Anatomical Differences Of Basic Eye Complaints

Whenever the Doctor tells a patient that they have an eye problem, the patient usually nods knowingly at them ,thanks the Doctor for their time and then as they leave the office, they turn to a technician and ask:
"So which is worse ?"
Back in my earlier days I would answer: I'd rather have cataracts than glaucoma, or I'd rather have conjunctivitis vrs EKC. And off the patient would go.

But the question rings out: Worse to WHO ?!
And can we really discuss their eye problem in turns of - I'd rather have this than that ?!

So… which is worse to have ?!

- Scleritis Versus Episcleritis
- Dry AMD versus Wet AMD
- OHTN versus POAG
- Fuchs vrs Bullous Keratopathy

Sclera Versus Episclera: Anatomy

a. **Episclera:**
1. covers the outer sclera
2. thin layer of elastic tissue.
3. numerous blood vessels that nourish sclera

b. **Sclera Proper:**
1. dense, fibrous tissue arranged in compact bundles of collagen fibers.
2. because of the arrangements of the bundles, the sclera appears dull, white
3. the arrangement of the bundles allows for the stress of movement of the EOMs and the changes in the IOP.
4. The middle, thickest layer is also called the stroma

Scleritis

a. **less common than episcleritis**
b. severe, **boring** ocular pain
c. scleral/episcleral vessels dilate
d. eye takes on a deep red or **bluish** color
e. gradual onset, photophobia, tearing and decrease vision, temple or jaw pain
f. scleral nodules
h. **need to treat immediately** to prevent secondary glaucoma, optic atrophy or cataracts and potentially blindness.Very aggressive.
h. women > men…ages 30 - 50

50% of cases are associated with systemic autoimmune diseases:
* rheumatoid arthritis (RA)
* systemic lupus erythematosus (SLE)
* ankylosing spondylitis
* relapsing polychondritis, Wegener granulomatosis, and giant cell arteritis.
* Syphilis

There are three types of scleritis:
- diffuse scleritis (the most common)
- nodular scleritis
- necrotizing scleritis (the most severe).

Episcleritis

- **common**, localized inflammation (*diffuse*) of the episclera
- benign… will heal in 1-2 weeks. *Self limiting*
- rarely progresses to scleritis
- cause unknown - but can be indicative of RA, Lupus, syphilis, TB, colitis or Crohn's Disease
- more commonly seen in young adults…women> men

Tests Your Doctor Might Do

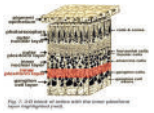
Scleritis may be differentiated from episcleritis by using phenylephrine or neosynephrine drops, which causes blanching of the blood vessels in episcleritis, but not in scleritis.
- *Other tests:*

Along with **visual acuity**, IOP check, slit lamp examination, and ophthalmoscopy, the doctor may order blood tests to rule out any potential systemic diseases.

Dry AMD vrs Wet AMD

Dry AMD is an early stage of the disease and may result from the thinning of the macular.
Dry AMD is diagnosed when yellowish-whitish "spots" (drusen) start to accumulate in and around the macula. Drusen are deposits/debris from deteriorating retinal tissue.
Gradual central vision loss will start to occur with Dry AMD, but usually is not as severe as Wet AMD symptoms.

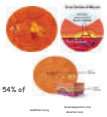
Layers Of The Retina



Macular Degeneration Facts

Age-related macular degeneration (AMD) is a leading cause of vision loss in Americans 60 years of age and older.

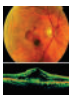
It is also the leading cause of legal blindness among white Americans age 40 and over, accounting for 54% of all such blindness.



1. Leading cause of new blindness in patients over age of 50 and increases in incidence each decade over 50 y.o.

2. Associations: race (Caucasian), sex (slight female preponderance), cigarette smokers and a family history

It's estimated that by 2020 there will be **2.95 million** people with advanced macular degeneration - *and* that there are 11 million people in the US with some form of macular degeneration that by 2050, will double to nearly 22 million!



- The "dry" form is more common. More than 85 % of all people with intermediate and advanced. AMD combined have the dry form
- The "wet" form is considered **advanced** AMD and leads to significantly more vision loss than the dry form.

Risk Factors For AMD

- **Obesity**. Studies suggest a link between obesity and the progression of early, intermediate to advanced AMD.
- **Race**. Whites are much more likely to lose vision from AMD than African Americans.
- **Family history.** Those with immediate family members who have AMD are at a higher risk of developing the disease.
- **Gender**. Women > men

Archives of Ophthalmology (June 2000).

Risk: Smoking

- Overall smoking **accounts for 32 % of AMD**. If you have AMD, stop smoking!
- Smoking increases the risk of macular degeneration about 3 times. Macular degeneration occurs 10 years earlier in smokers.
- Wet AMD progresses 4 times faster (a 400% increase) in smokers.
- **Passive smoking** is also harmful – if your partner smokes cigarettes, your body receives 25% of the smoke, so that is'equivalent' to you smoking 5 cigarettes a day.

Macular Degeneration: Nonexudative (Dry)

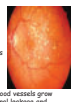
Atrophy and degeneration of the outer retina and choriocapillaris. "Atrophic" drusen is the most common thing seen. Gradual VA loss
Drusen = variable sized yellowish round spots located deep in the retina and scattered throughout the macula. They can enlarge, connect and calcify. NO TX

Dry AMD

The clinical findings in dry AMD are **drusen** and **pigmentary changes in the RPE layer**
- Drusen are round yellow lesions under the retina. Small drusen are called "hard" drusen and larger drusen are called "soft" drusen.

There **deposits within the Bruchs membrane** that can cause elevation in Bruchs membrane. Drusen generally do not cause a significant change in vision."

- Dry ARMD can progress to "wet" in approx. 4% of patients.
- Occasionally wet ARMD develops without any previous dry ARMD, although usually there is an area of retinal damage that triggers the process.
- **Wet ARMD** begins when new blood vessels grow in the macular area causing retinal leakage and swelling. The condition progresses to cause a scar in the macular area. If the scar is small, sight is reasonable; if large, the sight can be very poor.



Macular Degeneration: Exudative ("Wet")

Impairment of Bruchs membrane, the barrier between the RPE and the Choriocapillaris, can cause complications. Serous fluid or blood from the choroid can leak causing a "netting" of the RPE over the macular area.
This can cause the fovea to detach!

Wet macular degeneration gets its name from leaking blood vessels in the retina.

- Abnormal blood vessels that grow through Bruchs membrane and into the sub- retinal space.
- The choroid contains most of the eye's blood vessels. These new, fragile and abnormal blood vessels grow up through the retinal layers.

Choroidal neovascular membranes (CNV) can penetrate through a break in Bruchs membrane causing a serous detachment of the RPE. This can cause the formation of a disciform (macular) scar.
Accounts for 90% of severe vision loss due to ARMD.
Onset is often rapid and with a complaint of **metamorphopsia.**

Progesia is worse if a "**net**" is formed (sub-retinal neovascular membrane).

This can cause "**metamorphopsia**" = wavy lines in vision.

Nets can regress or can become severe and lead to loss of vision. If a net is present and the fovea is not involved, it can be lasered.

Hallmarks of CNVM

- An essential element of exudative AMD is CNVM.
- Eyes present with:
  - * sub-retinal fluid
  - * retinal pigment epithelial detachments
  - *sub-retinal hemorrhage
  - * sub-retinal lipid deposits
  - * RPE hypertrophy and/or RPE atrophy

The CNV itself can be seen as yellow/green sub-retinal discoloration that is sometimed surrounded by a pigment ring.

POAG vrs OHTN

POAG is a chronic disease that can be hereditary. It is generally bilateral, but not always symmetrical.
OAG where the IOP is 21 mm Hg or below, is known as Normal Tension Glaucoma (NTG) or "Low Tension Glaucoma".
Ocular hypertension (OHTN) is IOP higher than normal without Optic Nerve damage or visual field changes. Not all people with ocular hypertension will go on to develop glaucoma.

People At Risk

- African Americans have a 6-8% higher risk
- People over 60 y.o.
- POAG is/can be hereditary
- Asians run an increased risk for angle closure
- Past blunt trauma eye injuries may cause *secondary glaucoma*
- Patients that are very nearsighted
- Diabetics
- Patients with high blood pressure
- *Thin* corneal thickness ≤ 0.555 mm

So… *What is Glaucoma ?*

Glaucoma is a disease that can be categorized as a *tried disease process.*
This means that (3) "things" must occur before your eye doctor considers a diagnosis of glaucoma:
- * *Increased intraocular pressure*
- * Visual field changes
- * Optic nerve changes

If you think of the eyeball as a sink….

In half of the patients with glaucoma, the "faucet" (ciliary bodies) are on full blast. The drain is normal but the drain cannot handle the amount of fluid being created…
or…
the faucet is at regular flow but the "drain" (trabecular meshwork) is partially clogged.
The drain doesn't drain the fluid adequately causing the water to rise in the sink.

Aqueous Production: the drain versus the sink !

Trabecular meshwork is how the aqueous flows out of the eye. "The drain"

Ciliary processes & bodies makes aqueous - "the faucet"

How Is Aqueous Made ?

Aqueous is made by the ciliary body & processes.

It is a clear liquid that fills the **anterior** and **posterior chambers.**

What is Aqueous?

The composition of aqueous is similar to plasma. **IF** you were to increase the **protein** content - it would begin to take on more *plasmoid* features.

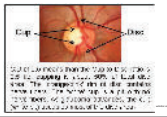
Aqueous nourishes the eye.

Clinical Assessment Of Glaucoma

a. Applanation
b. Gonioscopy
c. Optic Nerve evaluation:
- * cupping precedes VF in 80% of patients
- * asymmetry of C/D
- * vertical diameter ≥ than horizontal diameter

- * notching of rim
- * sector pallor and/or nerve fiber layer loss
- * splinter hemorrhages near rim
- * C:D ratio ≥ than 0.6

Optic Disc Cup to Disc Ratio: C/D



Pachymetry: Corneal Thickness

Eye with cornea thickness *less than 555* (thin corneas) were found to have a 3x **higher** risk of developing glaucoma and therefore, CCT under 555 should be seen as a ***potential risk factor*** for the development of glaucoma. So.. the 540 *normal* CCT **also** falls into the potential risk category !

** Thin corneas and high IOP = +++ risk ***

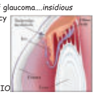
Corneal Thickness Affects IOP Accuracy

Goldmann TA is accurate with average corneal thickness—540 to 570 microns

- **Reads low** if cornea is **thinner** than average: approximately 500 microns or less
- **Reads high** if cornea is **thicker** than average: approximately 600 microns or more

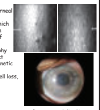
Primary Open Angle Glaucoma

* Most common type of glaucoma….*insidious*
* Strong family tendency
* Increased incidence with : ↑myopia, DM, people of color
* Degeneration of the trabecular meshwork
* Decrease in aqueous drainage leading to ↑ IO



Fuchs versus Bullous Keratopathy

Bullous keratopathy occurs when epithelial bullae appear due to corneal endothelial disease.
BK is caused by corneal edema which causes failure of the endothelium to maintain a dehydrated state of the cornea. Frequently, it is ***due to*** Fuchs endothelial dystrophy or endothelial trauma (i.e cataract surgery). Fuchs dystrophy is a genetic disorder that causes bilateral, progressive corneal endothelial cell loss, and sometimes leads to bullous keratopathy by age 50 +.



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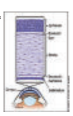
Layers of the Cornea

A. Epithelium:
- a. Nonkeratinized stratified squamous
- b. when covered with tears acts as a convex mirror

B. Basement membrane

C. Bowman's membrane:
- a. transparent, *hyaline* membrane (hyaline = "like glass ")
- b. although the epithelium easily pulls away from Bowman's, the stroma is firmly adherent to Bowman's.
- c. IF Bowman's is injured…it does ***not*** regenerate. The epithelium will fill in the missing areas.

D. Stroma:
- makes up 90% of the cornea
- made primarily of collagen fibrils

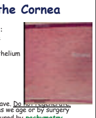


Layers of the Cornea

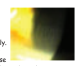
E. Descemets Membrane:
- a. clear, *elastic* membrane
- b. hyaline basement membrane of the endothelium

F. Endothelium:
- a. single layer of cells
- b. inner surface is bathed with *aqueous*
- c. the cells you are born with are the cells you have. Do not regenerate.

We will lose these cells as we age or by surgery (cataract surgery). Measured by ***pachymetry.***



Cornea in Disease

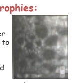


- a. **Avascular**
- b. Bowman's membrane offers little resistance to disease and can be injured very easily.
- c. Descemets is very resistant
- d. Many disease processes cause the cornea to become vascularized. IF the cornea has been vascularized , the vessels will remain throughout life. The may empty of blood (" ghost vessels ") but you can usually still them.
- e. Cornea will desensitize with aging

Definitions

1. **Dysgenesis**: developmental disorder that results in **congenital** malformations
2. **Dystrophy**: **Inherited**, bilateral disorder. Usually appear in the first or second decade and progress slowly
3. **Degeneration**: Unilateral or bilateral. Commonly because of **aging** or previous ocular disease.

Endothelial Dystrophies: Fuchs



Endothelium is reduced in number which causes the remaining cells to swell or thicken. The loss of endothelial cells can also allow abnormally shaped growths called **guttata** to form.
The cell changes may cause the cornea to become cloudy and swollen. A progressive disease that usually occurs in people after age 40 -50 F> M. Studies show that it is an inherited condition.

Bullous Keratopathy

Occurs because small vesicles (bullae) are formed in the cornea due to endothelial dysfunction. When affected by any number of reasons, such as Fuchs or trauma, endothelial cells will become damaged and/or die. The endothelial cells do not undergo mitotic cell division, so once cells are lost, permanent function loss occurs to the "pumps". When the "pumps" start to fail, fluid moves into the stroma and epithelium. The excess fluid causes swelling of the cornea to begin. As the fluid accumulates, blister - like bullae occur and can rupture, releasing *their* fluid into the cornea as well. This causes a decrease in vision and creates extreme pain.

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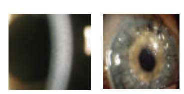

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