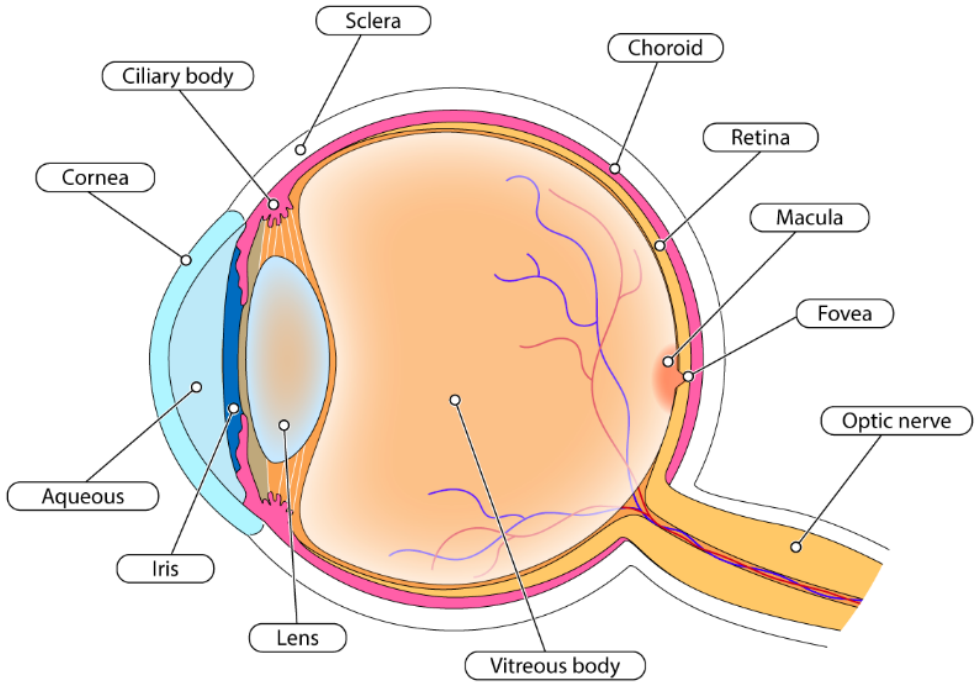
PREMEDSKL'S MEDICAL B PREP

Ophthalmology



This ophthalmology module was created by Dr. Sanjay Sharma, a professor of ophthalmology at Queen's University who is an award-winning medical researcher and eye surgeon. Dr. Sharma is also the host of the Medical B - a case-based medical competition for high school students interested in pursuing a career in medicine.

This module contains:

- Important information about the anatomy and physiology of the ocular and visual system. It should be considered to be core material for in class learning pertaining to the eye, special senses, and the visual system.

- Important medical cases to connect in class learning to how patients present in the real-world with eye and vision problems.

Each case follows the ***SOAPS*** format, an adaptation of the cornerstone, socratic method of teaching in medical school.

S **Subjective:** A brief history is given from the patient perspective

O **Objective:** The relevant features that are elicited on examination that help to rule in or out given diseases that are associated with the presenting symptom

A **Assessment:** These are the diagnostic tests that confirm or refute the diagnosis, and

P **Plan:** The medications or surgical procedures that will be recommended to help manage the patient's condition.

S **Science:** A discussion of an important scientific principle, research paper or novel scientific breakthrough.

All cases were developed for a non-majors human biology course, but could also be used in introductory anatomy and physiology, health science, or high school AP biology.

These cases are also meant to prepare your students for our quarterly Medical B, which is a case-based competition ideal for those who are interested in pursuing a career in medicine or the health field.

Anatomy and Physiology

I. ANATOMY OF THE EYE

The eye is the organ of vision. It allows you to see your world—everything from the bright colors and fine detail when scrolling on your phone to the ability to see in the dark when you are in a movie theater.

The eye receives visual sensory input from the environment and conveys it as images to the brain.

The adult eye is about 2.5 cm in diameter if we draw a line from the front to the back of the eye. It sits in the orbit, which is a body space or recession of the skull. Humans have 2 eyes—although, very rarely, babies can be born with one eye. This is called cyclops, but babies with cyclops have many other medical problems and usually are stillborn (this means that they are not alive upon birth).

The orbit is formed by many bones of the skull, including the maxilla, lacrimal, sphenoid, and ethmoidal bones. Sometimes when the eye is hit, the eye itself is not significantly damaged, but the pressure from something hitting the eye can result in an orbital fracture (one of the bones of the orbit can be broken). If this happens, fat from the orbit or some of the eye movement muscles can get trapped in the fracture. The eye may not move properly, and the affected patient may have double vision (this means that they see 2 images, either side-by-side or one on top of the other).

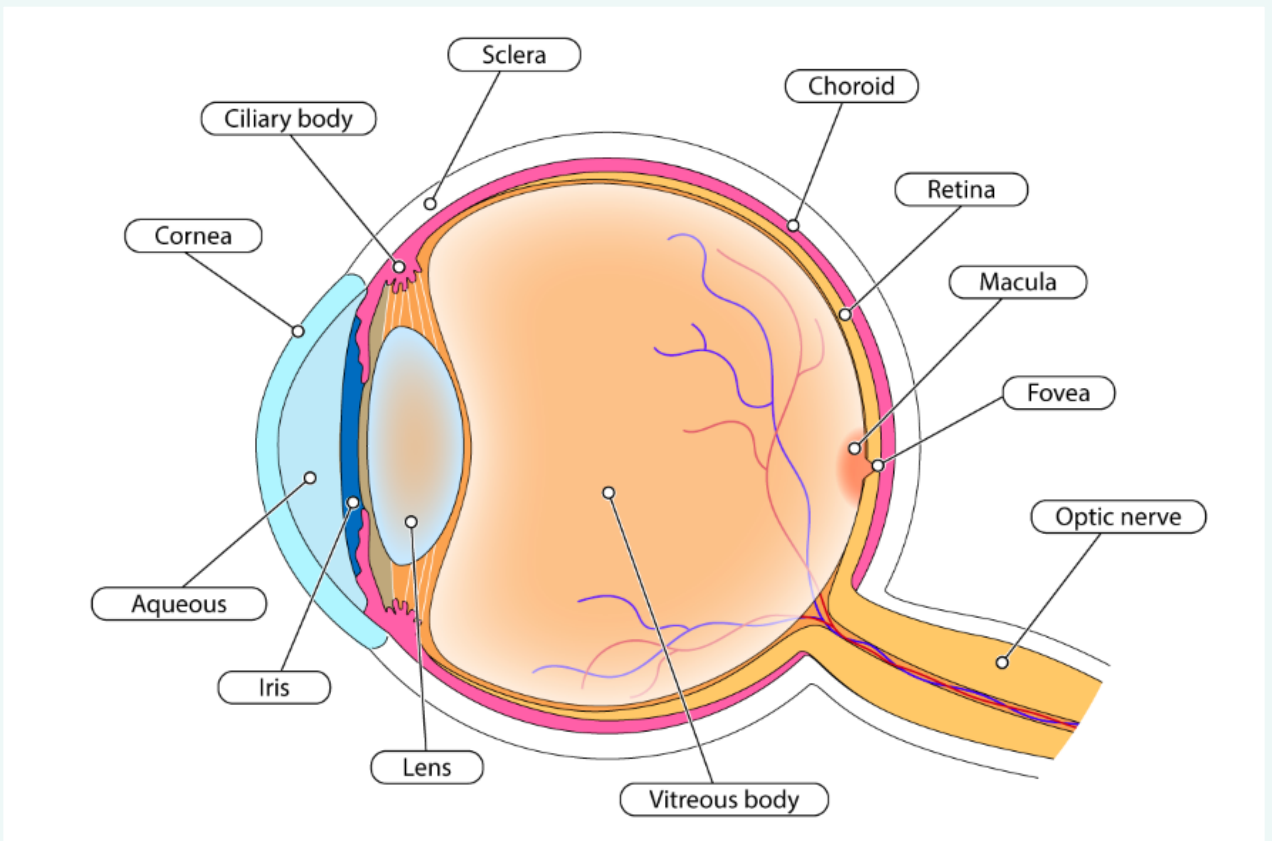


Figure 1: The anatomy of the eye

THE EYE PIZZA

The wall of the eye itself has three layers (**Figure 1**).

I describe the wall of the eye to my students as being like a pizza! I'm sure that many of you're a little grossed out by the fact that I'm saying the eye is akin to food, but rest assured, I'm not suggesting that you eat an eye! (side note - except if you're on a desert island, in which case eating an animal's eye is actually a great idea since it's a source of water that is not infectious and it is nutrient rich. Eating eyes has allowed many stranded souls to survive in the wilderness).

The reason I say it's like a pizza is because, like a pizza, it has 3 layers:

1. The thick hard outer layer, much like the crust, called the fibrous tunic. Anatomically, this consists of the cornea in the front and the sclera in the back. This layer provides protection and support to the eye.

2. The thin red layer, like the sauce, is called the vascular tunic. Comprised of the choroid, ciliary body and iris, this layer is responsible for providing nutrition and oxygen to the eye tissues.

3. The innermost layer, like the pizza toppings, is called the retina. This layer is a thin film that is responsible for trapping light rays and converting them to electrical impulses.

THE FIBROUS TUNIC (CORNEA AND SCLERA)

The fibrous tunic is the outermost layer of the eyeball and consists of the cornea at the front and the sclera at the back. The role of the fibrous tunic is to be a tough outer coat to protect all of the inner portions of the eye. Think of this like the phone case and screen protector that prevents your phone from becoming damaged if it is dropped.

The cornea is a clear coat of the fibrous tunic located at the front of the eye. It sits in front of the coloured part of the eye called the iris. If you try to touch the colored part of someone's eye, you wouldn't be able to because the cornea is in front of it (we, of course would not recommend it because this is a very painful experience because the cornea has many nerves).

The cornea is curved. This is really important because when light hits a curved, transparent surface, its rays are bent. This is the process of focusing, also known as refraction. The main purpose of the eye is to take light rays from the environment and use the cornea (and the lens, which is located inside the eye, more on this in the next section) to focus the light rays on the retina.

In the retina, light is converted into electrical impulses. These impulses leave the eye through the optic nerve, which is the large cable that connects the eye to the brain.

The sclera is the white part of the eye. It covers the entire eyeball except at the cornea in the front. The sclera gives the eye its shape and protects the inner parts of the eye.

The sclera is also the site of attachment for the muscles that are responsible for moving the eye; these muscles are called extraocular muscles. If these muscles become paralyzed, the patient won't be able to move his or her eye properly, potentially causing double vision. If you examine them, their eyes will not be lined up properly either due to the muscle imbalance.

THE VASCULAR TUNIC (CHOROID, CILIARY BODY, AND IRIS)

The vascular tunic (or uvea) is the middle layer of the eyeball. It consists of 3 parts:

- the choroid,
- ciliary body, and
- iris

The Choroid

The choroid is located in the back of the eye. It is sandwiched between the sclera (the pizza crust) and the retina (the toppings). The choroid is made up almost exclusively of blood vessels that create a network, almost like a carpet or a doormat.

While the choroid is mostly made of vessels, they also contain some pigmented cells called melanocytes. These cells are densely packed with a pigment called melanin. Melanin can absorb stray light rays and prevent scattering of light within the eyeball.

Melanocytes create a darker choroid in people who are more pigmented. Rarely, melanocytes can divide and replicate causing tumors. In some cases, these cells can give rise to cancers called malignant melanomas. These are similar to pigmented lesions (moles) on the surface of the skin. If this happens, the eye may need to be removed because this type of cancer can spread to the blood and other tissues, most commonly the liver.

The retina is a 10-layer tissue (more on this below). It has a dual blood supply. The inner half of the retina is supplied by a visible artery called the central retinal artery. The outer half of the retina receives its supply of nutrients from the choroid.

The Ciliary Body

The uveal component in the middle of the eye is called the ciliary body.

The ciliary body has 2 main functions:

- To hold the lens in place, and
- To create aqueous humour, which is a fluid that helps pressurize the eye to prevent it from collapsing and also provides nutrients and oxygen to cells in the front of the eye (**Figure 2**).

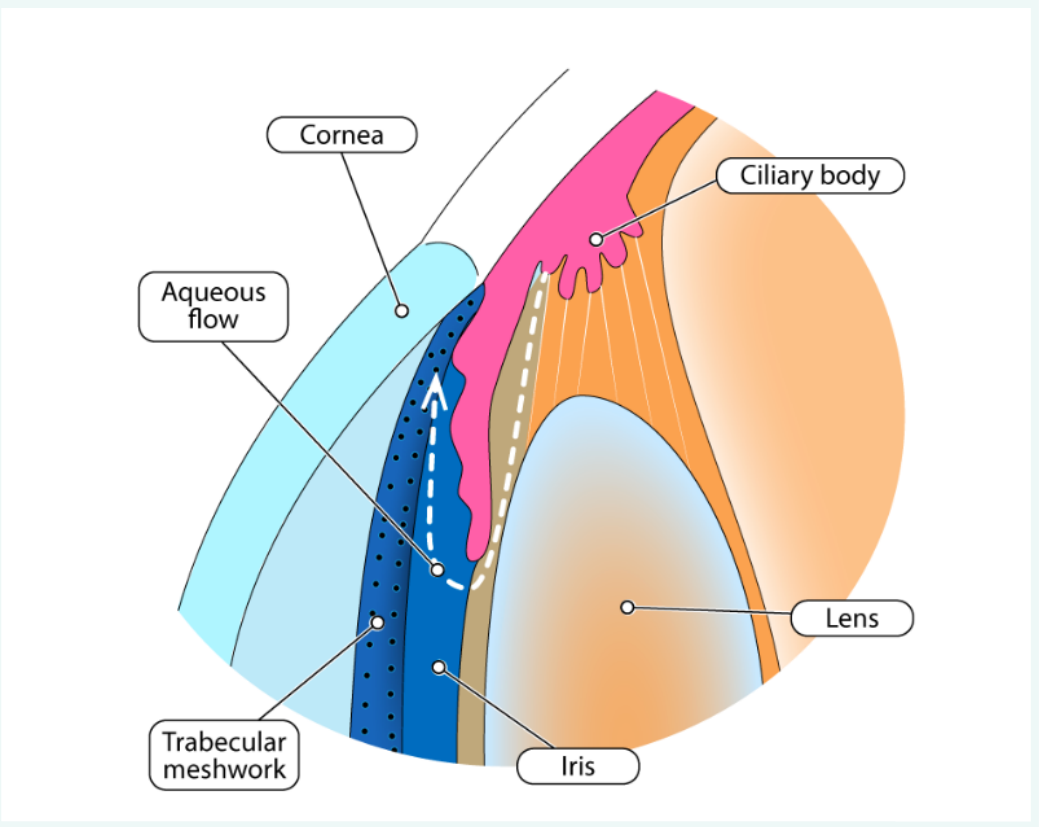


Figure 2: Aqueous humor movement in the front of the eye

The surface of the ciliary body consists of finger-like projections called ciliary processes. The ciliary processes are folds on the inner surface of the ciliary body; they contain capillaries, which secrete aqueous humor (more on this later).

The ciliary body also anchors the lens of the eye through tiny string-like structures called zonules. The ciliary body not only keeps the lens in place, but it is also responsible for changing its shape. When objects are far away, the lens stretches out and becomes thin; when objects get closer, the lens condenses to become thicker. The process by which the lens changes its AP diameter is called accommodation (AP means anterior-to-posterior, which refers to the distance from the front to the back of the lens).

There are many conditions in which patients' zonules can become weak. One of these is called Marfan's syndrome. People with this condition are typically very tall and they can have breakdown of the elastic fibers in other tissues. They are at risk for lens dislocation and retinal detachment. Most worrisome is that this condition can cause an aortic aneurysm (the aorta is the large arterial trunk coming off the heart that takes oxygenated blood to most of the body). Interestingly, there is a professional basketball player, Isaiah Austin, who had Marfan's syndrome but was unable to play in the NBA because of his medical condition.

The second important function of the ciliary body is to create aqueous humour. This fluid leaves the ciliary body and flows anteriorly bathing the lens, iris, and cornea in oxygen and nutrient-rich fluid.

The Iris

The iris is the most anterior part of the uvea. It is the part of the eye that provides its color. So, when we say someone has blue, green, or brown eyes, we are talking about the colour of their iris. This color is determined by the amount of melanin contained within the iris. The iris is in front of the ciliary body and therefore lies in front of the lens, but behind the cornea.

The iris also contains circular and radial smooth muscle fibers. One of the iris' main roles is to regulate the amount of light entering the eye through the pupil, which is the hole at the center of the iris. In a dark environment, the pupil dilates to allow more light into the retina; in bright light, the pupil contracts, reducing the amount of light entering the eye.

Pupil size is regulated by the autonomic nervous system (see the Neurology section for more information). When bright light stimulates the eye, parasympathetic fibers from cranial nerve III (oculomotor nerve) stimulate the circular muscles (sphincter pupillae) of the eye to contract, which causes the pupil to constrict (decrease pupil size). In dim lighting, neurons of the sympathetic nervous system stimulate the radial muscles (dilator pupillae) to contract, which causes the pupil to dilate (increase pupil size)

The pupil can also be dilated when our sympathetic nervous system is stimulated. This happens when we are nervous or fearful as part of the "fight or flight" response. This is a system that we inherited from our caveman ancestors: when they saw a sabretooth tiger, the hormone epinephrine was released. This hormone caused a number of effects, including speeding up the heart, moving blood to the muscles, opening up the airways, and dilating the pupils to allow more light to enter. To this day, if we are nervous or faced with danger, many of these same things happen.

THE RETINA

The retina is the innermost layer of the eye wall; the topping of the pizza!

It is a 10-layer tissue that is responsible for trapping light rays and converting them to electrical impulses.

The retina can be directly viewed in the clinic by doctors using an ophthalmoscope, which is an instrument that shines light into the eye to allow the user to look through the pupil at the retina, its blood vessels, and the optic nerve (cranial nerve II). When looking through the ophthalmoscope, several parts of the retina can be visualized. Here are some things that we commonly look at when examining a patient with an ophthalmoscope:

- The health of the optic nerve: this can be evaluated by looking at the optic disc, which is the surface of the optic nerve. The optic nerve can appear to be bulging in a person with brain swelling from a tumor or a bleed.

- The blood vessels: bundled in the individual nerve fibers that make up the optic nerve are two very important blood vessels: the central retinal artery, which brings blood to the inner half of the retina; and the central retinal vein, which drains blood away from the inner half of the retina.

- The macula: this is the central part of the retina that has the sharpest vision. It measures roughly 5 mm in diameter and is contained between the visible branches of the retinal arteries and veins.

The retina contains two layers: a neural (sensory) layer and a deeper pigmented layer.

Neurosensory Retina

The neural layer has 10 layers and consists of 3 important cells:

- Photoreceptors: cells that trap light energy and convert it into electrical impulses. There are 2 types of photoreceptors: rods and cones. Rods are cells that are found in the retinal periphery; they are responsible for our sight in the dark and our peripheral vision. Cones are located in the macula; they are responsible for fine vision and seeing color.

- Bipolar cells: integrate electrical impulses generated from the photoreceptors and relay them to the third cell type, called ganglion cells.

- Ganglion cells: move impulses out of the eye and into the brain. Ganglion cells have very long axons that go through an opening in the sclera and make up the optic nerve (in healthy people, the optic nerve is made up of around 1.2 million axons from the ganglion cells). Because the optic disc consists of a bunch of cables and no photoreceptors, if a light is shone on this area it cannot be seen—this is called a "blind spot". It's the same reason why we have to turn our head when we are driving and trying to change lanes so that we don't hit a car or cyclist who is moving into our blind spot!

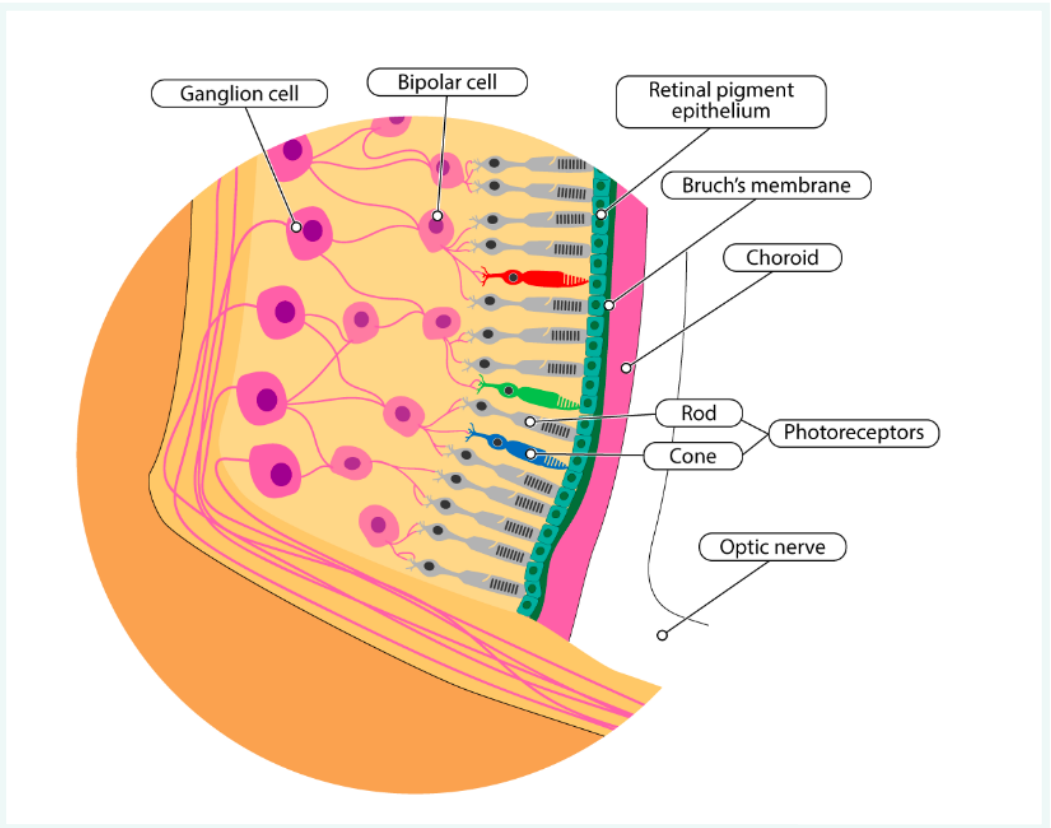


Figure 3: The cells of the retina responsible for vision

The Retinal Pigment Epithelium

The retinal pigment epithelium (RPE) is a single layer of cells that sits on top of the choroid (remember this is the vascular layer that sits on top of the sclera). These cells are very important because they provide oxygen and nutrients to the outer part of the retina. The RPE also plays an important function in recycling the light-sensitive pigments in the photoreceptors. The melanin in the pigmented layer also helps absorb stray light rays, similar to the choroid.

Interestingly, light passes through the ganglion and bipolar cell layers as well as both synaptic layers before reaching the photoreceptor layer.

The macula lutea is the most central part of the back of the retina. The fovea centralis is a small dip in the centre of the macula lutea that only contains cones. This area is not covered by the bipolar and ganglion cell layers, and as a result, the fovea centralis is the area of highest visual acuity (sharpness of vision). The main reason that we move our head and eyes when we look at something is to place images on the fovea centralis. If we look at cross-sectional OCT images from the back of the eye, you will see a small pit in the foveal region since it is not covered by the bipolar and ganglion cell layers. OCT stands for ocular coherence tomography— it creates images based on the reflection of energy waves, similar to ultrasound, but ultrasound uses sound waves whereas OCT uses light rays.

SPACES IN THE EYE

There are 2 important spaces in the eye: the anterior chamber and the vitreous. Both are very important to make sure that the eye sees properly.

The anterior chamber is a space in the front of the eye. The anterior chamber extends from the inside part of the cornea to the iris. This area contains a clear fluid called the aqueous humor, which is produced by a structure called the ciliary body. The aqueous humor (frequently called just “aqueous” by doctors) does 2 important things:

- Brings nutrition and oxygen to some of the cells inside the eye, and
- Creates the pressure in the eye that keeps it nice and round! If there wasn’t enough pressure in the eye, it would deflate (sort of like a basketball with no air). You might be wondering whether it’s actually possible for the eye to lose its pressure. Yes, it is! For instance, I recently saw a patient who had a fishhook lodged in their eye. It tore a hole in the eye and fluid started to leak. We had to sew the eye back together to eliminate the fluid from leaking further. If the eye’s coating rips open, eye surgeon’s call it an, “open eye”. This needs to be closed emergently because infection can quickly set in or the eye’s contents can start to be pushed out of the eye causing further damage.

You might also wonder, “if the eye is creating fluid to maintain pressure, and the eye is a closed system, can the pressure ever rise to a point where the eye can rupture?” This is a great question! It turns out that the eye has thought of everything! In fact, like a sink with a tap that allows water to fill it, there is also a drainage system that allows fluid to leave the eye. The eye’s drainage system is located where the iris and cornea meet. The drainage system is called the trabecular meshwork. Fluid moves through here and eventually drains into the veins on the surface of the eye, which eventually makes its way back to the heart through the veins that collect blood from the face and upper body.

VISUAL ACUITY

An objective measure of visual function is visual acuity. This refers to how well someone can see. It typically is recorded as 2 numbers: 20/40 or 20/30. The two numbers refer to the distance away from the eye chart that a patient has to stand to see the letters on the chart, in comparison to an able sighted person. So for instance when the vision is 20/40, it means that an able sighted person can see the image from a distance of 40 feet, but the patient has to move closer (20 feet) to see it. If the vision is 20/200, an able sighted person can see the image from 100 feet away, but the patient needs to be 20 feet away.

OPHTHALMOSCOPY

The retina is examined using a slit lamp (**Figure 4**) with a hand held lens (we also use a hand held light which contains magnifying lenses called an ophthalmoscope to examine the retina). The optic nerve is the white circular structure that stands out against the orangish retina. The retinal artery and veins are visible as thin structures that are reddish in color (**Figure 5**). The macula is the area located next to the optic nerve and within the vascular arcade (the area framed by the blood vessels). The patient in figure 5 has had diabetes for a number of years. Because of this, there are numerous hemorrhages in the retina which is an abnormal finding.

Figure 4: Patient examined with a slit lamp

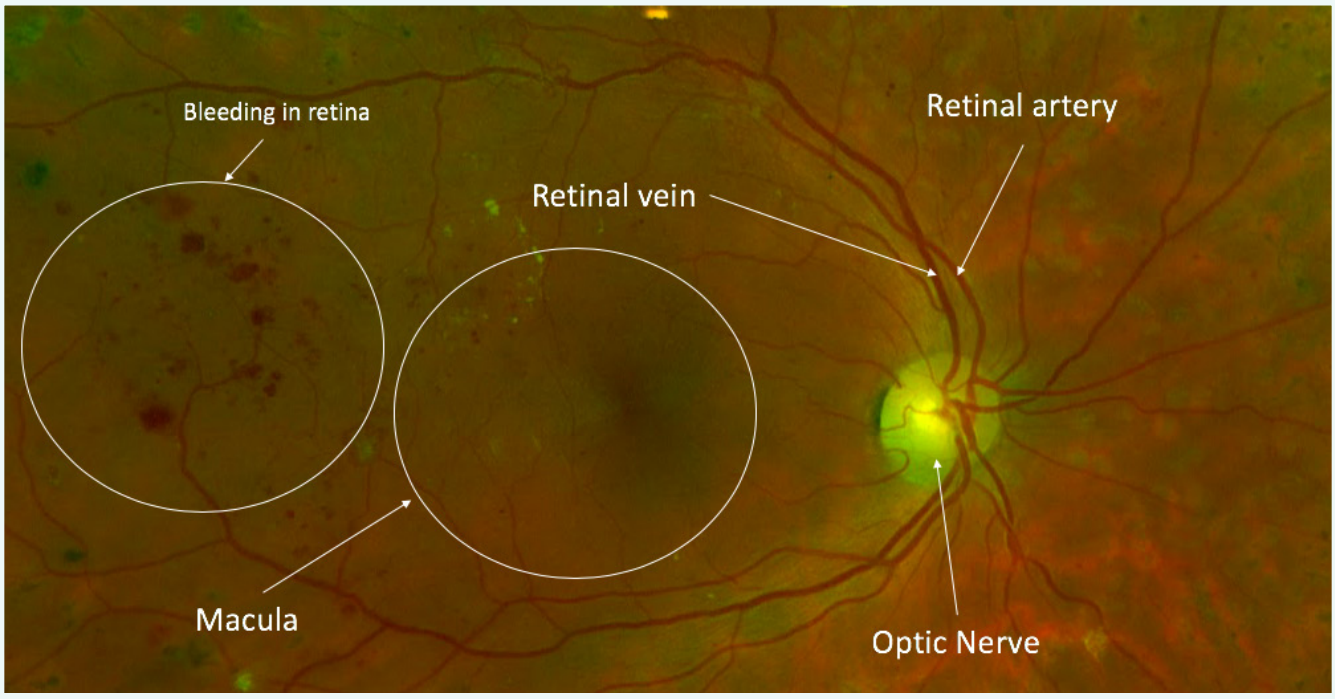


Figure 5: The retinal showing hemorrhage in the retina

A new diagnostic test called ocular coherence tomography (OCT) has revolutionized how we diagnose retinal diseases.

You have probably heard of an ultrasound test that uses sound waves to create images of tissues to help diagnose patients with a variety of conditions including differentiating a breast cyst from a solid tumor and confirming the diagnosis of appendicitis in a patient presenting with abdominal pain. OCT is similar to an ultrasound test, except instead of sound waves, the test uses incident light rays to create images. Additionally, this test has incredible resolution, with the ability to detect changes and pathology to the 20 micron level.

Figure 6 shows an OCT in a patient who presented with sudden visual loss. The right side of the OCT image is normal and shows the normal lamellar architecture of the photoreceptors, bipolar cells and ganglion cells, the 3 cells that allow for phototransduction and electrical impulses to move from the eye to the brain. You can also see that there is a significant elevation of the inner retina because this patient had a large aneurysm that ruptured. For reference, the normal retinal thickness is about 250 microns thick (and this person's aneurysm is about 150 microns in diameter).

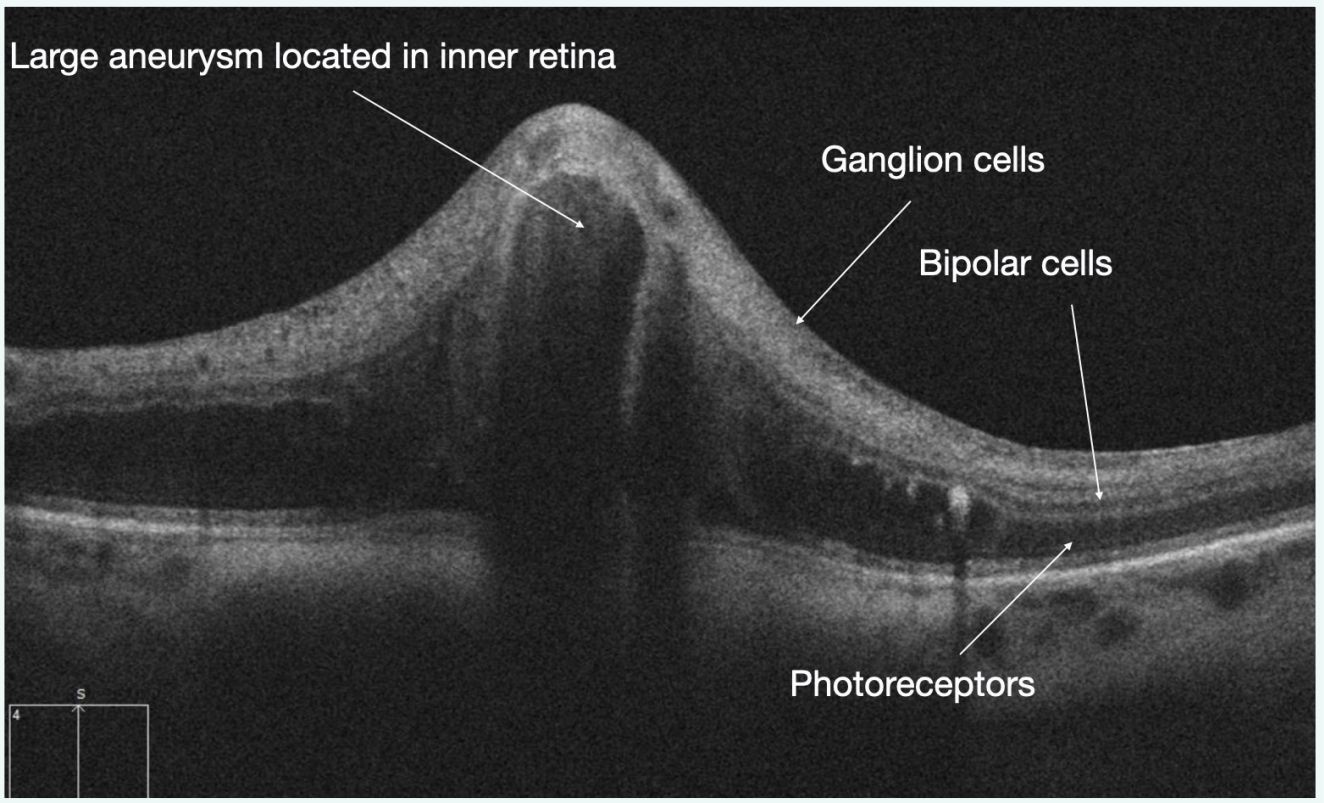


Figure 6: An OCT in a patient with a retinal artery macroaneurysm

THE LENS

The lens is a small structure that bends light rays to focus images on the retina. It is located behind the pupil and iris. It is made up of proteins and is normally transparent. The lens is enclosed by a clear capsule similar to a plastic wrap.

The lens divides the inside of the eyeball into two cavities called the anterior cavity and the vitreous chamber.

The anterior cavity itself contains two chambers:

- The anterior chamber, which is found between the cornea and the iris, and
- The posterior chamber, which lies behind the iris and in front of the zonular fibers and lens.

Both chambers of the anterior cavity contain aqueous humour, which is a clear fluid that nourishes the lens and the cornea. The aqueous humour continuously flows out of blood capillaries in the ciliary processes and into the posterior chamber. The fluid then flows forward between the iris and lens, then through the pupil and into the anterior chamber. From the anterior chamber, the canal of Schlemm drains the aqueous humour to go back into the blood. The aqueous humor gets replaced around every 1.5 hours.

The vitreous chamber is much larger than the anterior cavity. It lies between the lens and the retina. Inside the vitreous chamber is the vitreous body, which is a transparent jelly-like substance that holds the retina flat against the choroid, giving the retina an even surface to receive clear images. The vitreous body does not undergo replacement like the aqueous humor. The vitreous body also contains phagocytic cells that help to remove debris and keep this part of the eye clear so that vision is not obstructed.

As mentioned before, all eyes are pressurized to make sure that they don't collapse. Eye doctors routinely measure the pressure in the eye, which is called the intraocular pressure. The normal pressure in the eye is usually in the teens, with the upper level of normal being 21 mmHg. Very high pressures in the eye can cause compression of the axons as they make a right angle turn and leave the eye through the optic nerve.

ACCESSORY STRUCTURES OF THE EYE

Accessory structures of the eye include the:

- conjunctiva
- eyelids
- eyelashes
- eyebrows
- lacrimal glands
- extraocular muscles

The surface of the sclera has a thin vascular structure called the **conjunctiva**. This layer starts at the junction of the cornea and sclera and covers the front of the sclera and then folds over to the backside

of the eyelid like a taco. So, if an eyelash drops onto the surface of the cornea, it will never travel back to the optic nerve because the conjunctiva folds over and prevents objects from passing through. The conjunctiva is responsible for creating much of the tears which prevent our eye from becoming dry. It also creates mucin which is a fatty substance that prevents tears from evaporating.

The **eyelids** consist of a collagen plate on the back, with the conjunctiva lining the side of the lid touching the eye. This plate is called the tarsus. **Eye lashes** are anchored in the tarsus. This plate also contains many meibomian glands which create more fatty substances similar to mucin that prevent tears from drying. Lids are important because they protect the eyes from excess light and foreign bodies, cover the eyes during sleep, and help to lubricate the eye's surface.

The **lacrimal apparatus** is a group of structures that produce and drain lacrimal fluid (aka tears). The lacrimal glands secrete tears, which then drain into several lacrimal ducts that empty the tears onto the conjunctival surface of the upper eyelid. Then, tears pass through a series of canals to eventually reach the nasolacrimal duct, which carries the tears into the nasal cavity. This is why crying also creates a runny nose. Tears help to lubricate and clean the eyeball in the case of irritation but also are produced in emotional responses to happiness or sadness.

The **extraocular muscles** are a set of 6 muscles that move each eye in almost any direction. The 6 extraocular muscles are controlled by three cranial nerves (CN): the oculomotor nerve (CN III), the trochlear nerve (CN IV), and the abducens nerve (CN VI). The 6 muscles are listed below with their function and the nerve that controls them:

- **CN III: Oculomotor Nerve**
    - Superior rectus: elevates the eye (used to look up)
    - Inferior rectus: depresses the eye (used to look down)
    - Medial rectus: Moves the eye towards the nose (adduction)
    - Inferior oblique: Externally rotates, elevates and abducts the eye
- **CN IV: Trochlear Nerve**
    - Superior oblique: Internally rotates, depresses and abducts the eye
- **CN VI: Abducens Nerve**
    - Moves the eye away from the nose (abduction)

- **Superior rectus (CN III: Oculomotor Nerve)**
    - Elevates the eye (used to look up)
- **Inferior rectus (CN III: Oculomotor Nerve)**
    - Depresses the eye (used to look down)
- **Lateral rectus (CN VI: Abducens Nerve)**
    - Moves the eye away from the midline of the body (abduction)
- **Medial rectus (CN III: Oculomotor Nerve)**
    - Moves the eye towards the midline of the body (adduction)
- **Superior oblique (CN IV: Trochlear Nerve)**
    - Internally rotates, depresses and abducts the eye
- **Inferior oblique (CN III: Oculomotor Nerve)**
    - Externally rotates, elevates and abducts the eye

II. PHYSIOLOGY

IMAGE FORMATION – REFRACTION AND ACCOMMODATION

To understand how the eye forms clear images of objects, there are 3 processes to understand:

1. Refraction (bending) of light by the lens and the cornea.
2. Accommodation, which is the change in the shape of the lens.
3. Constriction (narrowing) of the pupil to allow more or less light into the eye.

Refraction

Refraction refers to bending of light. This is necessary, because light rays from distant objects need to come into sharp focus on the retina. And since the focal distance from where light enters the eye to where the retina is located is only 2.5 cm, the eye requires a powerful refraction system. In fact, the cornea and lens together result in a 60 diopter lens, with the cornea contributing 40D or power and the lens an additional 20D.

Many people have refraction abnormalities and are unable to refract light rays at the normal 6m position. Myopia (nearsightedness) is one of these abnormalities. This occurs when the eyeball is too long relative to the focusing power of the cornea and lens, or when the lens is thicker than normal, so rays converge in front of the retina rather than directly on the retina. People with myopia can see objects clearly up close but objects further away appear blurry. The opposite of myopia is hyperopia (farsightedness). This occurs when the eyeball is too short relative to the focusing power of the cornea and the lens, or when the lens is thinner than normal, so rays converge behind the retina. People with hyperopia can see objects that are far away clearly but objects up close appear blurry.

Astigmatism is another refraction abnormality and occurs when the cornea or lens has an irregular curvature. This causes parts of an image to be out of focus and thus images look blurry or distorted.

All of the above refractive errors can be corrected using eyeglasses, contact lenses, or through surgery such as LASIK (laser-assisted in-situ keratomileusis).

Accommodation

To see properly, the 2 surfaces that refract (or bend) light—namely, the cornea and lens—have to curve the rays such that they come to focus on the retina. If an image is very far away, its light rays enter the eye perpendicular to the lens and don't have to be bent very much. But if an object is very close to the eye, like the tablet or computer on which you are reading this, the rays that enter the eye are diverging (**Figure 7**). So, to get in focus on the retina, they need to be bent much more. This can only happen if the lens changes shape and becomes much thicker. This process of the lens getting thinner and thicker happens every time you focus on a new object that is at a different distance from the eye.

The way that the lens gets thinner and thicker is controlled by contraction and relaxation of the muscle in the ciliary body. The force of the muscle is transmitted to the zonules, which in turn is transmitted to the lens, causing the change in shape.

As we get older and into our 40s and 50s, the lens fibers become firmer and are not as pliable as they once were. This means that the lens has difficulty getting really thin and really thick. Because of this, people in middle age lose their ability to accommodate and need to wear glasses for reading.

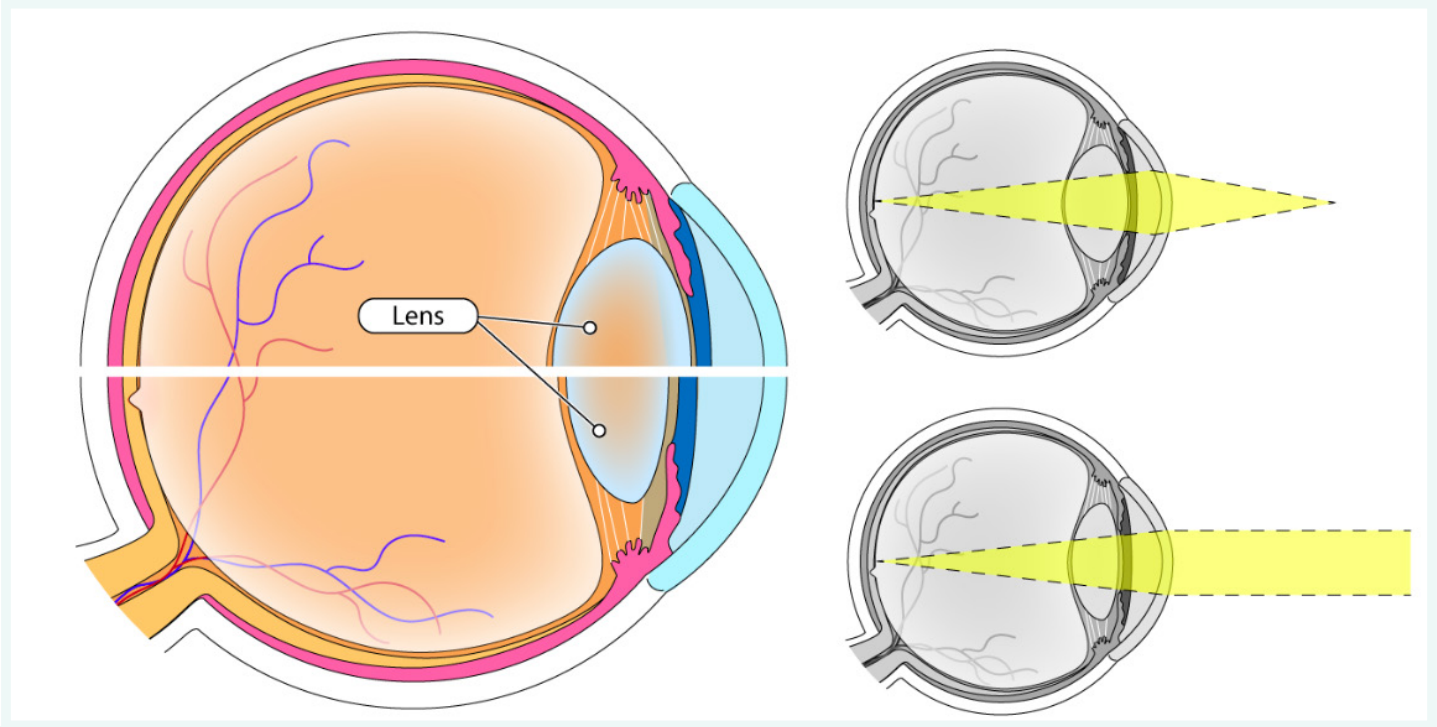


Figure 7: Accommodation of the lens allows a close object to be seen

Pupillary Constriction

Pupillary constriction occurs at the same time as accommodation through the autonomic nervous system. As mentioned previously, the pupil constricts when light enters the eye due to contraction of the circular muscle fibers of the iris. This prevents light rays from reaching the retina through the periphery of the lens. If light enters at the periphery, it will not be brought into focus directly on the retina, resulting in a blurry image.

As mentioned previously, visual information in the retina undergoes processing at a number of levels through different types of neurons. This information is then outputted from the retina to the brain through the optic nerve (cranial nerve II).

The information from the optic nerve travels through the visual pathway to reach the brain (**Figure 8**). The general pathway is as follows:

- Optic nerve (cranial nerve II)
- Optic chiasm
- Optic tract
- Lateral geniculate nucleus of the thalamus
- Optic radiation
- Primary visual area of the cerebral cortex in the occipital lobe

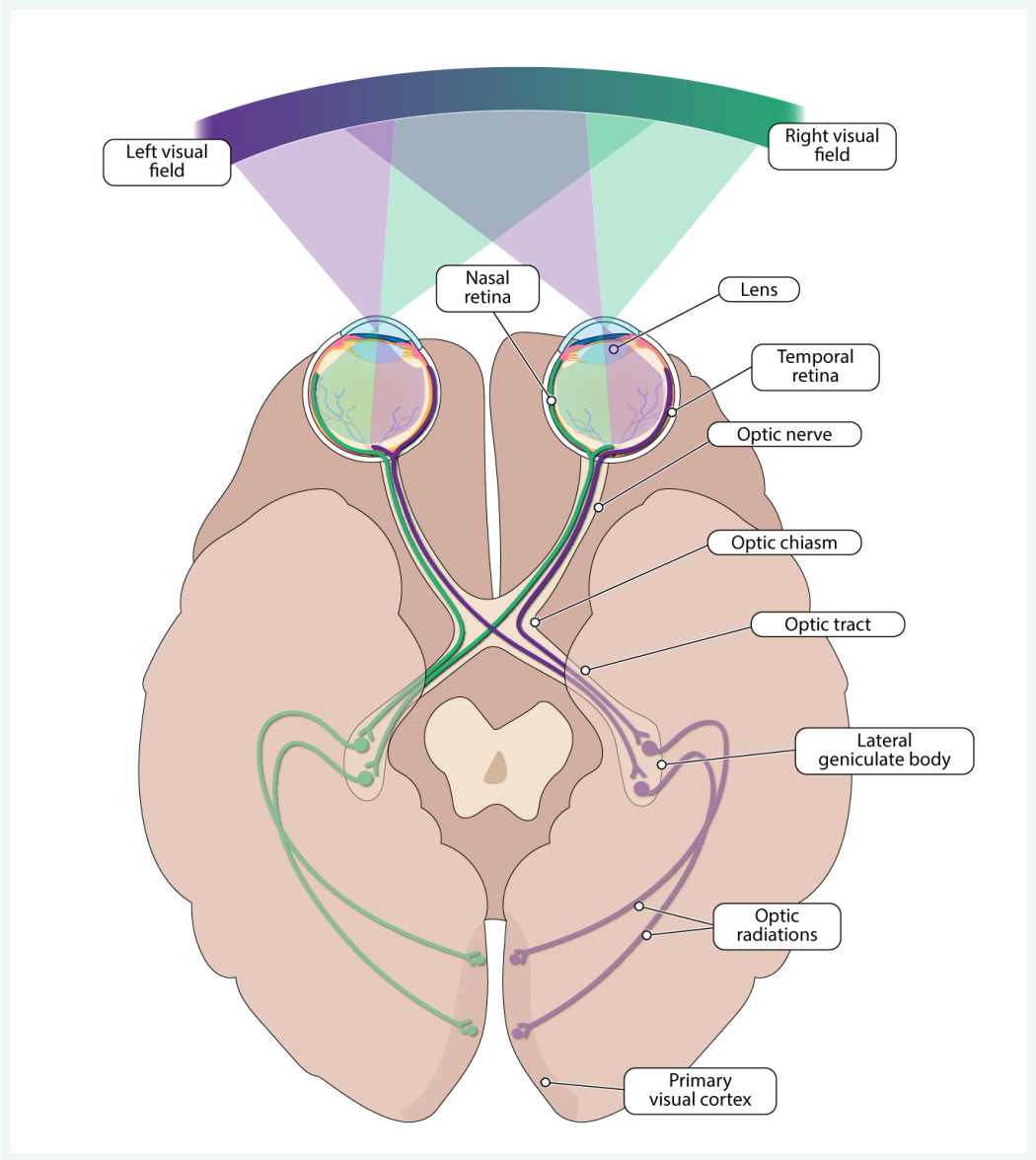


Figure 8: The visual pathway

Everything that can be seen by one single eye is referred to as that eye's visual field. As you might have gathered, the visual fields of both of our eyes overlap quite a bit. The region where both fields overlap is called the binocular visual field. If we look at each visual field individually, we can divide them into two halves: a nasal (central) half and a temporal (peripheral) half. For each eye, light rays from an object in the nasal half of visual field falls onto the temporal half of each retina, and vice-versa. Thus, visual information from the right half of each visual field is sent to the left side of the brain and visual information from the left half of each visual field is sent to the right side of the brain.

If you follow the diagram, axons from the retinal ganglion cells in one eye exit at the optic disc to form that eye's optic nerve. At the optic chiasm, axons from the temporal half of each retina continue to the lateral geniculate nucleus of the thalamus on the same side, eventually to the primary visual area of the occipital lobe. However, axons from the nasal half of each retina will cross over at the optic chiasm and continue to the opposite lateral geniculate nucleus and eventually the opposite primary visual area of the occipital lobe.

Ophthalmology Cases

CASE 1: A CASE OF DRY AGE-RELATED MACULAR DEGENERATION

Background

The macula is the central portion of the retina. It is only about 5-6 mm in diameter, but because it is an area of the retina that is very dense in cones, it is responsible for both fine and colour vision.

There are two types of macular degeneration: the dry and wet types. While wet macular degeneration, consistent with hemorrhage and fluid in the macula, accounts for the majority of cases of severe visual loss, dry AMD makes up the majority of cases of patients with AMD.

Risk factors for macular degeneration include, age over 50 years, smoking, a diet that is poor in antioxidants, nucleotide polymorphisms in AMD risk genes (linked to inflammation), obesity and hypertension.

Macular degeneration is a condition in which the deepest layers of the retina are affected: there is loss of photoreceptors, and the cells that support their nutrition (retinal pigment epithelial cells (RPE) supply oxygen and nutrients to the photoreceptors).

Patients with dry macular degeneration typically complain of blurred vision, black, gray or missing spots (scotomas). This can cause significant anxiety for patients; depression, falls and early nursing home admission has also been linked to this condition.

S - Subjective information

An 80 year old who has smoked for 60 years (2 packs per day), presents with the complaint that when they read, there is a central “smudge” in her vision. It comes from both eyes. The patient is on medication for blood pressure. They also mention that various family members have a history of visual loss.

O - Objective Exam

Vision is recorded at 20/40 from both eyes.

On examination, the retina shows drusen, or yellowish deposits are noted just below the retinal pigment epithelium (RPE). In addition, there are either hypopigmented areas (RPE atrophy) or areas of hyperpigmentation (RPE hyperplasia). Please see **Figure 9** to appreciate how drusen appear on clinical examination with an ophthalmoscope.

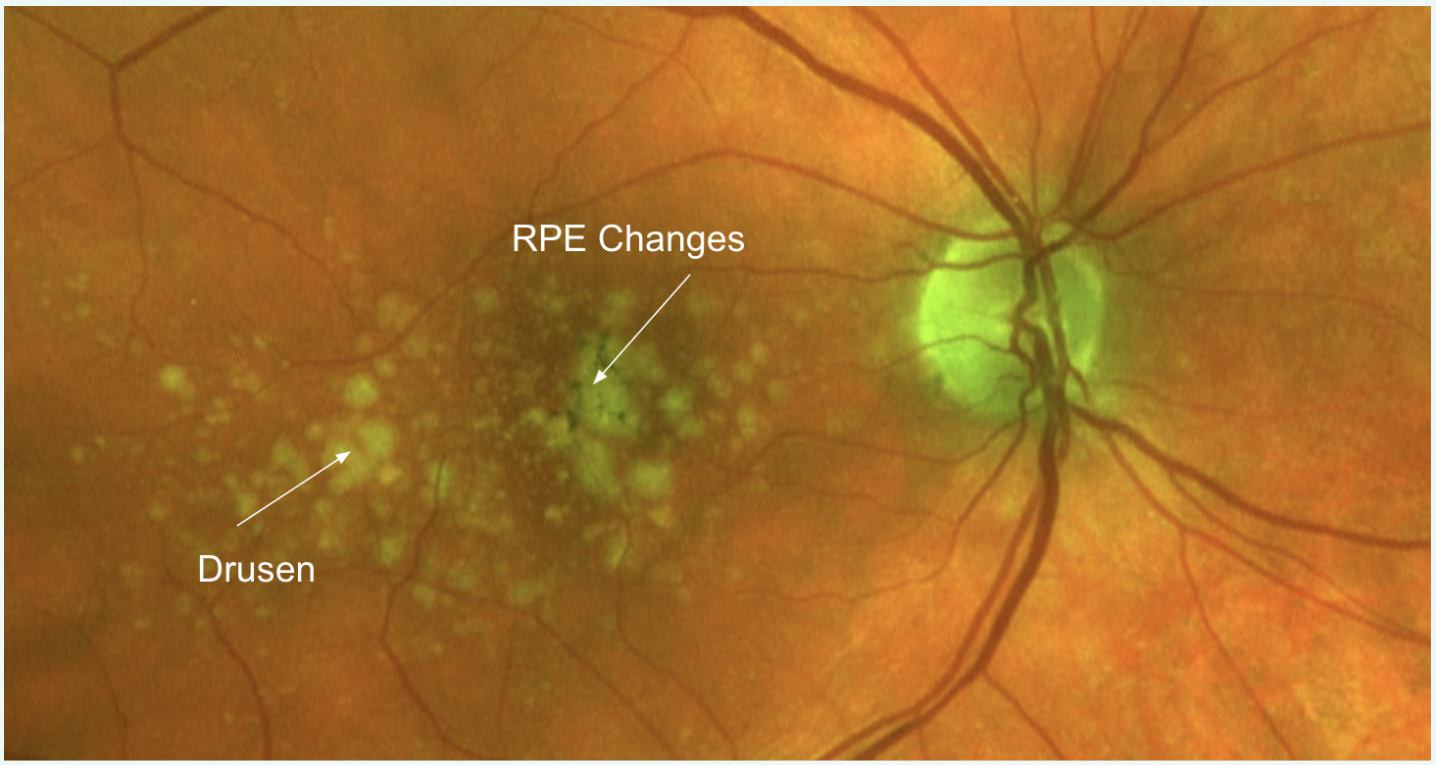


Figure 9: Drusen deposited in the deep retinal layers

A - Assessment

An OCT (ocular coherence tomography) shows elevation and thickening at deeper layers of the retina (**Figure 10**).

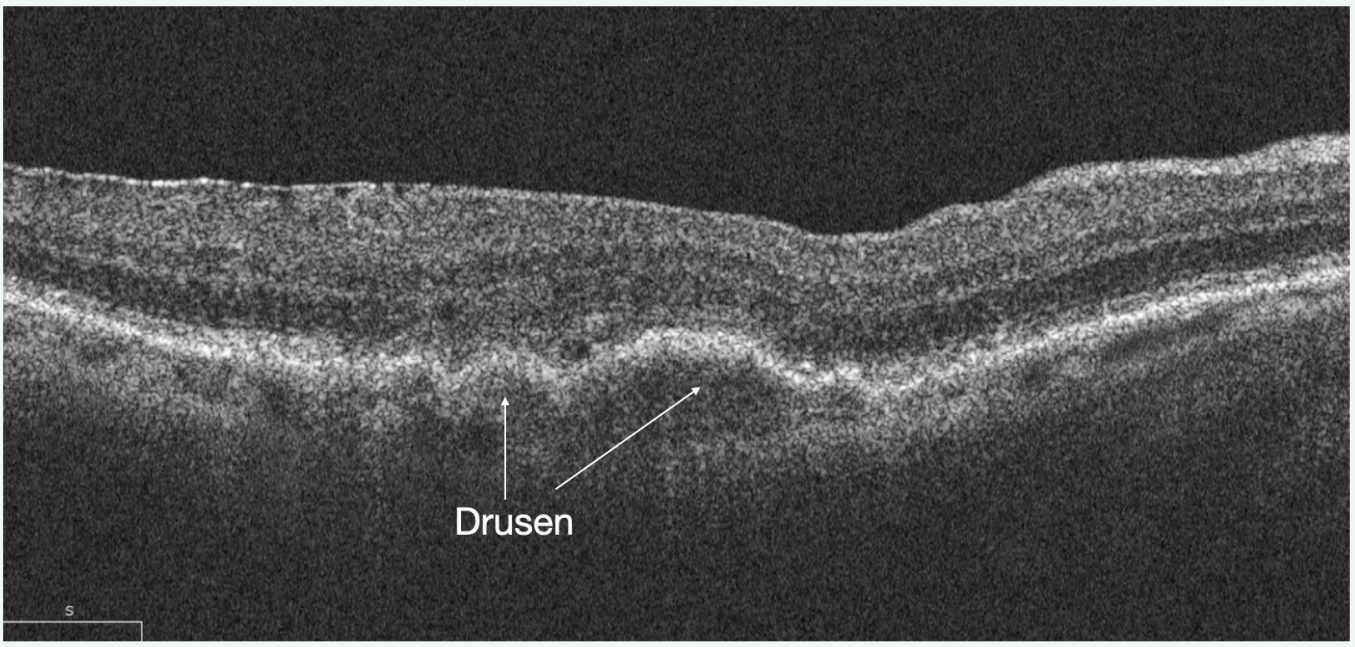


Figure 10: Drusen are visible as bumps present in the deep retina

P - Plan and discussion

Dry AMD is a chronic form of visual loss. However, many patients with the dry type of AMD can progress to the wet type.

AMD is known to be linked to a number of variables including genetic risk, smoking and poor nutrition. Treatments for dry AMD typically include lifestyle modification and the use of vitamins, smoking cessation and management of cardiovascular risk factors

An Amsler grid (a grid that measures distortion and scotoma size in the central 10 degree visual field) is also typically issued to document new changes.

S - Science

The use of high dose vitamin supplements has been shown to lower the risk of disease progression in patients with moderately advanced dry macular degeneration by 25%.

Age-Related Eye Disease Study Research Group. “A randomized, placebo-controlled, clinical trial of high-dose supplementation with vitamins C and E, beta carotene, and zinc for age-related macular degeneration and vision loss: AREDS report no. 8.” Archives of ophthalmology 119.10 (2001): 1417-1436.

What FutureMDs need to know about dry AMD:

- Any patient over the age of 50 years should have a dilated eye examination to rule out the possibility of macular degeneration (and other conditions like glaucoma and cataract).
- In any patient with new-onset visual loss or distortion (ie the perception where a window or door frame appears curved) urgent referral is warranted.
- Patients with dry AMD are typically followed by an eye doctor as this disease can progress to the more dangerous “wet” type which is linked to rapid and profound loss of vision.

Background

While cases of dry AMD greatly outweigh the number of cases of wet, nearly 90% of cases of AMD with severe visual loss are associated with wet AMD. The definition of "wet" AMD involves the presence of hemorrhage or fluid on clinical examination.

Wet AMD is caused by the presence of a new blood vessel growing in or under the retina. These vessels are called choroidal neovascular membranes (CNV), and are linked to the upregulation of intraocular vascular endothelial growth factor (VEGF). The location of the blood vessels can be under the RPE (retinal pigment epithelium), between the retina and the RPE (referred to as subretinal in location; these are called Type 2 membranes) or in the retina itself (type 3 membranes).

While dry AMD is associated with insidious visual loss, patients with wet AMD can lose vision very quickly and profoundly.

S - Subjective

A 77 year old presents with new distortion, describing that door frames appear curved from the right eye, as do telephone poles.

O - Objective

On examination, the patient's vision is recorded as 20/200 from this eye.

Retinal examination shows significant areas of hemorrhage and fluid in the deep retina of the right eye (**Figure 11**).

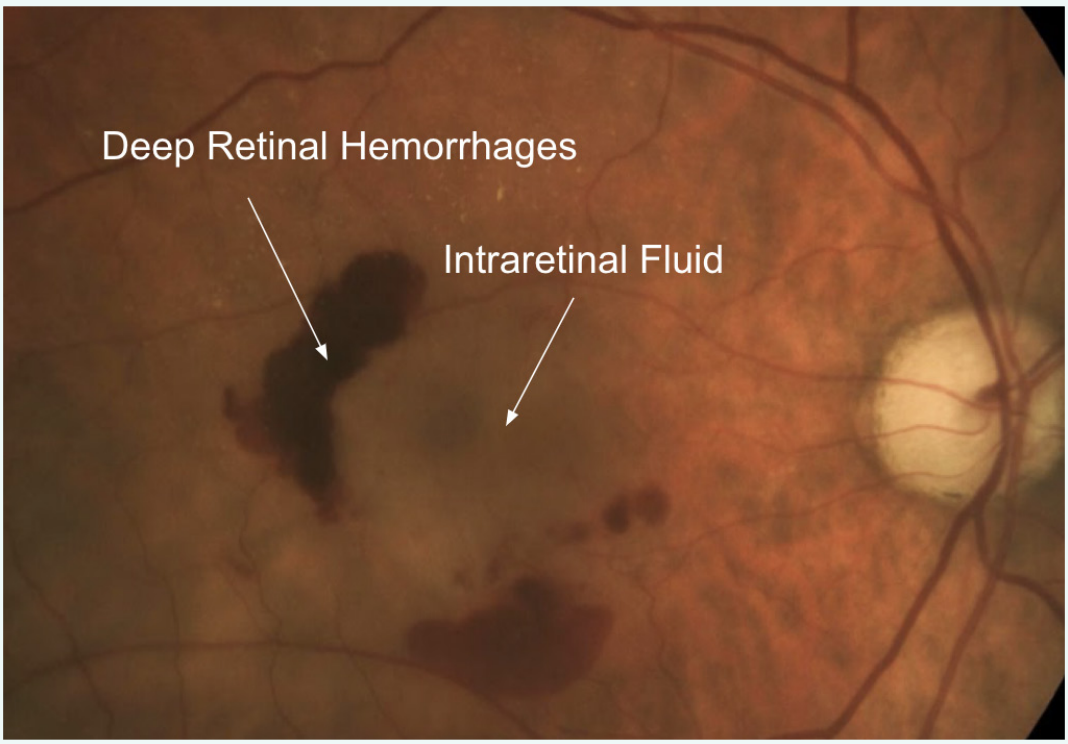


Figure 11: Hemorrhage and fluid in the macula consistent with wet AMD

A - Assessment

An OCT shows the presence of fluid under the retina. In addition there is some hyperreflective signal located deep to the retina confirming the presence of a new blood vessel that should not be there (**Figure 12**). These vessels are called choroidal neovascular membranes (CNV) as they are derived from the choroid, the vascular layer located beneath the retina.

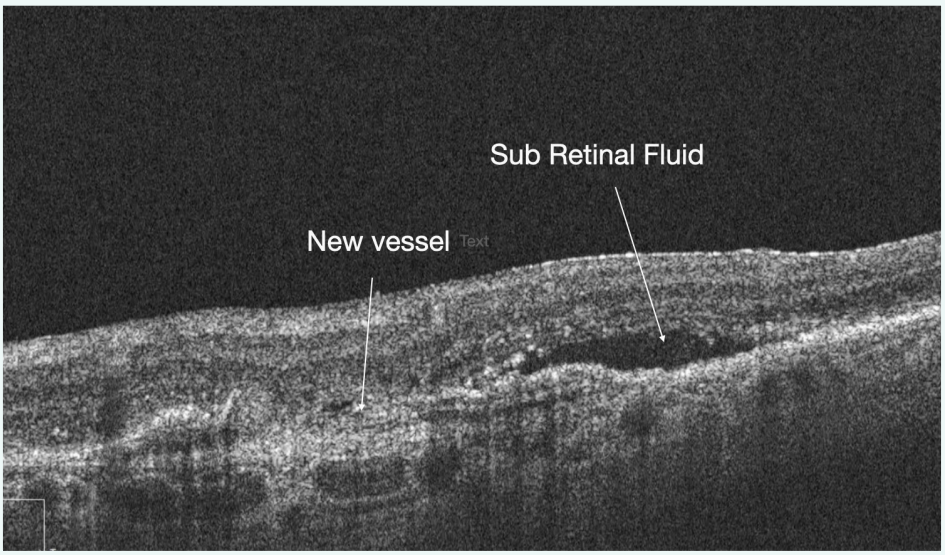


Figure 12: Subretinal fluid and CNV present on OCT

P - Plan:

While the treatment of wet AMD used to involve the use of lasers to cauterize the CNV, and the use of photodynamic therapy (here a photosensitizing dye is infused into a peripheral vein and once in the retina, it is photoactivated by the used of a laser to cause its involution), the mainstay of treatment is the delivery of an anti-VEGF compound through the use of an eye injection. Here a small needle is inserted into the space between the lens and the anterior retina and approximately 1/20 of a cc is injected into the vitreous cavity.

S - Science

There is a genetic predisposition to developing age-related macular degeneration. When I was in Boston, I studied a particular gene that is linked to this condition. It is now known that mutations to numerous genes are linked to macular degeneration Here is a link to that paper.

What FutureMDs need to know about wet AMD

- If a patient with dry AMD notices new distortion, urgent referral is necessary as this may represent progression to the wet type.
- In any elderly patient with new onset visual loss, urgent referral is necessary to rule out wet AMD or other cause of sudden visual loss, including retinal detachment, retinal artery or vein occlusion.
- Any person over the age of 50 years should have a dilated examination to rule our age-related eye conditions, including cataract, glaucoma and macular degeneration.

Background

Diabetes is a medical condition in which high sugars can deposit in the walls of the small vessels - called capillaries. This deposition can cause these small blood vessels to close down and is also associated with a higher risk of blood clot formation. Small blood vessel disease caused by diabetes can cause changes in the kidney (diabetic nephropathy), peripheral nerves (diabetic neuropathy) and the retina (diabetic retinopathy).

Diabetic retinopathy (DR) can be non-proliferative (NPDR) or proliferative (PDR), referring to the absence or presence of neovascularization (new blood vessels growing on the surface of the retina). The primary risk factors for DR include the duration of diabetes and the degree of glucose and/or blood pressure control.

Pathophysiology is characterized by upregulation of vascular endothelial growth factor (VEGF) which is secreted by the ischemic retina and causes retinal swelling, edema, and angiogenesis. Thus, anti-VEGF agents are used to treat DR (these are "biologic" agents and consist of monoconoal antibodies created to bind to VEFG. In addition, in advanced cases laser energy is used (this is called photocoagulation), as is surgery (this is called a vitrectomy, and here the vitreous is accessed and blood is removed from this cavity).

Symptoms of diabetic retinopathy include decreased or fluctuating vision, presence of floaters (vitreous hemorrhage), or defects in the field of vision. Findings include microaneurysms (these are small outpouchings of the capillaries), retinal hemorrhages, hard exudates (lipids and proteins that seep into the retina), and infarcted retinal tissue (these are called cotton-wool spots). As the disease progresses, new blood vessels can develop on the surface of the retina (these are called neovascularization elsewhere or NVE) or on the surface of the optic nerve (disc neovascularization or NVD).

S - Subjective

A 43 year old with insulin-requiring diabetes presents with blurred vision over the past month. They noted that yesterday, they woke up with the appearance of a "cobweb" floating from the left eye. The feel it is swirling around in their vision.

O - Objective

The vision is recorded at 20/30 from the right eye and 20/400 from the left (affected) eye.

Examination of the vitreous showed significant hemorrhage in this cavity. The retina shows evidence of retinal ischemia (lack of oxygen to the tissue) with the presence of cotton-wool spots and retinal neovascularization as well as intraretinal hemorrhage (**Figure 13**).

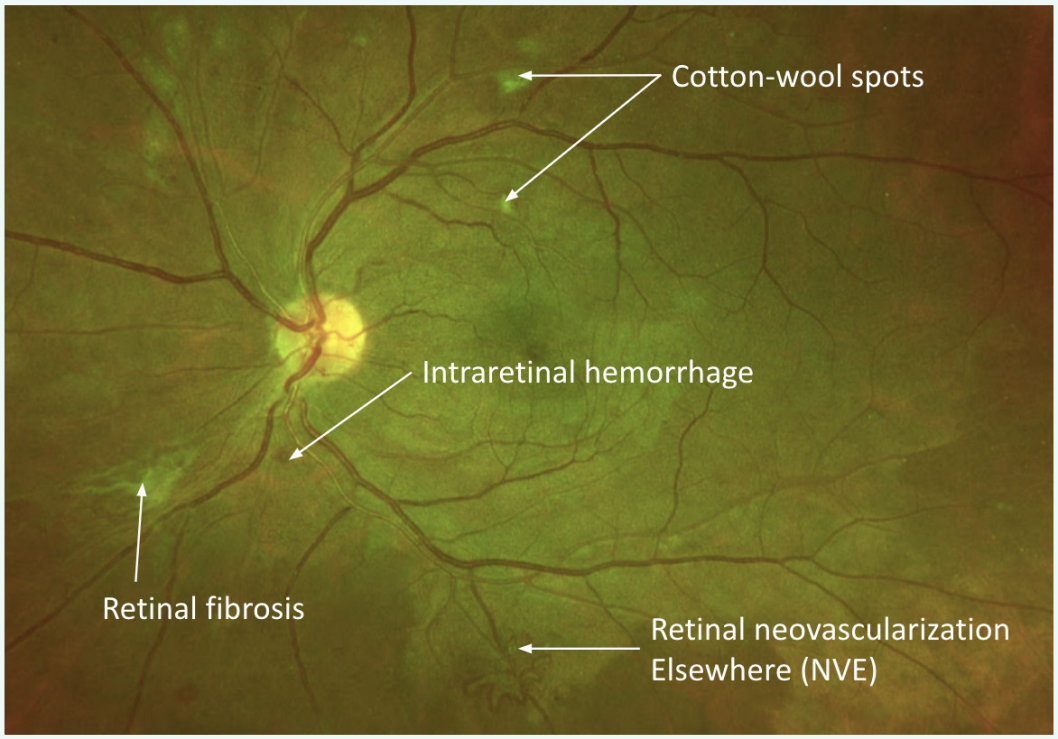


Figure 13: Hemorrhage and new blood vessels associated with diabetes

A - Assessment

Fluorescein angiography, a diagnostic test where a vegetable-based dye is injected into a vein in the arm, eventually making its way to the retina and then photographs of the retinal circulation are taken, helps determine the degree of ischemia and retinal vascular abnormalities.

In our patient, the angiogram shows the tiny microaneurysms light up like lightbulbs (**Figure 14**). In addition, the areas of NVE are associated with leakage and ischemia is visible (lack of retinal perfusion because of loss of the retinal capillary network).

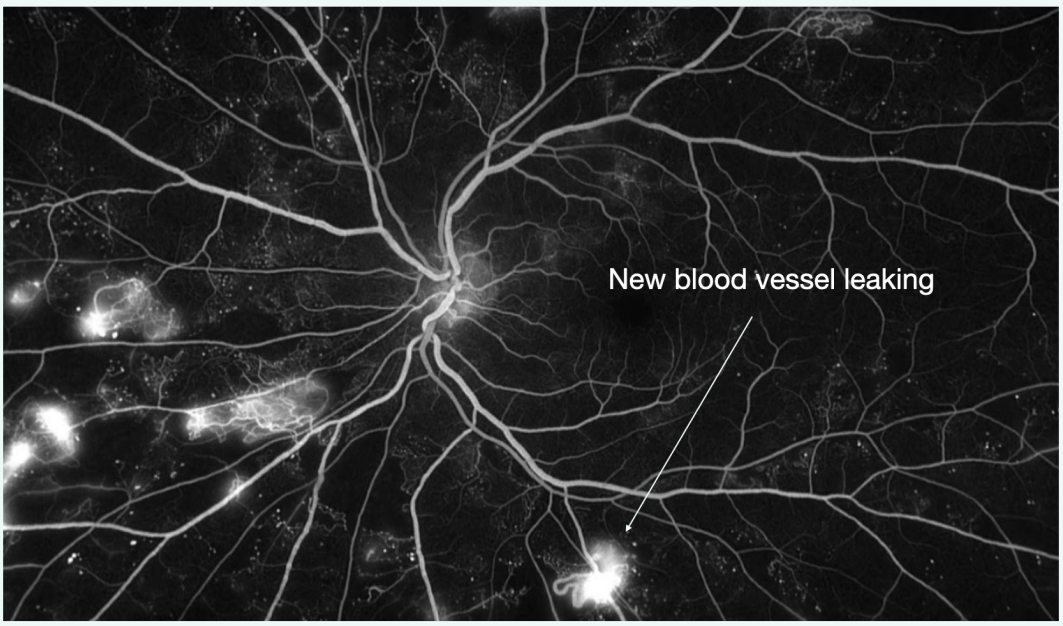


Figure 14: Angiogram confirming the presence of new blood vessels

P - Plan

Keeping blood sugars in the normal range is critical for diabetic patients, and will lower the risk of progression of eye disease.

Both lasers and eye injections are indicated for patients with leaking of fluid into the macula (diabetic macular edema) and the development of new blood vessels on the surface of the retina.

S - Science

Similar to patients with wet AMD, VEGF levels are very high in patients with diabetes. This causes new blood vessels to form. In addition, VEGF causes pre existing blood vessels to be leaky allowing lipids and proteins to enter the retina.

Here is the original paper that linked high VEGF levels in the vitreous to eye disease (most of the patients were diabetics and not AMD patients).

Important Points for FutureMDs to consider

- Any patient (diabetic or not) should be seen urgently if they develop new floaters to rule out a retinal tear
- New floaters in a diabetic is a vitreous hemorrhage until proven otherwise
- Newly diagnosed type 2 diabetics need to be screened for retinopathy at the time of diagnosis due to the high prevalence of retinal changes.

Background

All eyes have pressure. This is necessary because the cornea has to be a certain distance from the lens and retina so that proper refraction can take place (in fact in cases where the eye has no pressure, there is collapse and with the loss of this normal refraction system, vision is lost).

Intraocular eye pressure (IOP) is measured with instruments called tomometers; normally, eye pressure is below 21 mmHg.

To create pressure, fluid is secreted from the ciliary body, moves from through the pupil into the anterior chamber, and exits it through the trabecular meshwork. The trabecular meshwork can be thought of as the eye's drainage system and is locate in the angle that is created between the back of the cornea and the lens.

When a patient has glaucoma, it is important to understand if the angle is open or closed. Angle closure can occur if the base of the iris is blocking the trabecular meshwork (we call this angle closure glaucoma), if there are new blood vessels growing on or in the trabecular meshwork (neovascular glaucoma) or if there is scar tissue or membrane growing in this area.

Primary open angle glaucoma (POAG) is a leading cause of blindness that causes loss of the peripheral visual fields due to optic nerve head damage. Here the angle structure is open and nothing is visibly blocking the eye's drainage system.

The etiology of POAG is primarily related to raised intraocular pressure (IOP), although IOP-independent mechanisms may also exist (note: there are cases of low pressure glaucoma - so it is best to think of glaucoma as a condition of the optic nerve that is linked to high eye pressures in the vast majority of cases). Raised IOP occurs due reduced aqueous outflow caused by degeneration of the beams that make up the trabecular meshwork and the endothelial cells that line these beams.

Glaucoma causes loss of the inner retinal nerve layers (the ganglion cells and their axons which are seen in the nerve fiber layer). It is believed that with raised intraocular pressure, these forces are transmitted to the posterior vitreous/retinal interface. At the point where the nerve fiber layer of the retina makes a right angle turn to exit the optic cup and move into the orbit, these cells become susceptible to shearing. It is also possible that raised IOP can cause a reduction in perfusion pressure to the nerve head and that POAG may be a vascular disease.

IOP is certainly the most important risk factor for POAG, but other risk factors include increasing age, myopia, thin central corneal thickness (CCT), positive family history, diabetes, exercise, and diet. POAG is more prevalent with greater disease severity and treatment-resistance in individuals of African-Caribbean descent.

The disease is often asymptomatic until advanced stages. Patients are typically diagnosed as part of a routine eye test when signs of glaucomatous optic neuropathy are noted.

S - Subjective

A 68 year old presents because they notice that they are bumping into things, and are surprised that things are next to them that they did not see.

O - Objective

The patient's visual acuity is 20/20 from both eyes (this is a common finding as glaucoma affects the peripheral visual field. It is only in the latest stages of the condition that central visual acuity is lost).

Physical examination consists of assessment of IOP, anterior chamber angle (which should be open in POAG), optic disc, and visual fields. Signs suggestive of POAG include:

- An enlarged cup-to-disc ratio (usually greater than 0.5) - **Figure 15**
- Asymmetric cup-to-disc ratios between the two eyes
- A "notch" in the neuroretinal rim (this is the yellowish area of the disc which represents the axons of the retina as they make a right angle turn to exit the eye)

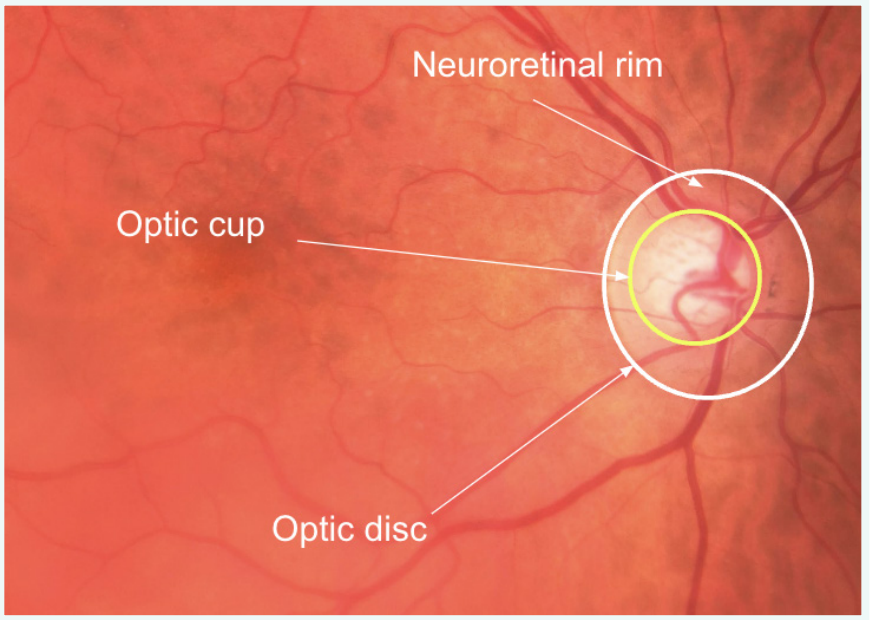


Figure 15: Enlarged cup to disc ratio in glaucoma

A - Assessment

Our patient presented for a routine assessment. The macula and retinal vasculature in the posterior pole is unremarkable. There is, however, an enlarged cup: disc ratio, which is estimated to be 0.7. There is thinning of the neuroretinal rim temporally, which appears as a "notch" at the 9 o'clock position. The blood vessels are noted to be nasally deviated and dive down deep under the neuroretinal rim. These are classic findings in POAG.

The patient's intraocular pressure was recorded at 29 mmHg.

Visual field testing shows areas of peripheral vision loss (**Figure 16**).

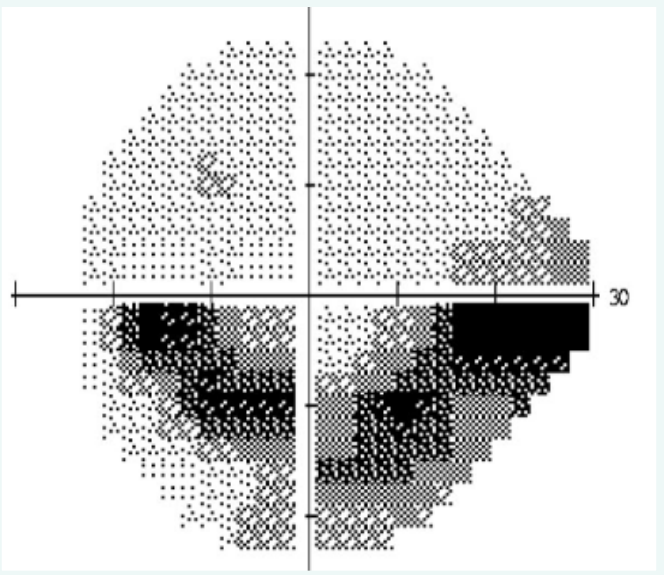


Figure 16: Black areas show visual field loss in a patient with POAG

Additionally, diagnosis of POAG is confirmed with OCT, which shows thinning of the inner retinal layers (nerve fiber layer). One of the earliest signs is the presence of thinning of this area. As the disease progresses, visual field can be lost (first there are subtle scotomas called "steps," and later there are large areas that are lost. At its end stage, POAG can cause total (no light perception) blindness.

P - Plan

Treatment modalities include drops to reduce the amount of aqueous production, others to open the angle. Lasers are also used to create holes in the trabecular meshwork or further open its beams. In addition, surgery is necessary in select cases.

S - Science

Numerous randomized clinical trials have shown the benefit of laser for the treatment of open angle glaucoma.

Here is a meta-analysis (a study that combines the results of numbers others) that showed a 29% reduction in disease progression when patients were treated with laser.

Here is what FutureMDs need to know about POAG

- Any patient over the age of 50 years needs an annual eye examination to rule out POAG
- Glaucoma patients need to take their eye drops daily, and to continue to see their eye doctors for regular monitoring as subtle changes in optic nerve head morphology and visual fields can take place without the development of symptoms.
- Glaucoma drops may cause redness of the eye and allergy; if anterior segment signs are noted, referral to an eye doctor is warranted.

Background

The retina has a dual blood supply. The inner retina is supplied by the central retinal artery, while the outer retina is supplied by the choroid which ultimately is perfused by the ciliary arteries. When the central retinal artery loses its perfusion, vision can be permanently lost within a matter of hours. In fact, monkey models of central retinal artery occlusion (CRAO) show irreversible damage occurring at 90 minutes following occlusion.

Patients with CRAO or branch retinal artery occlusion (BRAO) present with sudden monocular painless loss of vision. This can be mild or very profound (no light perception, in the case of an ophthalmic artery occlusion).

S - Subjective

An 83 year old presents with sudden, painless loss of vision. They are know to be a diabetic and have high blood pressure.

O - Objective

Visual acuity was 20/20 in their right eye. From the left eye they could not see letters on the eye chart, but could count fingers that were presented 1 foot from their eye (if people cannot see the eye chart, vision is quantified as finger counting, hand movement, light perception or no light perception).

On retinal examination, there is the presence of significant cloudy swelling of the retina (this appears as cloud-like swelling that is white in color) (**Figure 17**). This is related to increased inner retinal thickening because the axons of the ganglion cells are swollen. Visible emboli can sometimes be seen in the arteries and indicate that a lesion of the carotid artery or heart may be the cause.

Ocular stroke (called central or branch retinal artery occlusion) is associated with numerous medical conditions, including diabetes, hypertension, atrial fibrillation, cardiac valvular pathology and carotid artery stenosis.

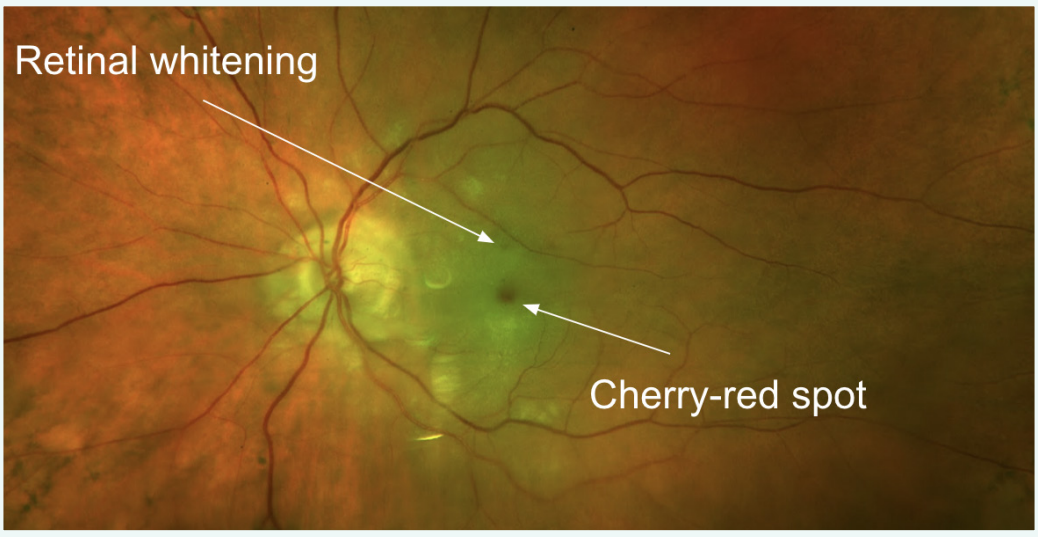


Figure 17: Cloudy swelling in our patient with ocular stroke

A- Assessment

An OCT in a patient with an ocular stroke will show inner retinal swelling or thickening (**Figure 18**). In addition, an angiogram will show reduced blood flow in the affected artery.

Systemic testing may show narrowing and reduced blood flow through the carotid artery (this is the main artery in the neck that branches off of the aorta and supplies blood to the retina). Echocardiogaphy (an ultrasound of the heart) may snow cardiac pathology like problems of the valves.

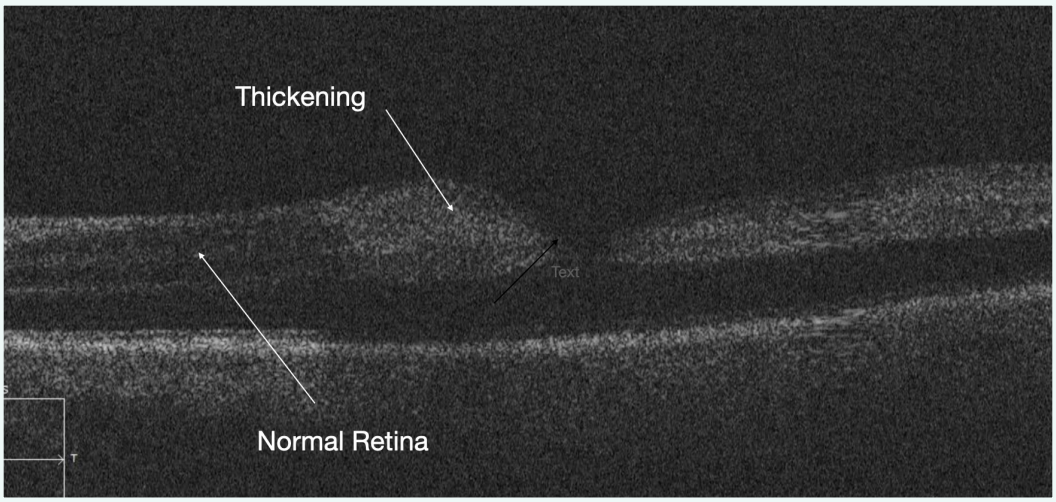


Figure 18: Swelling of the inner retina due to an ocular stroke

P - Plan

While the retina dies very quickly if blood supply is lost, putting a needle in the front of the eye and draining the anterior chamber of its fluid to rapidly lower intraocular pressure in an attempt to increase the "relative perfusion pressure" of the central retinal artery may be of benefit. In addition, intra-arterial TPA (cannulation of the ophthalmic artery) may be of benefit.

Systemic evaluation of a downstream source of embolic matter should also be performed, including both cardiac echocardiography and carotid doppler ultrasonography.

S - Science

I have done significant research in the area of ocular stroke. Here is a paper that shows that nearly 20% of people with ocular stroke had significant artery narrowing of the carotid artery.

Important things for FutureMDs to know about ocular stroke

- Any patient with sudden visual loss needs an emergency referral to rule out a retinal artery occlusion or retinal detachment (minutes matter for both of these)
- In elderly patients, an inflammatory vascular condition called giant cell arteritis, may be the cause of ocular stroke. If this condition is missed, it can cause death.
- Many cases are linked to carotid artery stenosis or cardiac valvular pathology

Background

The uvea is the vascular tissue in the eye. It consists of 4 parts:

- The choroid is located between the sclera and retina and provides oxygen and nutrients to the outer retina
- The pars plana is the transition zone between where the lens is located more anteriorly and where the retina is located, more posteriorly
- The ciliary body which produces the eye's aqueous humor is located in the middle of the eye (its additional function is to anchor the lens), and
- The iris, the colored part of the eye, which contains the musculature that controls the size of the pupil.

Uveitis occurs when any portion of the uvea is inflamed. Iritis occurs when when the iris is inflamed (iridocyclitis when both the iris and ciliary body are inflamed); pars planitis, when the pars plana is inflamed (here there are "snow ball" collection of WBCs located near the retinal periphery) and choroiditis occurs when the choroid is inflamed (which is often linked to a retinal detachment).

Symptoms of iritis/iridocyclitis include acute onset pain, redness, and photophobia with variably blurry vision. Tearing, lid puffiness, and some drooping of the eyelid may also be present. Anterior uveitis may occur as a single episode, which resolves with anti-inflammatory therapy; recurrent episodes, which occur after at least three months of treatment cessation and symptom resolution; or chronic disease, in which symptoms recur after medication is tapered or stopped.

Acute anterior uveitis (AAU) may occur in isolation, or in association with systemic infection (such as herpes simplex or syphilis) and/or inflammation (such as arthritis or inflammatory bowel diseases). Systemic inflammatory conditions may be associated with the HLA-B27 genotype, particularly if your patient also has joint and/or bowel symptoms depending on the underlying disease process.

In order to establish a diagnosis of AAU, there must be white blood cells in the anterior chamber visible by slit lamp examination. Classically, the eye is red with the presence of a ciliary flush as the deeper vessels around the limbus (corneoscleral junction) are injected (note- in a case of conjunctivitis a ciliary flush will not be present).

Keratic precipitates, flare (here cells are not visible, but there is a haziness like a light in the fog, and is related to a higher specific gravity of the fluid circulating in the aqueous), hypopyon (WBCs layering out in the bottom of the anterior chamber), iris nodules, and posterior synechiae (scarring of the pupil to the lens). Patients must also undergo a dilated exam to assess for posterior uveal inflammation as well. Evaluation of intraocular pressure and gonioscopy should also be performed.

S - Subjective

A 34-year-old patient presents with sudden painful loss of vision. They also note significant sensitivity and pain when in bright light. The patient is known to have a systemic inflammatory condition called sarcoidosis.

O - Objective

The patient's visual acuity is 20/60 from both eyes.

On examination, the corneal light reflex was dull and the cornea was thickened. Folds in Descemet's membrane (one of the deeper layers of the cornea) are seen. A hypopyon (layering of white blood cells in the anterior chamber) is visible (**Figure 19**). The patient also had a ciliary flush (vascular injection of the sclera just next to the iris). This patient was diagnosed with iritis or anterior uveitis.

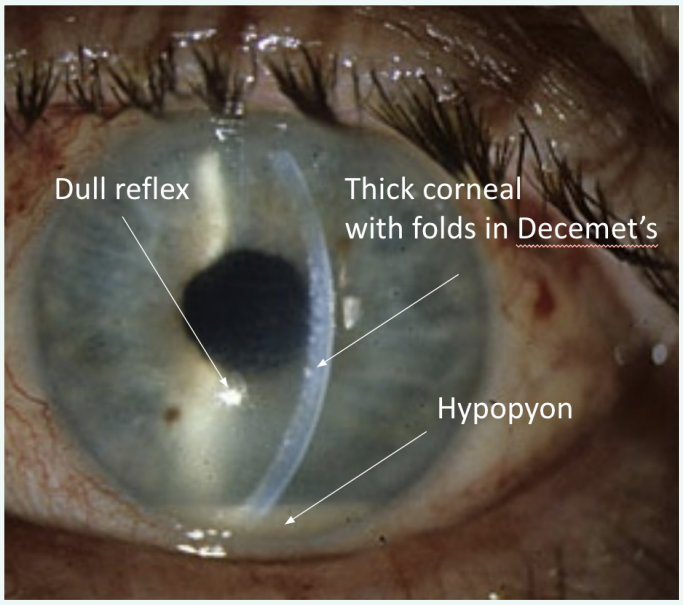


Figure 19: Hypopyon in our patient with iritis

P - Plan

Management of iritis comprises topical corticosteroids drops with or without a dilating agent (which helps to reduce pain and prevent development of posterior synechiae). It is important to elucidate the cause of iritis to guide treatment of the underlying cause, particularly in cases of infection or systemic inflammatory conditions.

S - Science

This research paper shows that 73% of cases of uveitis is associated with systemic disease, the most frequent of which is sarcoidosis.

Important Points for FutureMDs to know about iritis/AAU

- Urgently refer any patient with a ciliary flush to rule out the possibility of AAU (it may also be present in a corneal problem like an ulcer or angle closure glaucoma)
- In a patient who has AAU and no known systemic disease, consider a thorough history and guided investigations to rule out common systemic diseases including arthritis, sarcoidosis and inflammatory bowel disease.
- Steroid drops should be discarded when inflammation is not active as many people will erroneously use them for ocular dryness or discomfort. Steroid use can be associated with cataract formation and glaucoma.

Background

Cranial nerves are similar to peripheral nerves, except instead or arising from the spinal cord, they exit from the brain. There are 12 cranial nerves, 3 of which control the movement of the eyes:

- The 3rd cranial nerve moves the eye upwards, downwards and inwards
- The 4th cranial nerve, which moves the eye downward and inwards and
- The 6th cranial nerve which moves the eye outwards.

The 3rd cranial (ocolomotor) nerve is a large nerve that innervates many of the eye's muscles, including:

- The levator muscle which is responsible for elevating the upper lid
- The superior rectus which elevates the eye
- The inferior rectus which depresses the eye
- The inferior oblique which torts the eye outward, and
- The medial rectus which moves the eye inward.

In addition, the 3rd nerve also contains sympathetic fibers. These are located on the outer part of the nerve. It also provides innervation of the eye's internal muscles, which are responsible for accommodation.

While the nucleus of the oculomotor nerve is located in the midbrain, the nerve exits the anterior brainstem between the superior cerebellar and the posterior cerebral arteries. The nerve then enters the cavernous sinus and exits into the orbit in 2 branches (the superior and inferior branches) through the superior orbital fissure.

Patients with a 3rd nerve palsy present with a droopy lid, and the inability to move the eye in multiple directions (horizontal diplopia or double vision, if the medial rectus is affected, and vertical diplopia if the superior or inferior rectus are affected).

Clinically, it is very important to distinguish between a "pupillary-involving" from a "pupillary-sparing" third nerve palsy. The former represents a diagnostic emergency as it is often linked to serious intracranial pathology including a ruptured posterior communicating artery aneurysm, and vascular imaging is urgently needed.

In any patient with a 3rd nerve palsy, it is important that multiple nerve palsies be ruled out as it may be seen in conjunction with 4th, 6th and 5th nerve palsies. Multiple nerve palsies need to have pathology in the cavernous sinus ruled out.

S - Subjective

The patient presents with a new onset of double vision which is noted to be both horizontal and vertical.

O - Objective

This patient had normal vision but presented with a left ptosis. In addition, the left eye fails to elevate or turn inward (**Figure 20** - here the patient is asked to look up and to their left). The eye, however, was able to move outward. The pupils were also dilated by 2 mm.

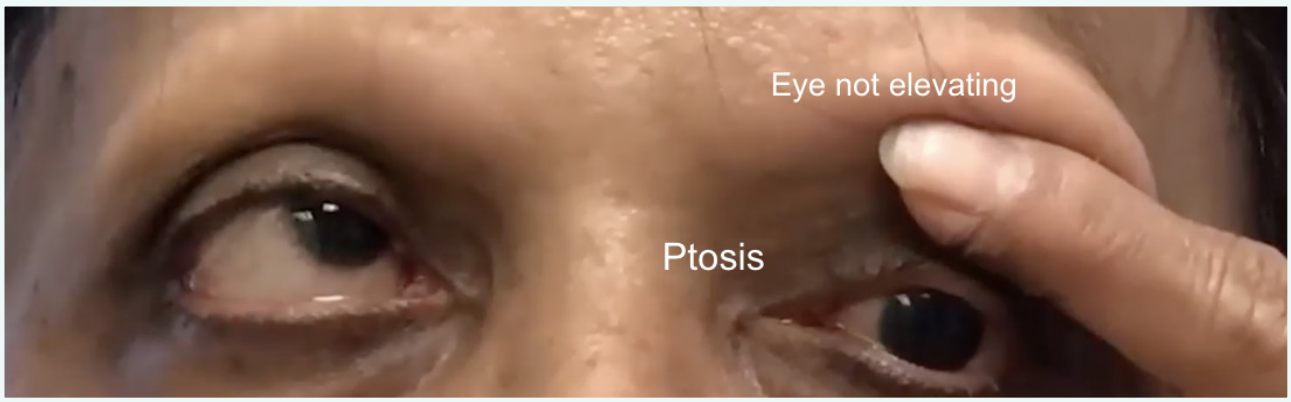


Figure 20: Finger elevating droopy lid; patient asked to look up

A - Assessment

The patient had an urgent MRI of the brain performed which confirmed the presence of a cerebral artery aneurysm.

P - Plan

Significant intracranial pathology is linked to new onset double vision (as can other conditions like thyroid disease). This patient was urgently referred for brain surgery to clip their aneurysm.

S - Science

Closed head injuries are common in high school students. These can sometimes be associated with cranial nerve palsies. Here is a paper showing that patients with closed head injury that also had a cranial nerve palsy had a much higher risk of having a cranial nerve palsy.

Here is a link to this paper.

Important points for FutureMDs to know about 3rd cranial nerve palsies

- A pupillary- involving 3rd nerve palsy is an emergency and need imaging to rule out intracranial pathology
- In any 3rd nerve palsy, always rule out multiple palsies as this may represent a cavernous sinus lesion
- In any elderly patient with multiple neuropathies, ask about constitutional signs as it may represent giant call arteritis.

Background

The retina is a 10 layered tissue at the back of the eye. At its deepest level, the photoreceptors, made up of rods and cones, interdigitate with the terminal projections of the retinal pigment epithelial (RPE) cells. There is a plane between the ends of the photoreceptors and the projections of the RPE cells. Fluid can gain access to this space (called subretinal fluid) and cause it to significantly increase in size.

There are 3 different types of retinal detachment (RD):

- Rhegmatogenous (RRD). Here fluid from the vitreous moves through a hole or tear in the retina and gets access to the subretinal plane. As this fluid increases in amount it can cause the retina to become very elevated from the underlying RPE.

- Serous or Exudative. Here a problem such as a deeper choroidal tumor (like a choroidal melanoma) or inflammation of the choroid can cause fluid to move through the RPE and seep into the subretinal space.

- Tractions (TRD). Here a process on the surface of the retina (such as growth of a membrane from proliferative retinopathy) can cause the retina to become pulled up.

The vast majority of retinal detachments are of the rhegmatogenous type. The pathogenesis typically involves separation of the vitreous from the retina (which is usually an age-related event). Because the retina is strongly adherent to the retina in the retinal periphery, when the vitreous falls forward, it can cause a retinal tear. While most patients with a posterior vitreous detachment (PVD) do not have a retinal tear, this complication occurs in approximately 10-15% of cases.

The principles of retinal detachment include re-approximating the protoreceptors to the RPE cells and sealing off the hole. Re-approximation can occur with the injection of gas into the eye (pneumatic retinopexy), vitrectomy, or by sewing on a scleral buckle to the external part of the eye to move the sclera more anteriorly.

S - Subjective

This patient presented with sudden painless loss of vision. They noted that it appears like there is a dark area inferiorly. Two weeks earlier, the patient noted a new shower of floaters or cobwebs from that eye. They also noted flashes of light from that eye.

O - Objective

The patient's vision was 20/30 from both eyes. Hemorrhage was seen in the vitreous. Peripheral retinal examination showed a tear in the retina with significant fluid under the retina (**Figure 21**).

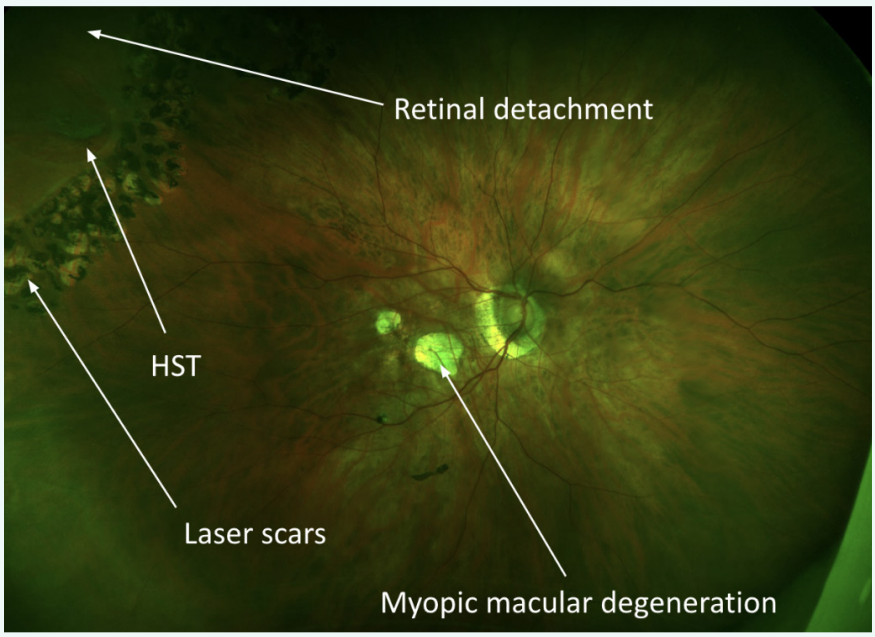


Figure 21: Retinal tear and detachment with laser scars surrounding it

A - Assessment

Most retinal tears and detachment will not need additional testing. However, at times, there is associated hemorrhage in the vitreous which precludes thorough examination of the retina. In these situations, ultrasound may be beneficial to be able to rule out the presence of a retinal detachment.

P - Plan

Retinal tear is managed by using laser to spot weld around the retinal hole to prevent fluid from seeping under it.

If there is a significant retinal detachment, gas can be injected into the eye to tamponade the retina down or an operation to the inside or outside of the eye may be required.

S - Science

We published a study in JAMA that showed that 14% of people with new-onset of flashes and floaters have an associated retinal tear or detachment. Here is a link to that article.

Important points for FutureMDs to know about retinal tears

- In a patient with visual loss and preceding floaters and flashing lights, retinal tear or detachment needs to be ruled out
- Retinal detachment is an emergency, as vision can be permanently lost within hours
- 14% of patients with new onset flashes or floaters will have a retinal tear or detachment.

Background

Cataract refers to opacification of the eye's native lens. It can be classified by the location of the opacification:

- cortical, if there is clouding of the outer portion of the lens (cortex),
- nuclear, if the center of the lens is affected or
- posterior subcapsular, the layer between the posterior cortex and the posterior portion of the capsule.

The vast majority of age-related cataracts are related to nuclear sclerosis. However, in those who are on steroids for uveitis or are diabetic, the posterior subcapsular type is more common.

The treatment of cataract involves surgery. Modern cataract surgery involves the use of phacoemulsification, a procedure in which the lens is broken up with high frequency sound waves and the fragments are aspirated with suction. Typically, once the cataract is removed, an intraocular lens is placed within the remaining capsular bag. The indication for surgery is based on the ability of the patient to perform their activities of daily living and is driven by the desire to maximize their quality-of-life.

While cataract surgery has a very high success rate, complications include rupture or the capsule, retinal swelling in the macular area, and infection. Any patient with a painful, red eye with worsening vision post surgery needs emergent referral to rule out endophthalmitis - a potentially blinding infection (most frequently related to gram positive Staph bacteria from the patient's ocular surface entering the eye at the time of surgery).

While surgery almost universally results in excellent vision, a "secondary" cataract may develop with time (months or even years after the initial operation). Here cortical cells that were initially left at the time of surgery can grow on the surface of the posterior capsule leading to opacification of this tissue (called posterior capsular opacification or PCO). To treat this condition, in-office laser needs to be applied to create an opening in the capsule such that light can now move through this plane unencumbered.

S - Subjective

This patient noted blurring of their vision over the past 6 months. They also noted "haloes" around car lights while driving at night.

O - Objective

This patient presented to us with reduced vision 20/100.

Slit-lamp examination showed some cortical opacification (whitish material) and a very brunescent nucleus (brownish-orange) - **Figure 22**.

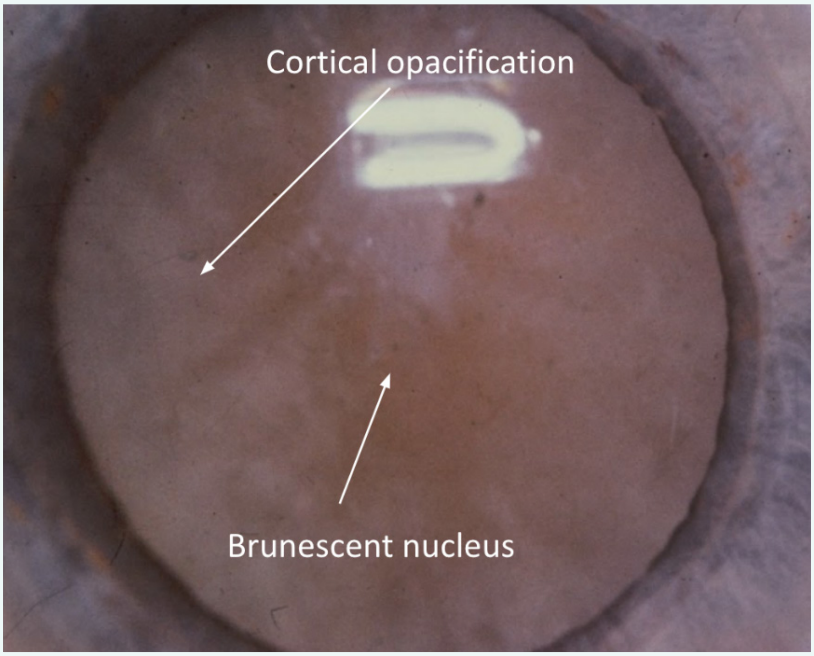


Figure 22: A brunescent cataract needing surgery

A - Assessment

Every cataract needs a thorough examination because an eye can have a tumor or retinal detachment. Both of these should not be operated on. Because of this if an cataract is very dense, an ultrasound test should be performed to rule out the possibility of either of these 2 conditions.

Furthermore, ultrasound is also used to measure the dimensions of the eye as this variable is used in the calculation of the power of lens that will be inserted into the eye at time of surgery.

P - Plan

The patient was diagnosed with a cataract and phacoemulsification and with IOL insertion was performed.

S - Science

Here is a nice review of the physics formulas that are relevant for calculating the power of an intraocular lens.

Important Points for FutureMDs to know about cataract

- Indications for cataract surgery are based on the effect on quality of life as opposed to the "ripeness" of the lens
- Cataract uses ultrasound to break up the cataract and the insertion of an intraocular lens (the power of which uses your physics equations!)
- Any patient who presents with pain and worsening of vision after cataract surgery needs emergent referral as it may represent endophthalmitis (infection of the inner coats of the eye centering around the vitreous), a potentially blinding complication

Background

The cornea consists of 5 layers: the surface epithelium, its basement membrane called Bowman's layer, the stroma, the endothelial layer, and its basement membrane (which is located between the stroma and the endothelium).

A corneal ulcer refers to a breakdown of the epithelial layer with an associated infiltrate. There are numerous causes of a corneal ulcer ranging from infectious and non-infectious etiologies. Common causes include bacterial infections (mostly from Staph Aureus), or viral (from Herpes Simplex or Varicella Zoster). Less commonly, fungal and parasitic infections occur. Non-infectious etiologies can be causes by problems in the branches of the facial nerve, and are called neurotrophic ulcers.

Patients with corneal ulcers typically present with a red eye which is very painful. On examination, vision can be reduced and a visible ulcer (absent epithelium, loss of corneal stroma and infiltration) is present.

Corneal scrapings are usually performed and the material will be analyzes cellular activity and plated for growth of bacteria. One of the pathogens with is linked to poor contact lens hygiene, Acanthamoeba, a parasite, may warrant corneal biopsy, looking for intrastromal cysts.

S - Subjective

A contact lens wearing patient presents with a painful eye and poor vision over the past month. They have been sleeping with their contact lens in the eye for the past week.

O - Objective

This patient has light perception only from this eye.

On examination, a visible corneal ulcer is present. In addition, there is significant ocular redness; a hypopyon is seen in the anterior chamber as WBCs are layering out (**Figure 23**).

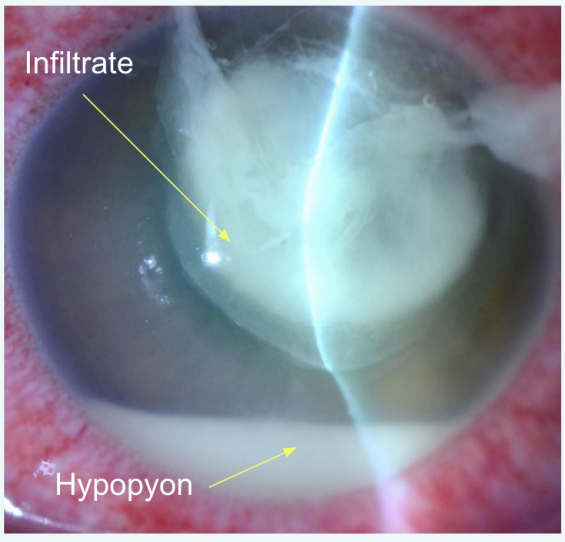


Figure 23: A corneal ulcer

A - Assessment

Corneal swabs and scraping was performed which grew a gram positive bacteria: Staphylococcus Aureus.

P - Plan

The patient was treated with topical antibiotics.

Many corneal ulcers can be adequately treated with topical antibiotics. Some however, will eventually require a corneal transplant.

A known risk factor for corneal ulcer is contact lens use, especially with poor hygiene.

S - Science

A paper published in JAMA showed that overnight wear of contact lenses increases the risk of corneal ulcer by 13 times. Here is a link to the paper.

Important concepts for FutureMDs to know

- Any patient with a red painful eye warrants emergent assessment for corneal ulcer (and angle closure glaucoma)
- Contact lens patients are at higher risk of corneal ulceration (those who sleep with their contact lenses are at a 13-fold higher risk)
- Contact lens use with swimming, taking a sauna or cleaning with tap water are at much higher risk of Acanthamoeba infection