GLAUCOMA

• Leading cause of irreversible blindness in the world
• Glaucoma is an optic neuropathy associated with an elevated intraocular pressure (in the anterior chamber)
• Optic nerve damage leads to progressive retinal ganglion axonal cell loss. This loss of cells leads to progressive visual field loss resulting in irreversible blindness when untreated.
• 4 types:
  o *Acute angle closure glaucoma (narrow angle glaucoma) – see below for more information
  o Secondary glaucoma – secondary to a different ocular pathology
  o Congenital glaucoma
  o *Primary open angle glaucoma (wide angle glaucoma) – see below for more information
(*Will be explained in this handout)

• Primary open angle glaucoma (POAG) is the most common type of glaucoma.
  o Develop peripheral visual field loss followed by central field loss
  o Intraocular pressure (IOP) may or may not be elevated. In most cases, it is usually elevated. It was once considered necessary for IOP to be elevated in order to be given the diagnosis of glaucoma. However, newer studies indicate that POAG can be present even in the context of normal IOP, and that an elevated IOP is neither necessary nor sufficient for the diagnosis of glaucoma.
  ▪ Normal IOP is 8-22 mmHg
  ▪ Measure the IOP by using a digital instrument called a Tonopen – uses ultrasound to measure the pressure within the anterior chamber
  o On fundoscopic exam, the optic disc appears to take on a hollowed-out appearance – this is described as “cupping”. This is due to the loss of ganglion cell axons.
  o This type of glaucoma is asymptomatic and must be screened for and confirmed on comprehensive ophthalmic exams
  ▪ Primary care physicians can screen by using visual field testing and fundoscopic exam
  o Visual field loss is irreversible
  o Risk factors: elevated IOP, advanced age, black race, and family history
  o Pathogenesis of POAG is not known
  o Treatment – lowering the IOP is the primary modality for therapy of POAG
    ▪ Topical medications are used to either increase aqueous outflow or by decreasing aqueous production
    ▪ Agents that increase aqueous flow:
      • Prostaglandin analogues, alpha-adrenergic agonists, cholinergic agonists
    ▪ Agents that decrease aqueous production:
      • Beta adrenergic blockers, alpha adrenergic agonists, carbonic anhydrase inhibitors
    ▪ Laser and surgical therapies are also available – these act to increase aqueous flow
      • Both of these act to change the trabecular meshwork of the eye to allow better flow of the aqueous into the Canal of Schlemm (the trabecular meshwork is a fenestrated layer anterior to the iris which allows the aqueous fluid to flow to the Canal of Schlemm and into the venous system). See pictures below.

• Acute angle closure glaucoma – relatively uncommon
  o Considered an ophthalmologic emergency
Acute closure of the angle blocks the Canal of Schlemm leading to blockage of the aqueous fluid from entering the venous system. This results in an elevated IOP.

The presenting patient will appear to be in distress, often complaining of headache and malaise. With the increased IOP, the patient can develop nausea and vomiting. The pain of the angle closure is usually described as a dull ache, and is often confused for a unilateral headache. The affected eye will appear to be red, and this should warrant assessment of the eye as the source of the patient’s presenting symptoms. The patient may also complain of photophobia.

As the duration of the attack increases, visual acuity progressively reduces.

On examination of the pupils – the affected pupil is fixed in mid-dilation (~4-5mm) and does not react to light.

Treatment involves emergent lowering of the IOP. Topical pupillary constrictors are agents of choice.

**Angle anatomy**

The angle is the recess formed by the irido-corneal juncture. The scleral spur, trabecular meshwork, and Schwalbe's line lie within this angle. The trabecular meshwork is a fenestrated structure that transmits aqueous fluid to Schlemm's canal, from which it drains into the venous system. The normal flow of aqueous is demonstrated here.

**Acute angle-closure glaucoma**
The iris root occludes the trabecular meshwork completely obstructing drainage of aqueous fluid from the anterior chamber. The resulting rapid elevation of intraocular pressure requires urgent intervention to prevent permanent visual loss.

**Assessment of the optic disc in healthy and glaucomatous eyes**

A: Optic nerve photography: small central cup in healthy eye; enlarged cup and loss of inferotemporal neuroretinal rim in glaucomatous eye; B: Retinal nerve fibre layer photography: uniform reflections in healthy eye; poor reflections in inferotemporal region (arrows) in glaucomatous eye.

**AGE-RELATED MACULAR DEGENERATION (ARMD)**
- Classified as either dry (atrophic) or wet (neovascular or exudative)
- Risk factors: advanced age, smoking, family history, hypertension, ethnicity (more prevalent in non-Hispanic whites than in African Americans or Mexican-Americans), BMI (positive correlation between BMI and the prevalence of ARMD), diet (fat intake is associated with increased risk of ARMD), previous cataract surgery, chlamyphila pneumoniae infection
- Clinical Presentation:
  - Dry type – complains of gradual loss of vision in one or both eyes.
  - Wet type – complains of an acute distortion of vision, especially of straight lines, or loss of central vision. Symptoms usually present in just one eye, but the disease will be present in both.
  - Distortion of straight lines is characteristic for ARMD and is what sets it apart from other causes of vision loss in the elderly.
- Dry ARMD – several proposed mechanisms, but pathogenesis remains unclear
  - Vision loss tends to be slow and gradual in nature
On fundoscopic exam may see drusen deposits (deposits of extracellular material concentrated in the macula), chorioretinal atrophy (areas of thinning and loss of tissue in either a focal or more geographic pattern in the macula), or disturbance of the retinal pigment epithelium with development of retinal pigment epithelium mottling beneath the retina. (Below are pictures of these findings)

Treatment: antioxidants and zinc (proposed to prevent cellular damage in the retina); laser therapy (still undergoing clinical trials to prove efficacy of this treatment modality).

• Wet ARMD – aka exudative or neovascular. More common than dry type.
  o Characterized by abnormal growth of vessels from the choroidal and retinal circulations (retinal is less frequent) into the subretinal space. These vessels are leaky which can result in exudative retinal detachment and/or hemorrhage under the retina.
  o Characterized by a rapid onset of distortion and loss of central vision. Onset of symptoms is on the order of weeks to months.
  o Pathogenesis:
    ▪ Abnormal activity of VEGF (vascular endothelial growth factor). Vascular growth is normally controlled by a dynamic balance between substances that promote and inhibit vessel development. In wet ARMD, VEGF is overactive, and thus leads to promotion of vessel growth.
    ▪ Genetics also play a role in development of wet ARMD.
  o Treatment: VEGF inhibitors (injected into the vitreous of the eye); thermal laser photocoagulation (does not restore lost vision, and can cause damage to overlying retina which can cause a permanent blind spot); surgical options also available.

Normal retina
Dry type ARMD with drusen

Small, bright yellow drusen are visible.

Dry type ARMD with drusen and chorioretinal atrophy

The bright yellow drusen are coalescing, and a large area of atrophy is seen in the macula (arrow).

Wet type ARMD with subretinal hemorrhage

A large area of subretinal hemorrhage is present.

Wet type ARMD with neovascularization

An area of neovascularization appears as a grayish discoloration in the macular area (arrow). There is also some lipid exudate visible.
CATARACTS

• An opacity of the lens of the eye resulting in partial or total loss of vision.
• Can be congenital or acquired (will only discuss acquired, specifically age-related cataracts)
• Most cataracts occur in patients ≥ 60y/o and who have risk factors (see below)
• Pathogenesis:
  o The lens of the eye is composed of specialized cells that are arranged in a highly ordered complex manner. These cells are stratified epithelia. However, unlike other epithelia, the dead cells of the lens are not shed. As a result, the lens is quite susceptible to the effects of aging.
• Risk factors:
  o Advanced age; Smoking; Alcohol consumption; Sunlight exposure; Low education (no obvious biological link); Diabetes mellitus; Systemic corticosteroid use
• Clinical Presentation:
  o Painless, progressive process; highly variable process among individuals
  o Typically bilateral, but often asymmetrical
  o Complain of difficulty with night driving/vision, reading road signs, and/or difficulty with fine print
• On fundoscopic examination will see a diminished or completely lost red reflex. A cataract that still allows some of the red reflex to be seen, is called an immature cataract; once the red reflex is entirely lost, it is called a mature cataract.
• Treatment – Surgery for removal of the cataract and implantation of an intraocular lens

All pictures were taken from UpToDate.
For further information, look up in UpToDate.