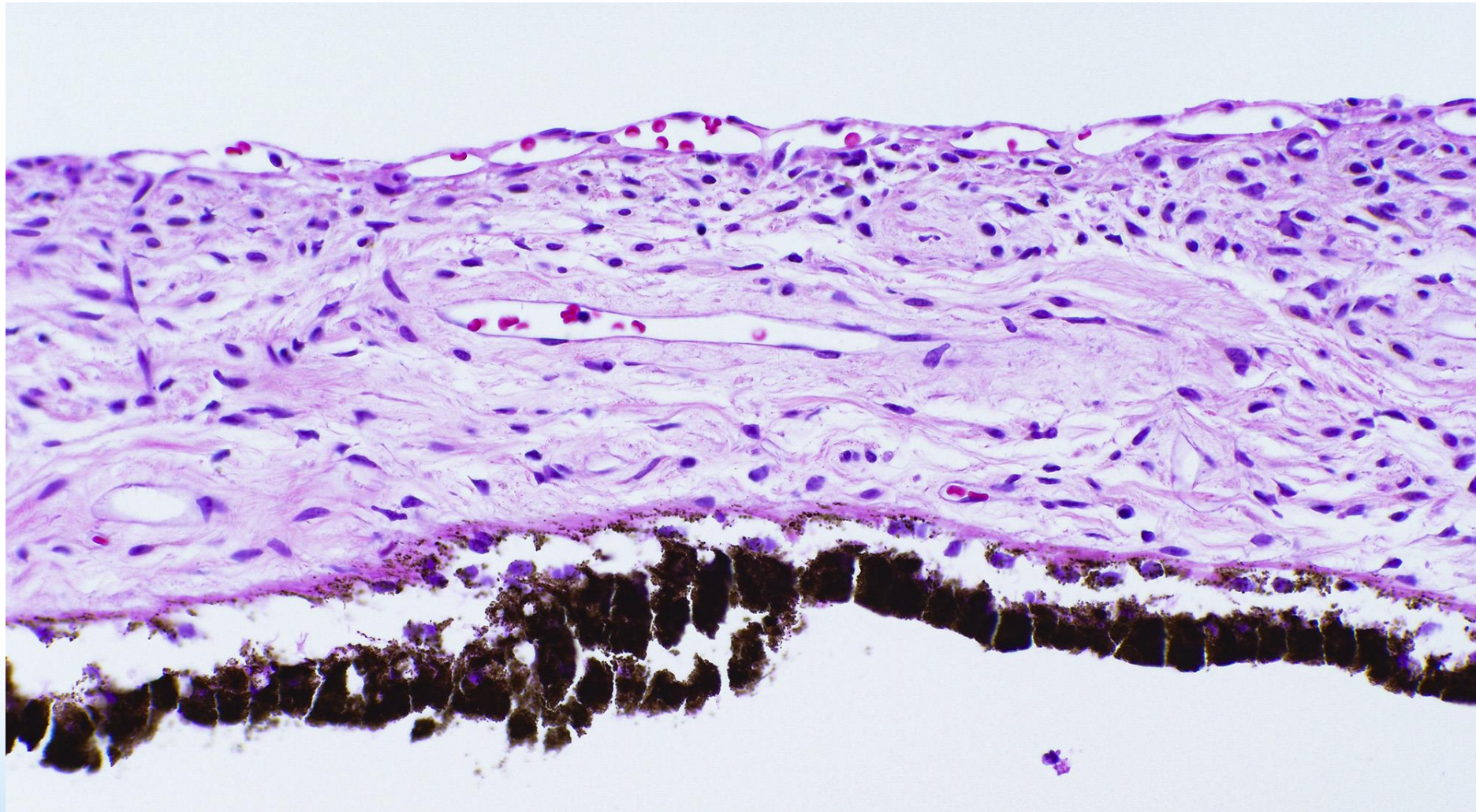


## Neovascular glaucoma: Iris neovascularization (NVI: anterior surface of iris)

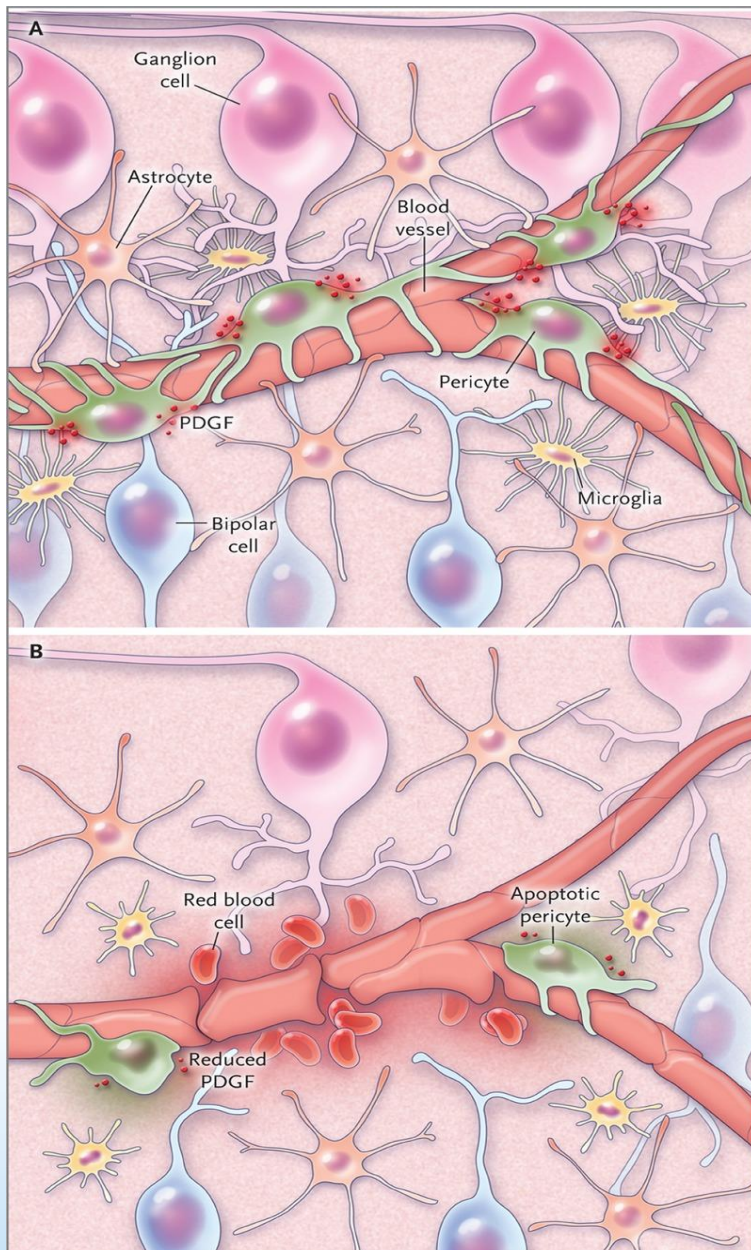




# Neovascular glaucoma: Iris neovascularization







## Disruption of the Neurovascular Unit of the Retina by Diabetes.

**A:** the neurovascular unit in the retina. Pericytes and glial cells, including astrocytes, promote formation of the blood–retina barrier in the vasculature, helping to create the environment for proper neural function. Microglial processes monitor the retinal environment.

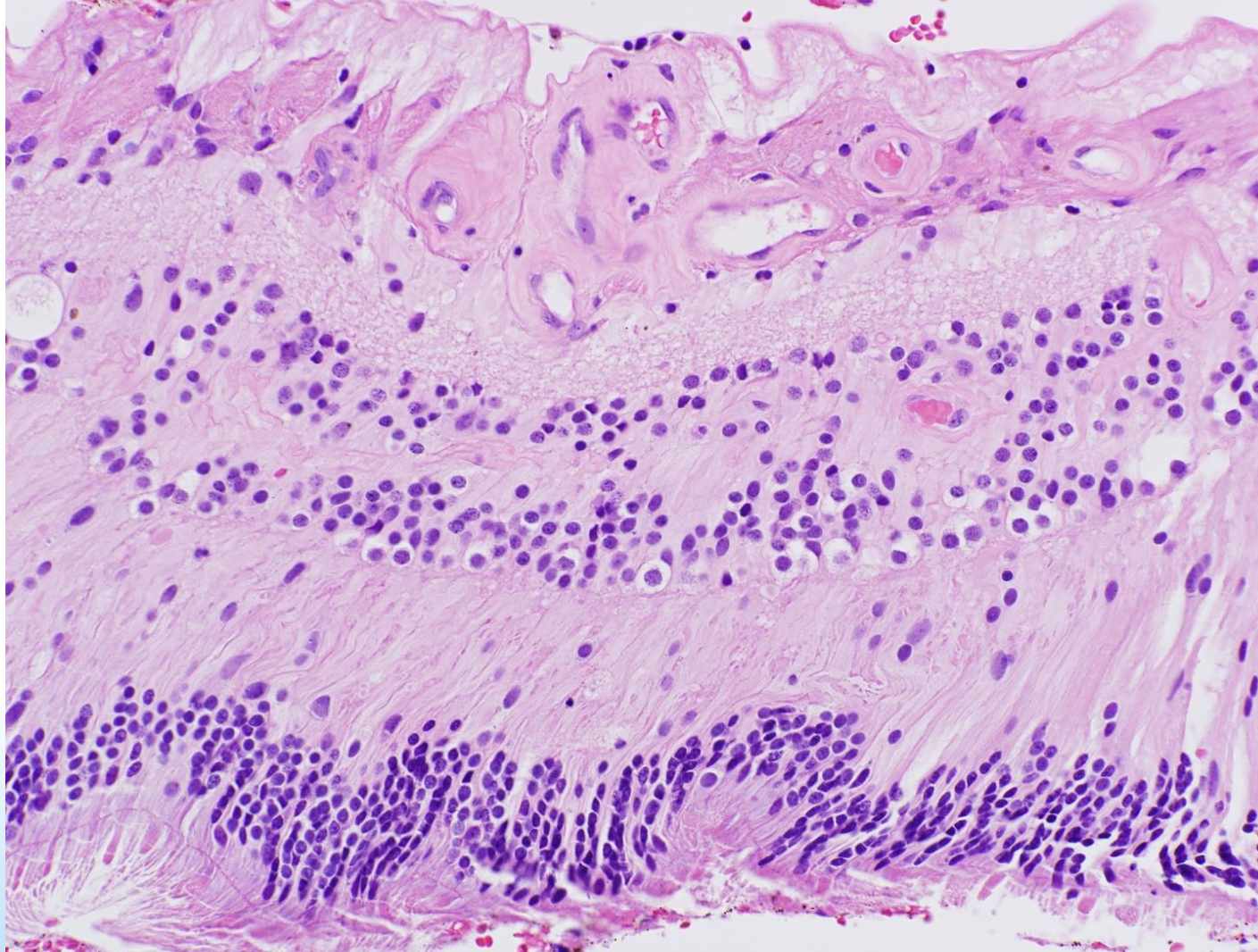
**B:** shows how normal cellular communication is altered in diabetes, with elevated VEGF from glial cells, combined with increased inflammatory cytokines, in part from activated microglia and adherent leukocytes (not shown), and the loss of platelet-derived growth factor (PDGF) signaling in pericytes, contributing to the **breakdown of the blood–retina barrier and, in some cases, to angiogenesis.**

Antonetti et al., N Engl J Med 2012; 366:1227-1239





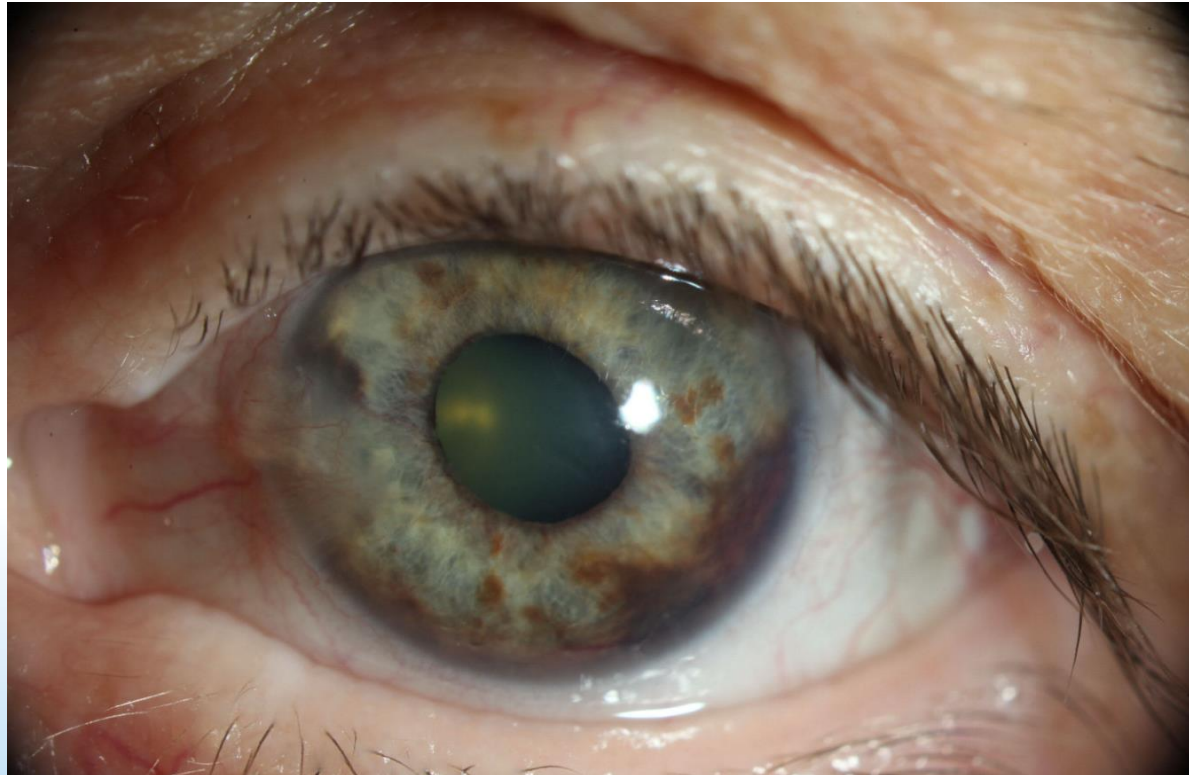
# Diabetes: Retinal neovascularization



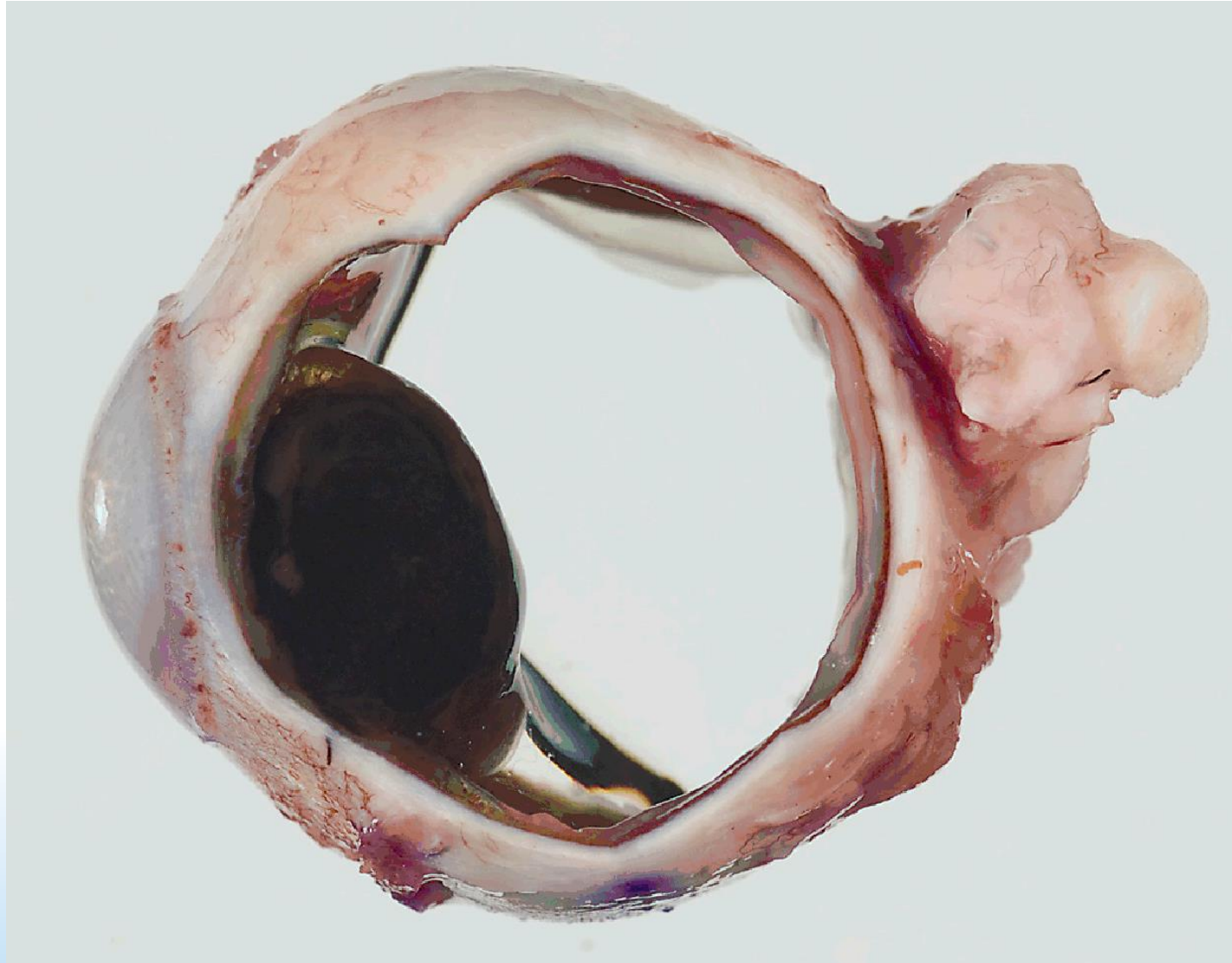


# Tumor-associated melanomalytic glaucoma

- Blockage of the anterior chamber angle / Schlemm canal
- Iris / Ciliary body melanoma



# Ciliary body melanoma

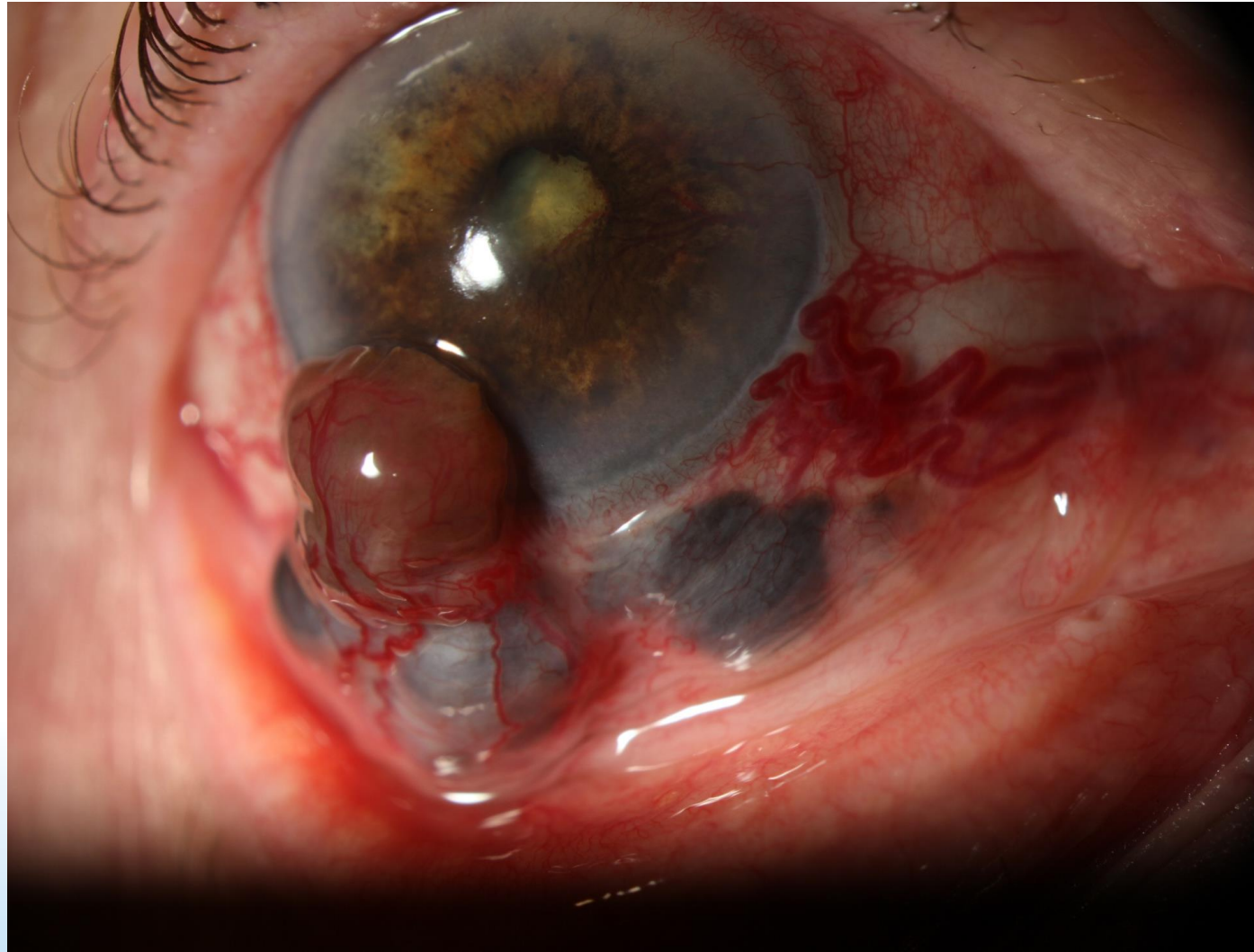


# Melanomalytic glaucoma

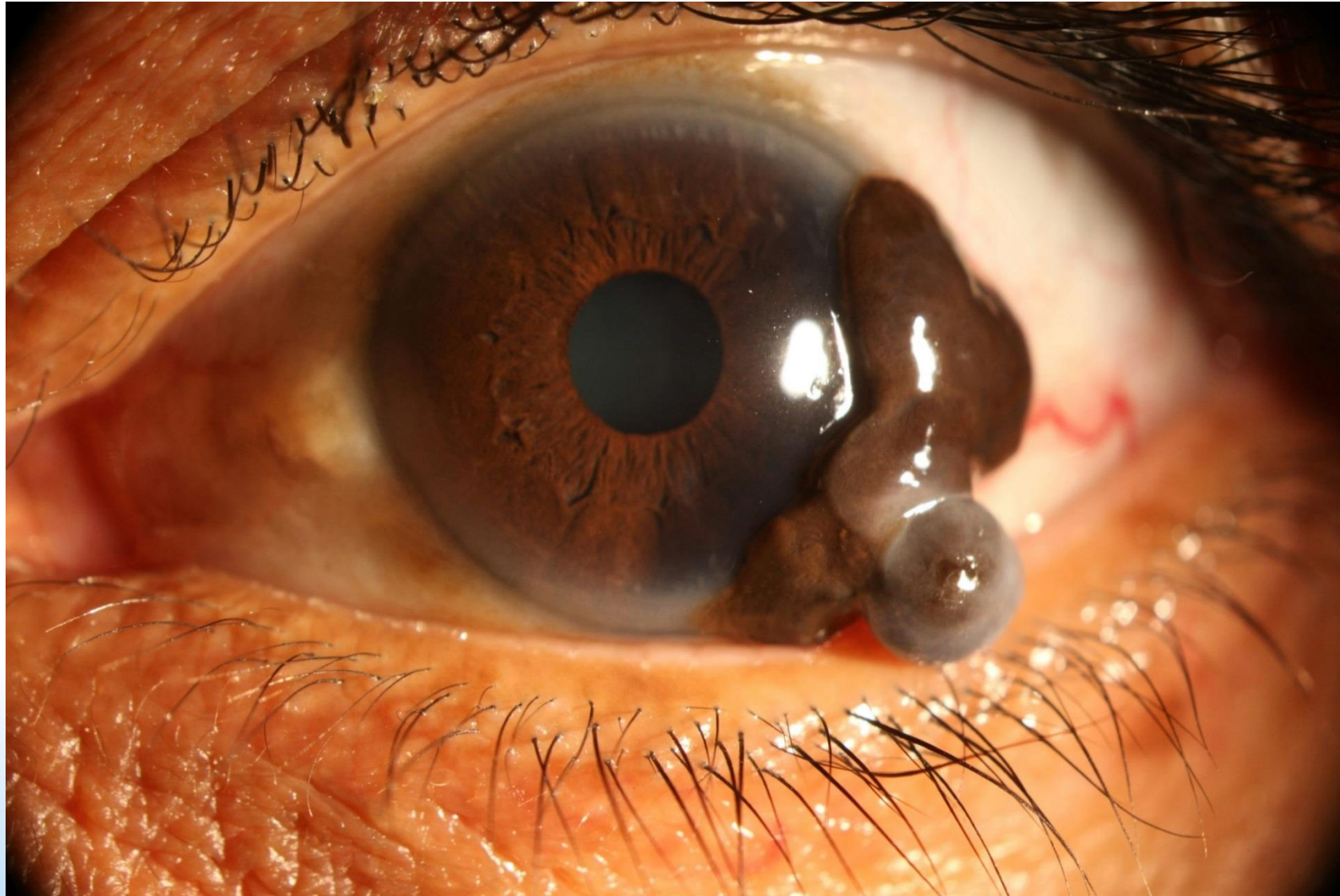




# Melanomalytic glaucoma

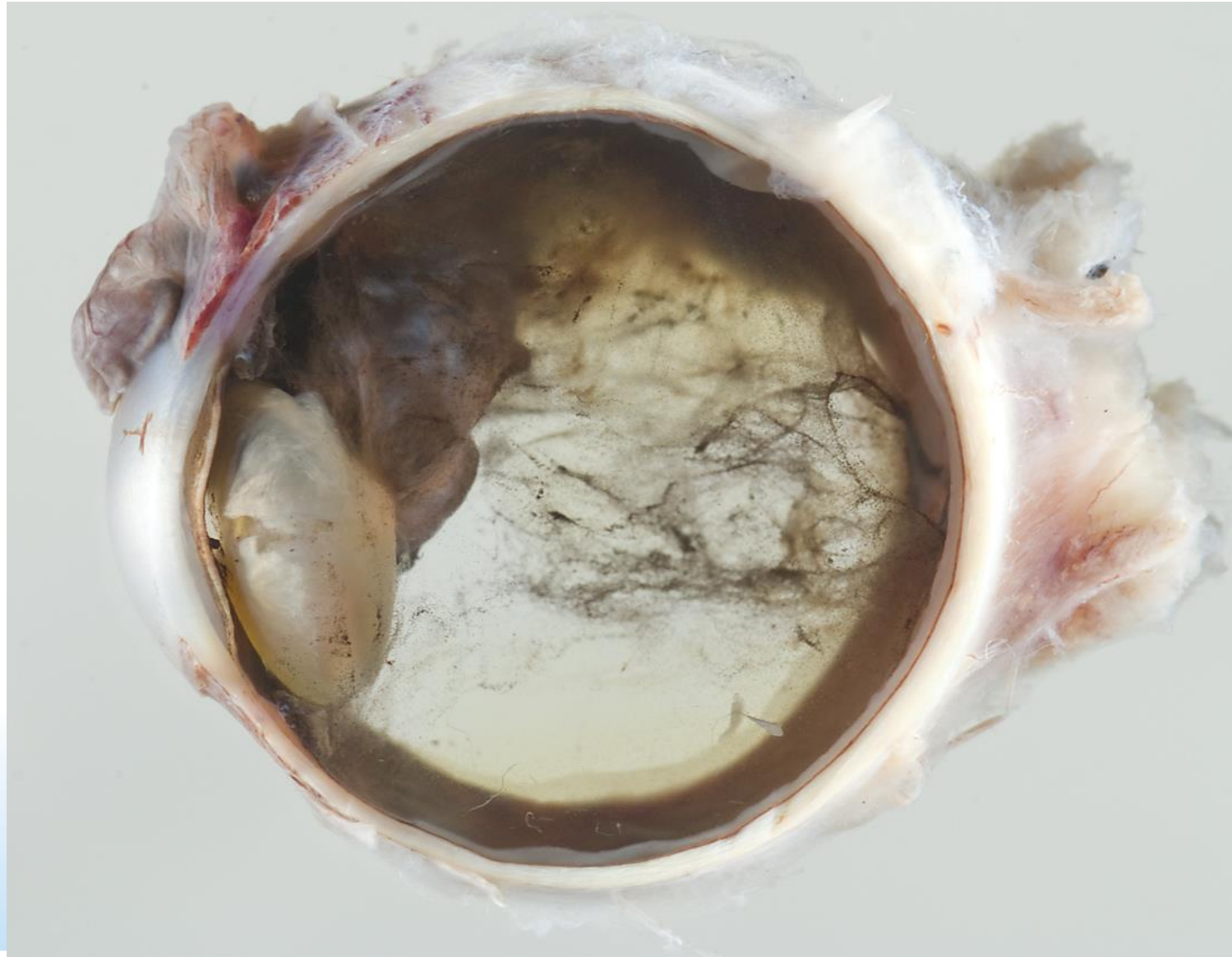


# Melanomalytic glaucoma





# Melanomalytic glaucoma



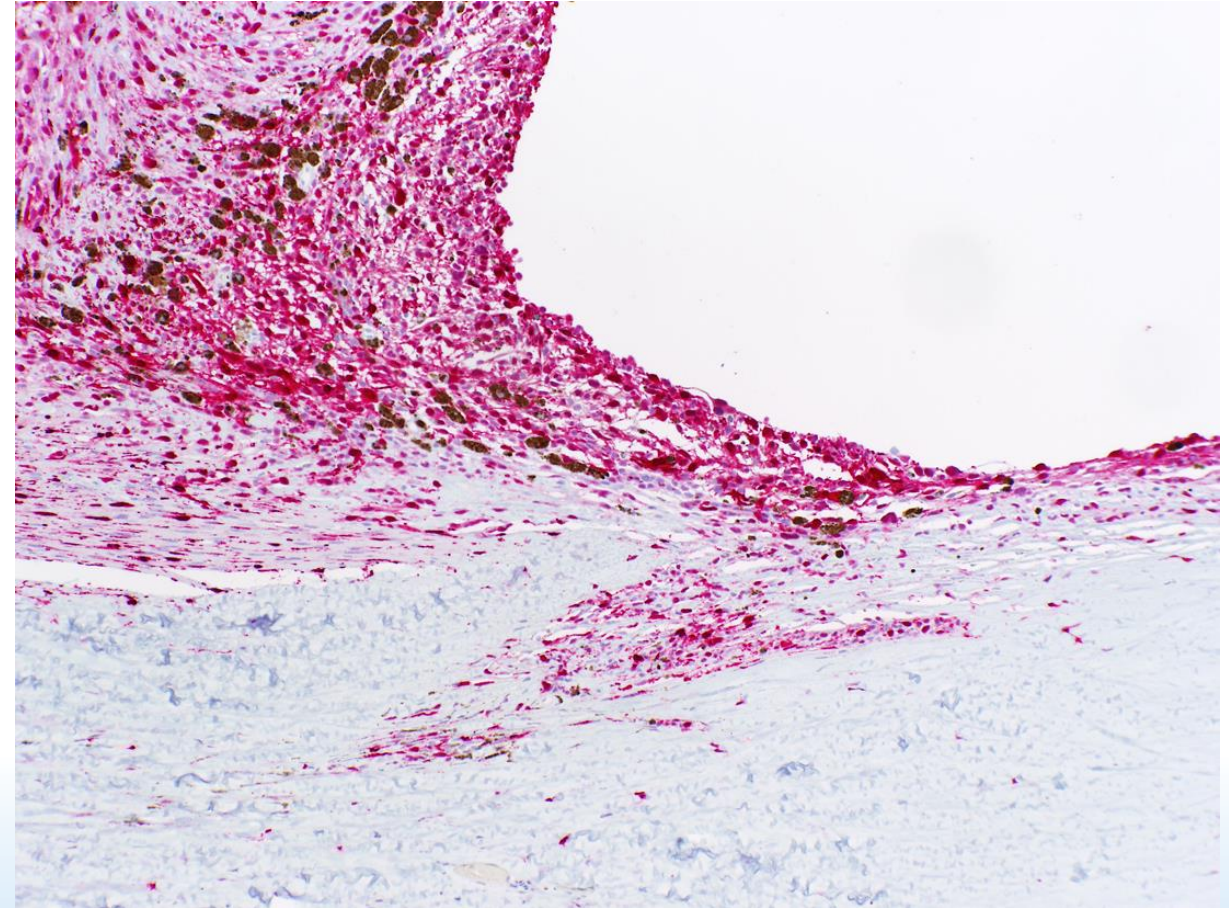
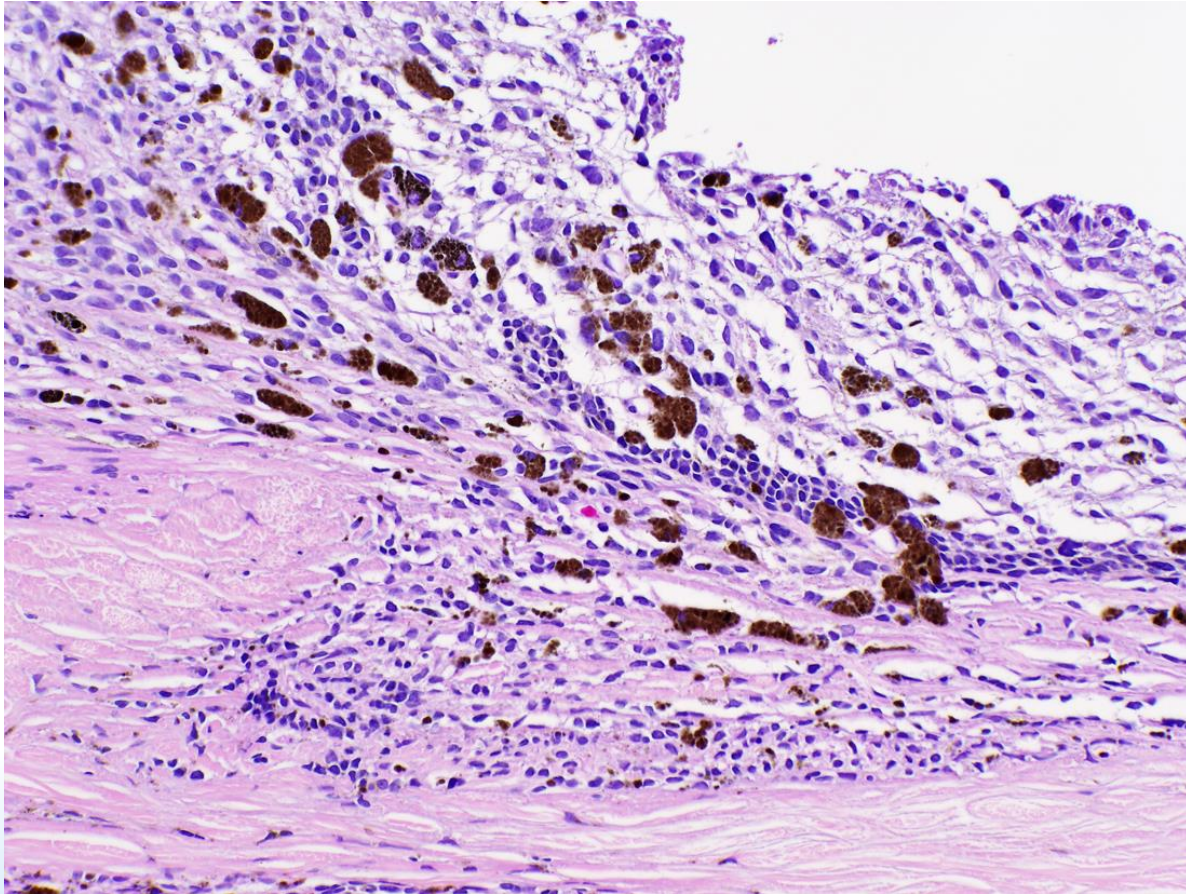


# Melanomalytic glaucoma





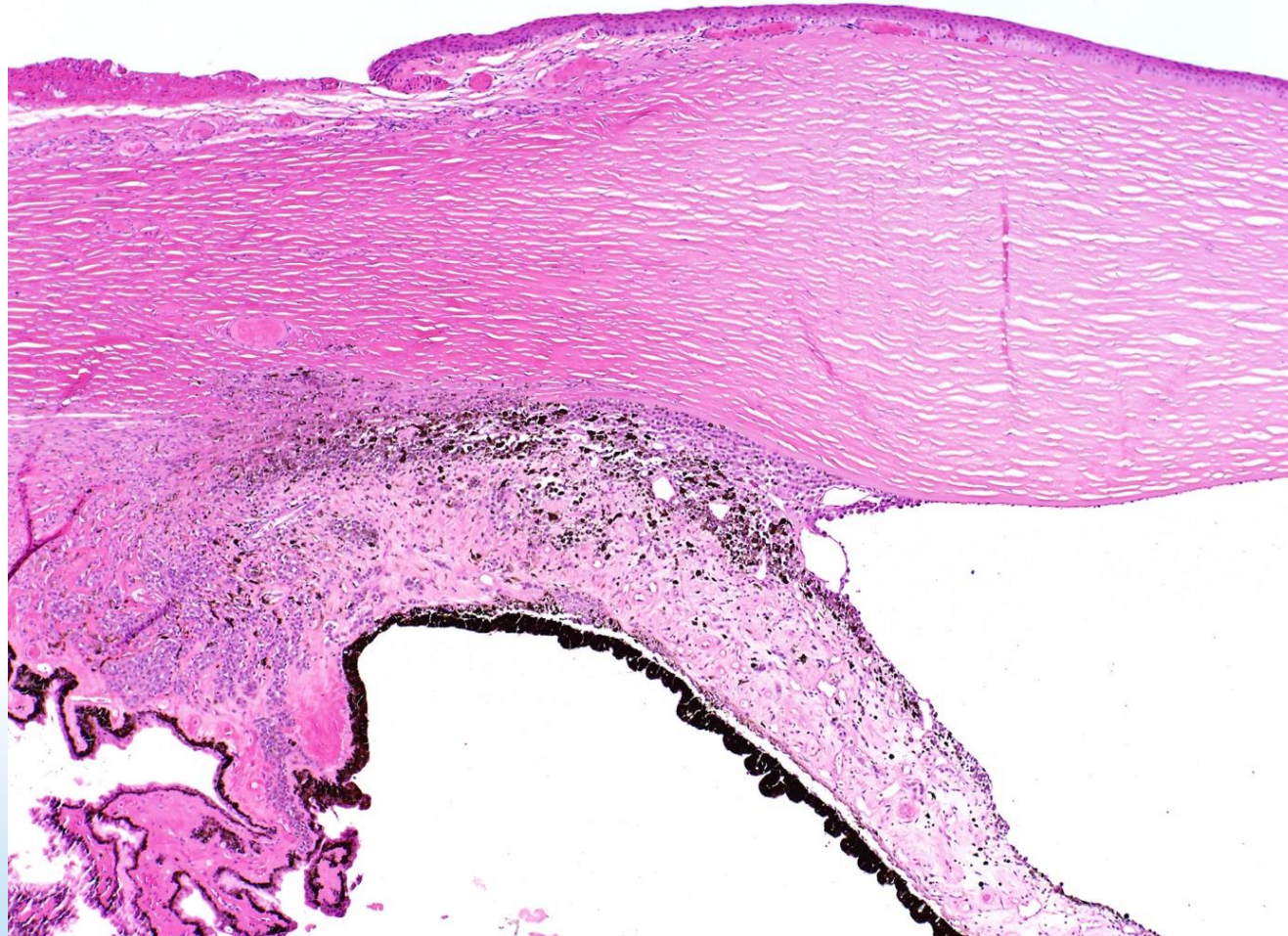
# Melanomalytic glaucoma MART1, tumor in Schlemm canal





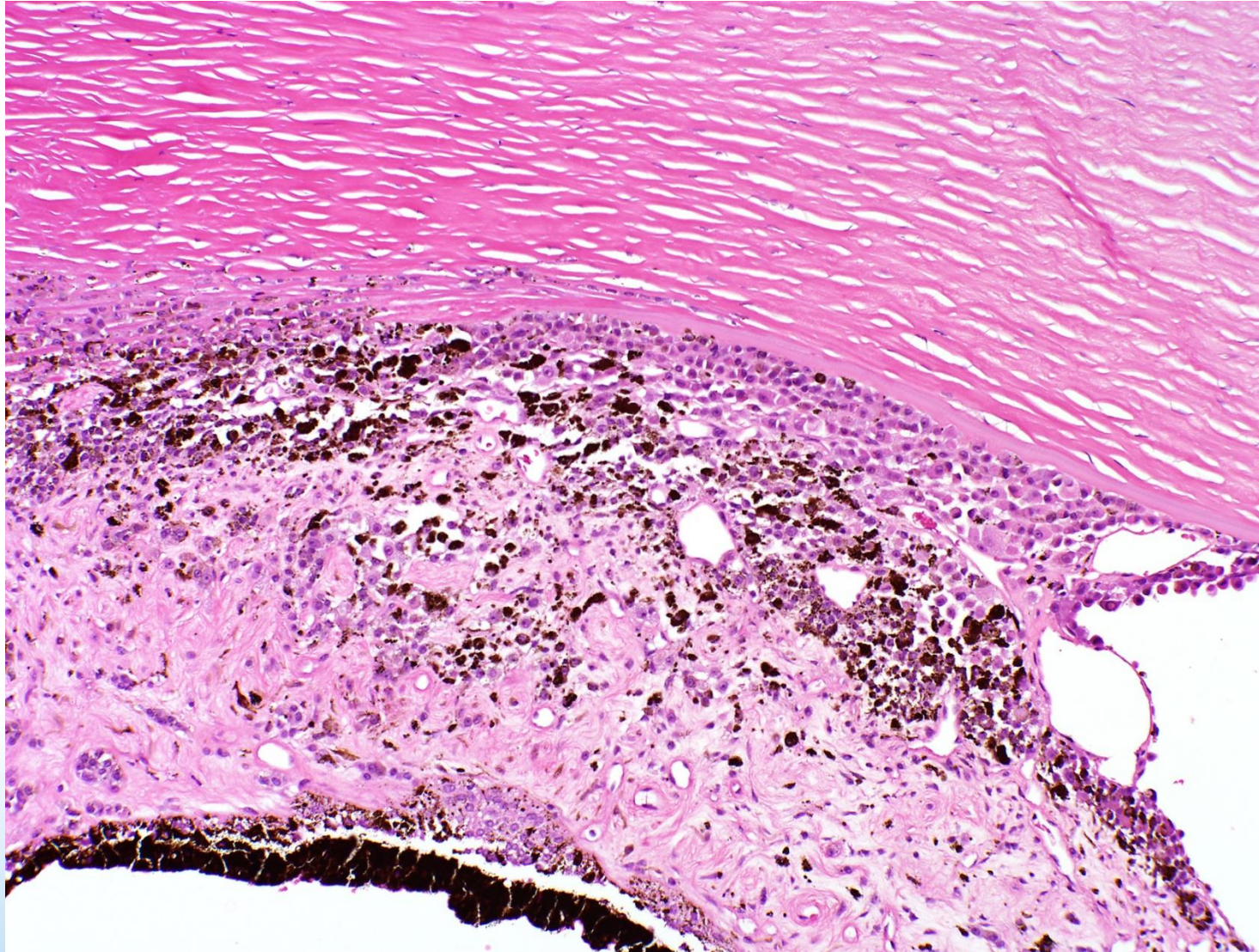
## Closed angle glaucoma (secondary)

Uveal ring melanoma involving iris root, with angle blockage



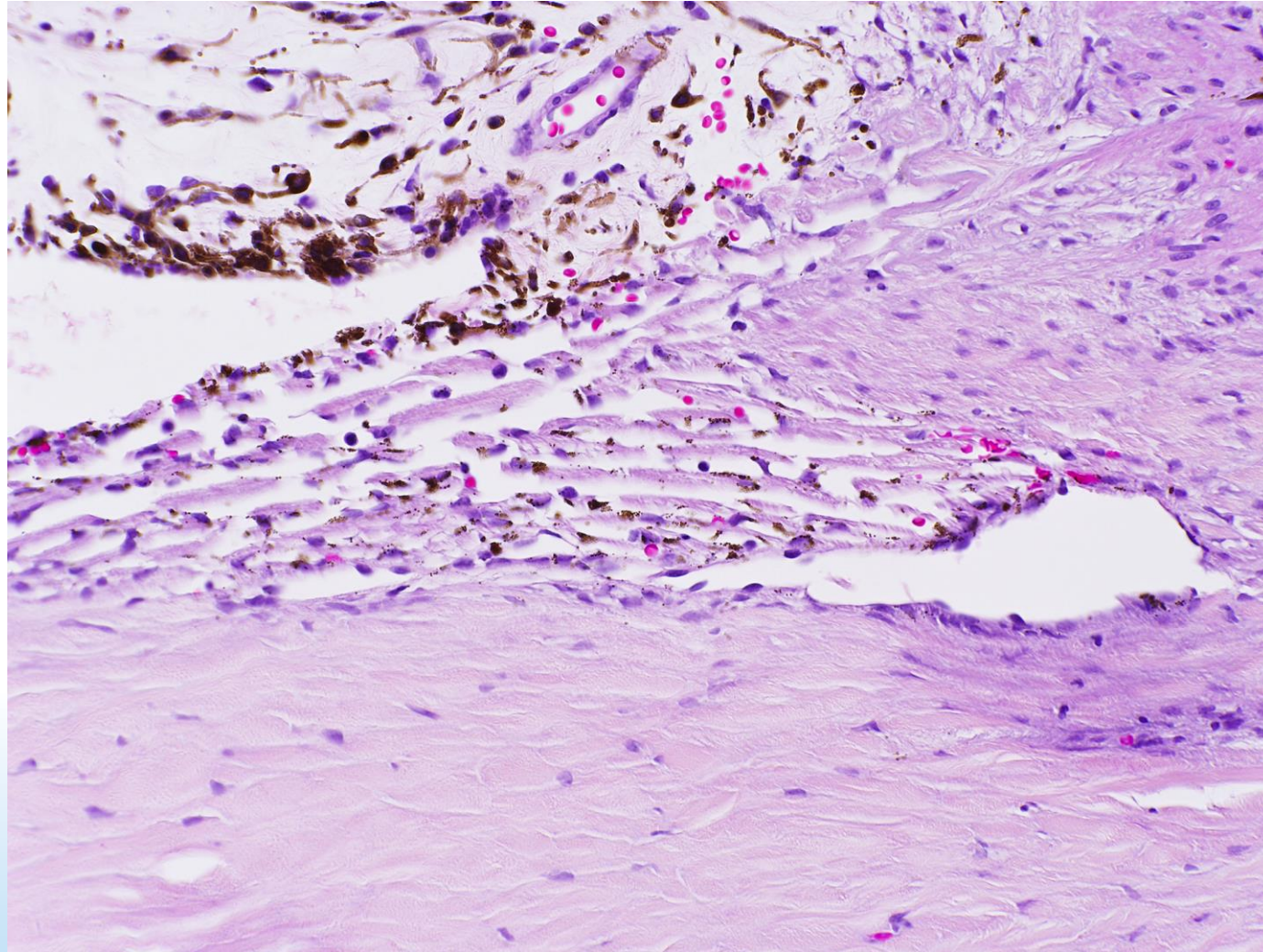


# Uveal ring melanoma

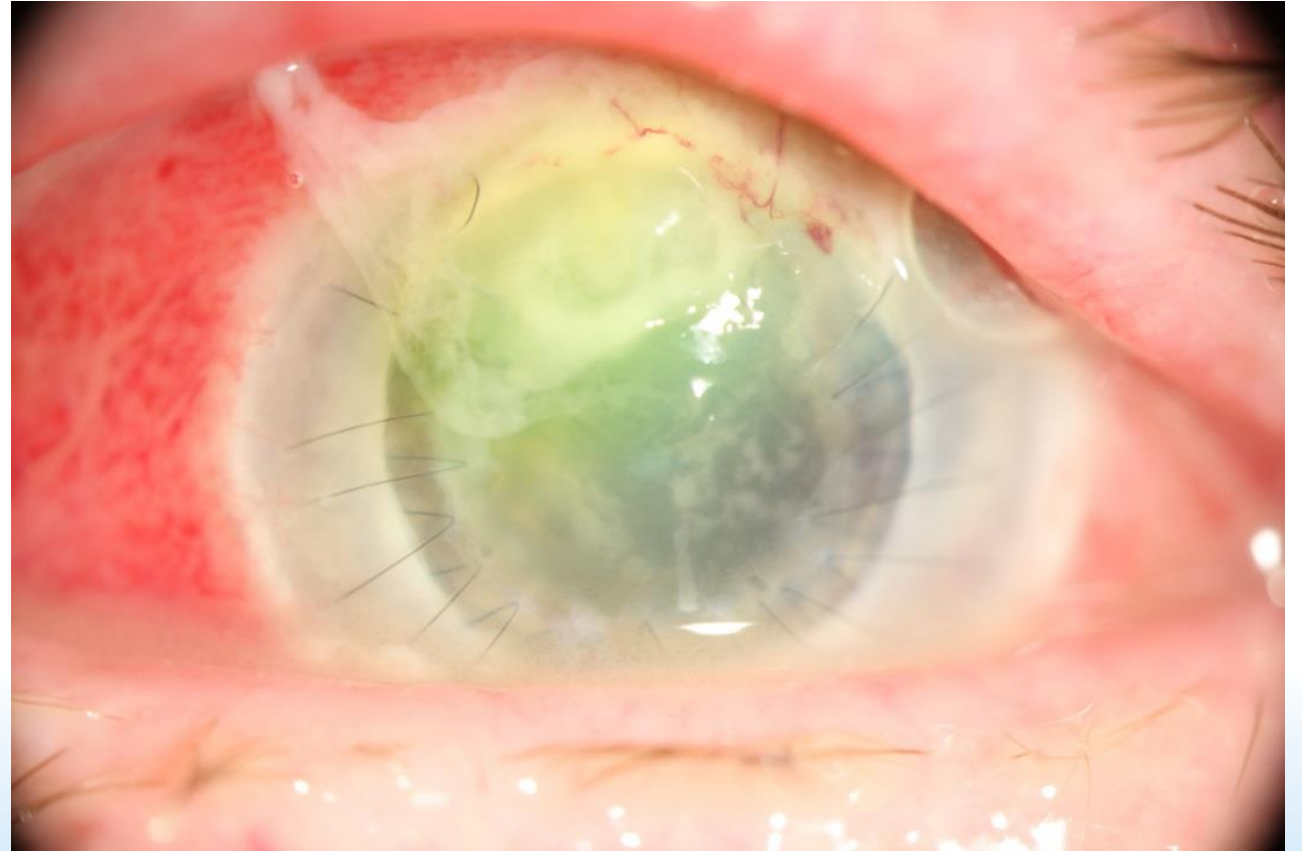




**Pigmentary glaucoma: Melanin pigment granules released from the iris pigment epithelium accumulate in the trabecular meshwork and interfere with aqueous outflow**



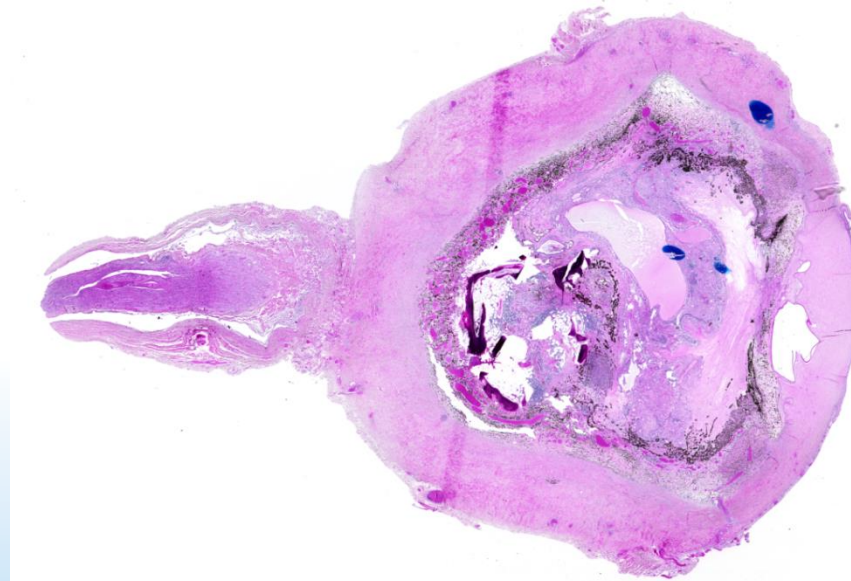
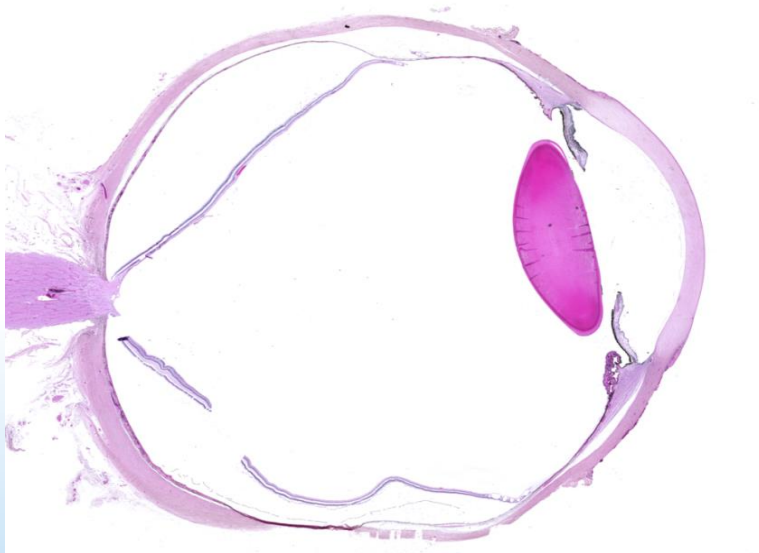
# Traumatic glaucoma, repair of penetrating corneal injury



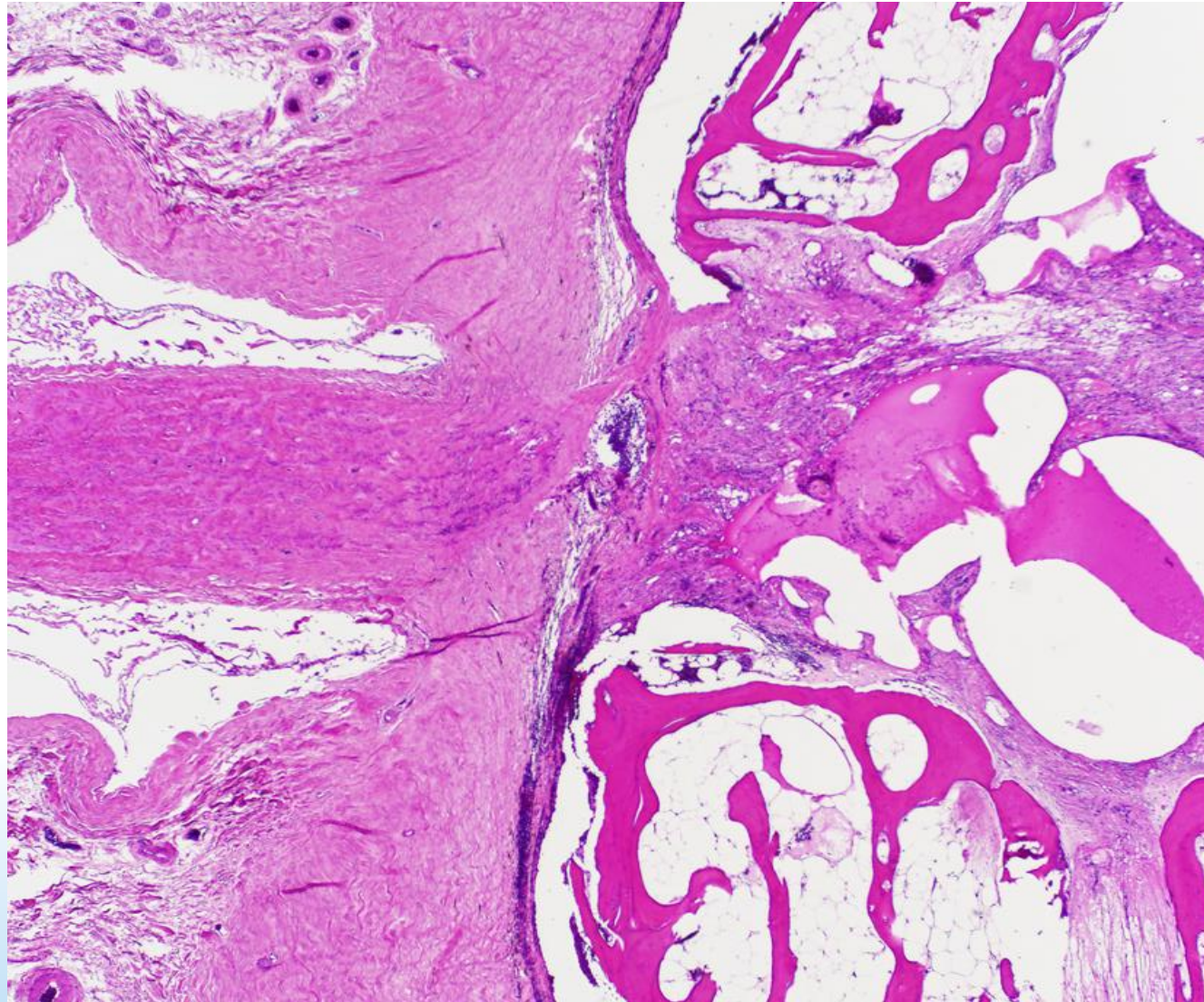


# Absolute glaucoma

- The final stage of glaucoma
- Permanent and complete vision loss
- May require enucleation for comfort
- Phthisis bulbi, optic nerve atrophy



## Phthisis bulbi with ossification and optic nerve atrophy



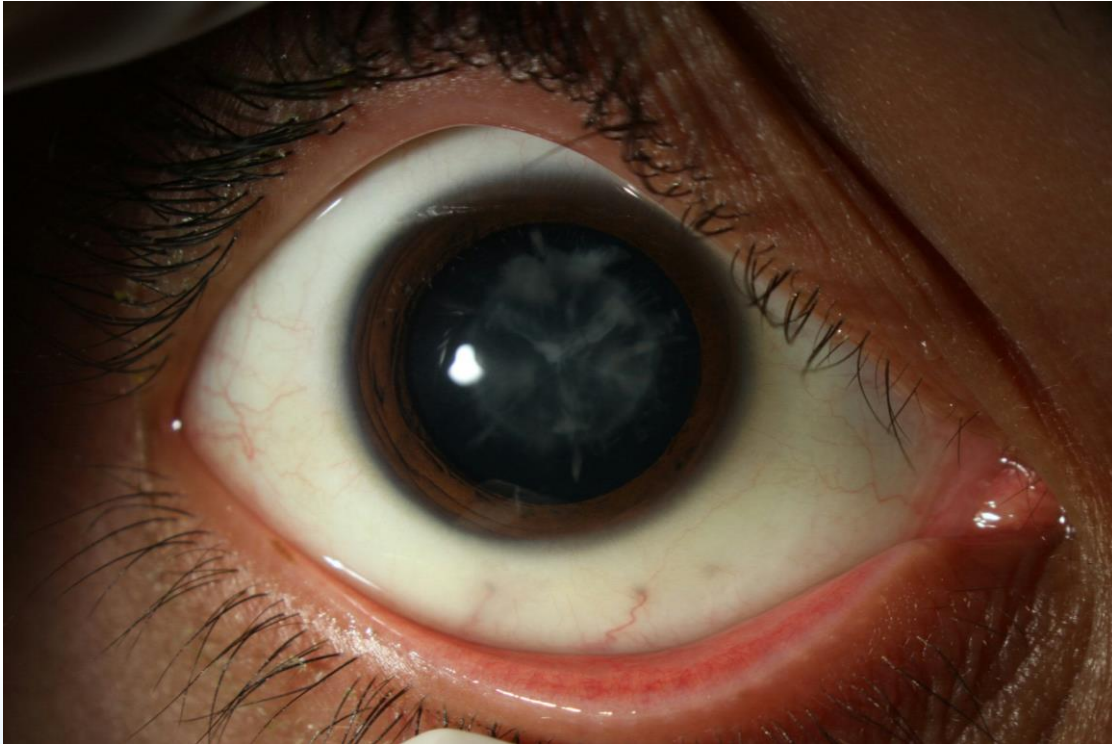
# Cataract

- **Commonest cause of blindness worldwide**
- **Opacity of the lens of the eye**

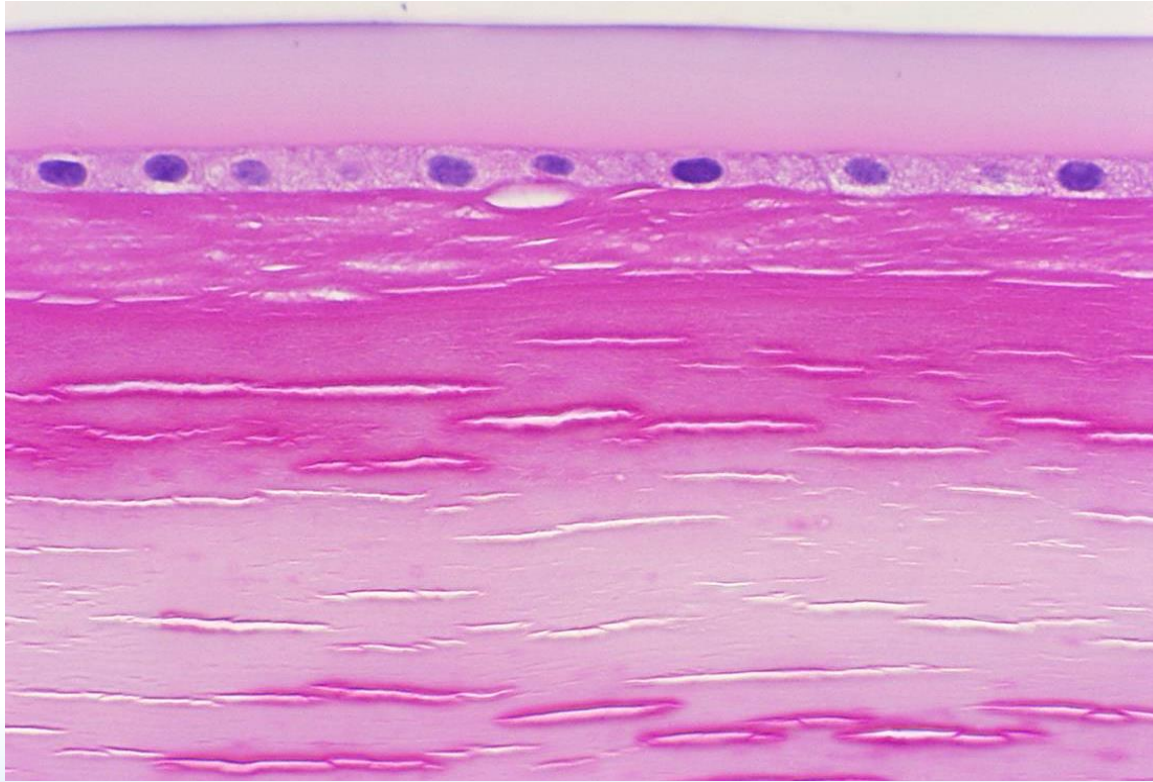




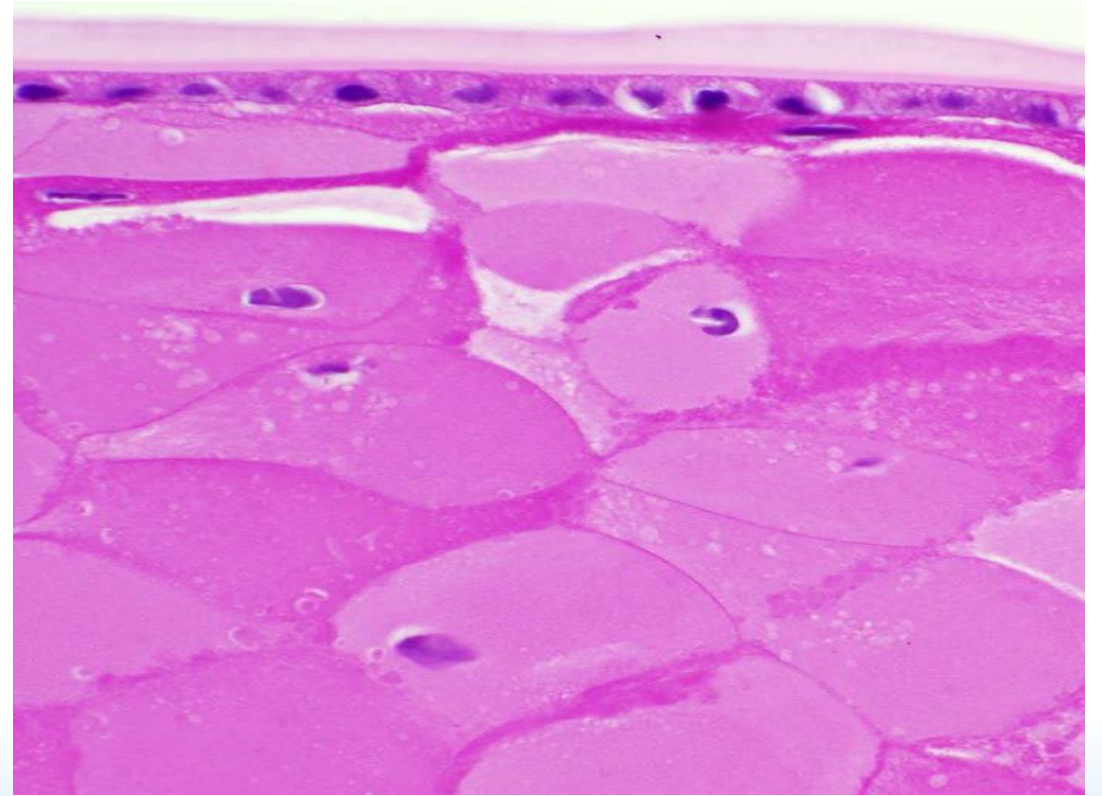
**Although cataracts are common, secondary phacolytic glaucoma (macrophage ingestion of degenerated lens fibers that leak through the capsule and block the trabecular meshwork) is rare.**



## Normal Lens Histology



## Cataract: Swollen (bladder // Wedl) cells



# Question 1

1. The basic pathological abnormality involving the retina in primary glaucoma is:
- a. Accumulation of sub-retinal pigment epithelium drusen
  - b. Degeneration of photoreceptors
  - c. Degeneration of retinal ganglion cells in the ganglion cell layer
  - d. Neovascularization and infarcts (cotton wool spots) in the nerve fiber layer
  - e. Vasculitis involving the peripheral nerve fiber layer





## Correct answer and rationale: C

- **Degeneration of retinal ganglion cells is the basic pathological abnormality involving the retina in primary glaucoma.**
- **Choice A (accumulation of sub-retinal pigment epithelium drusen) refers to age related macular degeneration.**
- **Choice D (neovascularization and infarcts (cotton wool spots) in the nerve fiber layer) refers to diabetic retinopathy.**
- **Photoreceptor degeneration (B) and vasculitis (E) typically do not occur in glaucoma.**



## Question 2

2. **Cupping of the optic disc is associated with:**

**A Primary optic atrophy**

**B Coloboma**

**C Pilocytic astrocytoma of the optic nerve**

**D Glaucomatous optic atrophy**

**E Acute retinal necrosis**



## Correct answer and rationale: D

- **Cupping of the optic disc distinguishes glaucomatous optic atrophy from primary optic atrophy. Cupping also suggests that elevated intraocular pressure (IOP) is a major risk factor in the pathogenesis of glaucomatous optic atrophy.**
- **Coloboma is a congenital defect that can involve optic nerve tissue.**
- **Pilocytic astrocytoma may result in expansion of the disc.**
- **Acute retinal necrosis does not result in cupping.**





## Question 3

<sup>3</sup> The anatomical nodular prominence of Schwalbe line demarcating the termination of Descemet membrane and the anterior border of the trabecular meshwork is known as:

- a. Scleral spur
- b. Schlemm canal
- c. Trabecular meshwork
- d. Pars plana
- e. Posterior embryotoxon



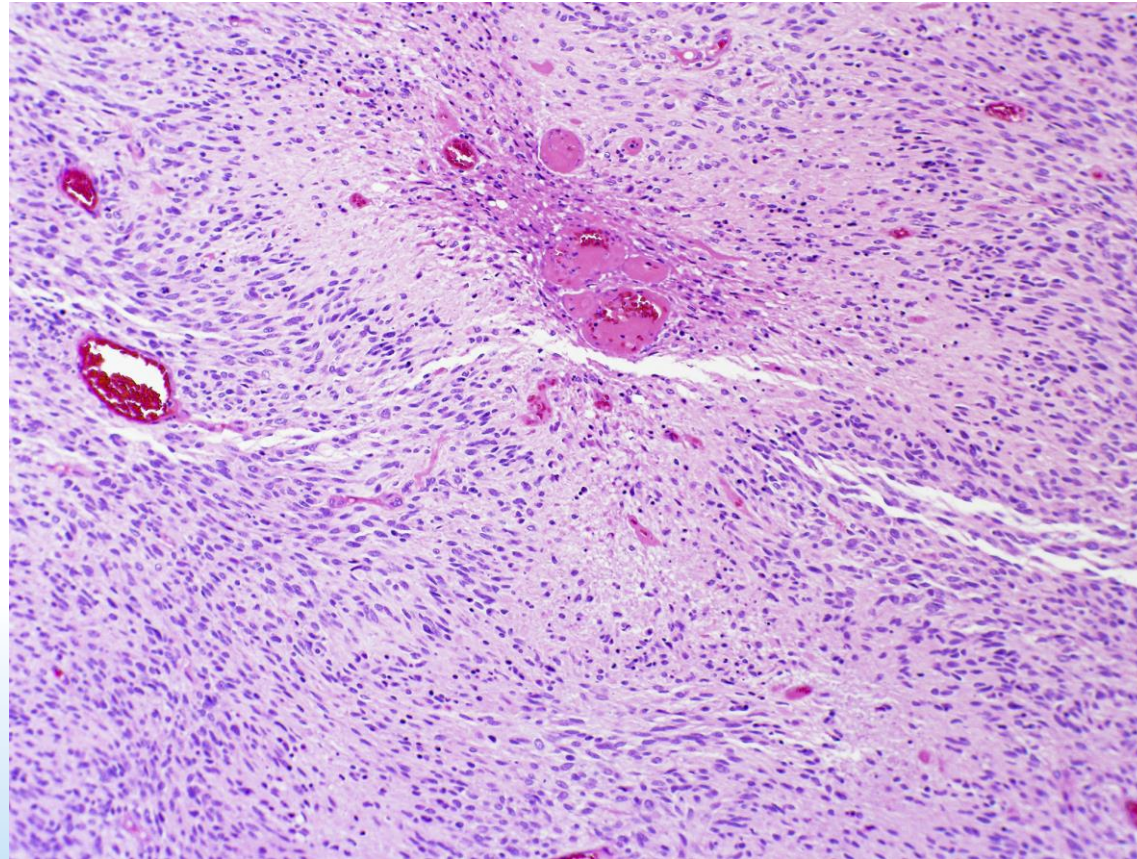
## Correct answer and rationale: E

- **Posterior embryotoxon is the anatomical nodular prominence of Schwalbe line demarcating the termination of Descemet membrane and the anterior border of the trabecular meshwork.**
- **The scleral spur is the posterior edge of the trabecular meshwork.**
- **Schlemm canal is the lymphatic-like vascular drainage site for aqueous humor.**
- **The pars plana merges the ciliary body with the retina.**



**The End, Thank You!!**

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**AMERICAN ASSOCIATION  
OF NEUROPATHOLOGISTS**

