Glaucoma

Glaucoma is a chronic disease characterized by damage to the optic nerve, and by progressive loss of visual field. It usually affects both eyes, and it is a common cause of blindness. There are many types of glaucoma, but eachtype can be classified as either open angle or closed angle.

Closed angle glaucoma is always associated with rising intraocular pressure. When the eye pressure rises suddenly (acute angle closure glaucoma) there is pain and impaired vision. **Chronic** angle closure glaucoma occurs when the angle is slowly closed off. It may be painless because the intraocular pressure rises slowly. Angle closure glaucoma may be **primary** (not associated with other eye diseases), or secondary (caused by systemic or ocular diseases such as diabetes, carotid artery occlusion, retinal vein occlusion, or inflammation in the eye).

Whether the angle is open or closed is determined by temporarily placing a special lens (goniolens) on the cornea. This allows the ophthalmologist to examine the trabecular meshwork located at the intersection of the iris (colored part of the eye) and sclera (white part of the eye wall). If the trabecular meshwork, which acts as a filter, is visible and not scarred or damaged, the angle is considered "open." If the filter is blocked, or not visible, then it is "closed." Angle closure glaucoma must be treated. Most of the time a surgical procedure is required, either by using an office laser to make a hole in the iris **(iridotomy)**, or by performing filtration surgery in the operating room.

Open angle glaucoma is much more common. It comprises 80-90% of all cases. There are many subtypes within this category, but most patients have primary open angle glaucoma, or **POAG**, a condition in which the eyes appear normal except for damage to the optic nerve. POAG usually doesn't occur until middle age, although some patients, especially those with a strong family history of glaucoma, will develop it at an earlier age. People with certain ocular characteristics, such as: higher intraocular pressure, thinner central cornea thickness, increased cup-to-disc ratio, or asymmetric cup-to-disc ratio, are at increased risk for POAG. African-Americans and people of Hispanic/Latino heritage develop glaucoma three to five times more often then Caucasians. Patients with diabetes mellitus and myopia (near sightedness) also seem to have a higher risk.

POAG is an insidious disease since it does not cause symptoms until the optic nerve is severely damaged. Patients may not develop symptoms until nearly 80 percent of the nerve fiber layer [retinal cells that send signals to the brain] has been lost. When enough of these cells dysfunction or die, patients will notice defects in their peripheral vision. Because damage to the peripheral vision is irreversible, early detection and treatment is imperative.

Glaucoma can not be detected by laboratory testing; it is diagnosed in the medical office through clinical examination and testing. These tests include measurement of the intraocular pressure and the nerve fiber layer thickness, a dilated examination of the optic nerve looking for characteristic changes, and testing of the peripheralvisual field.

Intraocular pressure (IOP) is an important risk factor for developing POAG. IOP normally measures between 10 and 21 millimeters/Hg. Because it fluctuates throughout the day and night, it needs to be measured at different times. In healthy eyes, the day time IOP (diurnal pressure) may fluctuate by 6 mm/Hg. The IOP in patients affected by or at risk for glaucoma may fluctuate by 10 or more mm/Hg. The IOP also fluctuates at night (nocturnal IOP) with the peak IOP for a 24 hour period often occurring during sleep. Studies have shown that one way to approximate nocturnal eye pressure is to measure the **supine IOP** in the office. This is done by measuring the IOP after the patient has been lying flat in a dark room for 5 minutes. The higher the IOP and the wider the range of IOP the more likely glaucoma will develop. This is why it is important to check the IOP frequently and at different times of the day before

making a treatment decision. A single measurement will not suffice because most patients with glaucoma will have IOP in the normal range for at least part of the day.

Glaucomatous optic nerves have a characteristic appearance. Unlike other optic neuropathies, the supporting tissue within the optic nerve has either been destroyed or it has collapsed. Detailed drawings and photographs of the optic nerves are made to detect the subtle changes that occur over time. Loss of nerve fiber layer is one of the earliest signs of glaucoma. It is identified by using red-free light at the slit lamp, or by measuring its thickness by using instruments like Optical Coherence Tomography (OCT). OCT is a non-invasive test that bounces light off the retina and measures the thickness of the nerve fiber layer near the optic nerve. Serial comparisons are used to detect change.

Visual field tests , like Humphrey Visual Field Perimetry (HVF), are used to detect damage to the peripheral field. In this test, the patient is asked to identify various intensities of light. Each eye is tested separately and the results are compared to results from control groups. Based on standard analysis, most patients do not show HVF defects until 40 percent of the nerve fiber layer has been lost.

Short Wave Automated Perimetry (SWAP) is used when the HVF is normal. It measures the response to light from a small subset of cells, that are the first to be damaged by glaucoma, and may identify visual field loss two to four years before it shows up on the HVF.

Damage from glaucoma is permanent but early detection and prompt treatment can minimize vision loss. All treatment is directed at lowering IOP. We suspect there are other mechanisms causing damage to the optic nerve, but so far we have not found a way to counteract their adverse effects. IOP is lowered by using medicines, laser, or incisional surgery. **Medications (eye drops)** may be used in combination because they have different mechanisms of action. Beta-blockers (Timoptic®, Istalol®, or Betagan®) and prostaglandin analogs (Lumigan®, Travatan®, or Xalatan®) are most often used, but alpha2 adrenergic agonists (Alphagan®), oral, or topical carbonic anhydrase inhibitors (Azopt, Trusopt®, or Diamox Sequels®) and parasympathomimetics (Pilocarpine) have important roles. Medications may have side effects and are not always effective. **Laser treatment** to the trabecular meshwork (laser trabeculoplasty) is a first or second line treatment that is often very effective. It is done in the office and has few adverse effects. In this country, medications and laser are usually tried before incisional surgery. **Glaucoma filtration surgery** is performed in the operating room as outpatient surgery under local anesthesia. It is usually effective although like all surgeries, it carries a risk of complication.

The type of treatment used depends on the type of glaucoma and the severity of the disease. No matter which treatment modality is chosen, close follow up with frequent testing is necessary to minimize damage to the optic nerve that can lead to irreversible vision loss.

In summary, glaucoma is a clinical diagnosis that requires life long follow-up. There are 4 important parameters used to detect glaucoma or monitor its progression. They are IOP, optic nerve appearance, nerve fiber layer thickness, and visual field loss.