**Glaucoma**

What is glaucoma? Glaucoma is defined as a group of diseases that cause a characteristic change in the optic nerve with associated loss of visual field/function. More than fifty different causes of glaucoma have been described. Traditionally, glaucomas are classified based on the appearance of the drainage system within the eye, also known as the anterior chamber “angle”. Below, some of the more common types of glaucoma are discussed.

Primary Open-Angle Glaucoma



Primary Open Angle Glaucoma (POAG), also known as chronic open angle glaucoma, is the most frequent type of human glaucoma. It is characterized by elevated intraocular pressure (IOP), cupping and atrophy of the optic nerve head, and typical visual field defects. By definition, in “primary” glaucomas there are no specific ocular abnormalities or systemic diseases causing the glaucoma. Furthermore, [gonioscopy](https://glaucomaassociates.com/glaucoma-tests-and-diagnosis/gonioscopy/) of eyes with POAG reveals a normal appearing anterior chamber angle without any obvious abnormalities.

Both eyes tend to be involved at the same time and to a similar degree. The prevalence of POAG in the United States is estimated to be between 1.5% – 2%, with most cases detected after age 40.

Although the cause of POAG is unknown, there are a number of known risk factors including: increased intraocular pressure, advanced age, racial background (African and Hispanic ancestry), decreased corneal thickness, and a positive family history. Evidence also suggests that diabetes and myopia (near sightedness) are also risk factors but to a lesser degree. There is no gender predilection for glaucoma.

Normal Tension Glaucoma (Low Tension Glaucoma)

Normal tension glaucoma (NTG), sometimes referred to as low tension glaucoma (LTG), is often considered to be on a continuum with POAG, with very similar characteristics. While there are a number of subtle differences between POAG and NTG, there is one key difference compared with POAG: eyes with NTG do not have an elevated intraocular pressure as classically defined (usually defined to be greater than 21mmHg).

Treatment for patients with NTG is the same as for all types of glaucoma, namely, lowering the intraocular pressure. Studies have demonstrated that although the eye pressure is not elevated by the normal definition, further lowering of the pressure does decrease the risk of optic nerve damage and visual field loss.

Acute Angle-Closure Glaucoma

Acute angle-closure glaucoma is a dramatic disorder of sudden onset and represents a true ophthalmic emergency. If not properly diagnosed and treated, progressive and permanent ocular damage occurs in a matter of hours to days. The history is characterized by the sudden onset of pain, redness of the eye, and blurred vision. The pain is usually severe and generally centered over the brow. Nausea, vomiting, and profuse sweating are commonly associated symptoms which may lead to improper diagnosis and treatment in an emergency room setting.

Ocular examination shows a very shallow anterior chamber, corneal edema, and a mid-dilated pupil. [Gonioscopy](https://glaucomaassociates.com/glaucoma-tests-and-diagnosis/gonioscopy/) reveals a closed angle without any visible angle structures.

Treatment of an acute angle closure attack is aimed at breaking the attack and lowering the intraocular pressure. Initially, a combination of eye drops and oral medications are used to lower the pressure; miotics (e.g. Pilocarpine) have the additional potential benefit of breaking the attack. A [laser iridotomy](https://glaucomaassociates.com/laser-treatment-for-glaucoma/laser-iridotomy-for-glaucoma/) is usually necessary to break the attack and prevent a repeat attack; the non-affected eye typically requires a laser iridotomy as well to prevent a similar angle closure attack.

There are numerous sequellae of an acute angle-closure attack. Vision is often affected and visual recovery is variable. Following an attack the cornea frequently develops folds in Descemet’s membrane; recovery is usually complete, although some degree of endothelial cell loss may occur. Occasionally, chronic corneal edema develops despite normalized intraocular pressure. This most often occurs in patients with pre-existing Fuch’s dystrophy, or long-standing angle-closure prior to treatment.

During the acute attack, sector ischemia of the iris may occur due to closure of its stromal vessels, and if marked, causes an aseptic anterior uveitis. Ischemic necrosis of the iris causes sectoral atrophy, usually in the horizontal meridian. The pupil may remain permanently dilated and unresponsive to miotics or mydriatics because of iris sphincter damage. In many cases there may also be characteristic “spiraling” of the superficial iris stroma. In such instances, the iris fibers appear to originate at a point near the pupil margin and fan out, in an oblique fashion. Due to the sudden sustained rise in intraocular pressure, lens opacities called glaukomflecken may appear. These opacities represent areas of lens epithelium necrosis. Clinically, they initially appear as small whitish cloudy areas beneath the anterior capsule in the pupillary zone.

Chronic Angle-Closure Glaucoma

The primary angle-closure glaucomas are conditions which by definition result from obstruction of the anterior chamber angle by iris tissue, without an obvious underlying ocular or systemic cause. In chronic angle-closure glaucoma (CACG), peripheral anterior synechiae (PAS; scar tissue extending between the iris and the trabecular meshwork) close off the anterior chamber angle. This blocking of the trabecular meshwork ultimately causes elevation of the intraocular pressure. Angle-closure glaucoma caused by a pupillary block mechanism can be divided into either acute (discussed above) or chronic angle-closure. The initiating event is a functional block between the anterior lens surface and the posterior pupillary portion of the iris. This results in trapping of aqueous in the posterior chamber thus pushing the peripheral iris forward and closing of the angle; outflow resistance increases, intraocular pressure rises, and with time permanent synechial closure ensues.

Chronic angle-closure glaucoma is often overlooked as a diagnosis and may be mis-treated as primary open-angle glaucoma. [Gonioscopy](https://glaucomaassociates.com/glaucoma-tests-and-diagnosis/gonioscopy/) is the key to their recognition and classification. A thorough knowledge of the normal angle is imperative before the subtle changes of PAS formation can be discovered and quantitated. Such knowledge is dependent on the ability to visualize all aspects of the angle, a skill all [GAT Doctors](https://glaucomaassociates.com/glaucoma-specialists/) have as a result of their fellowship training in glaucoma.

Similar to other forms of glaucoma, the primary treatment for CACG is lowering of the intraocular pressure. However, treatment of the pupillary block component of the disease with a [laser iridotomy](https://glaucomaassociates.com/laser-treatment-for-glaucoma/laser-iridotomy-for-glaucoma/) is important. The goal of a laser iridotomy is to slow or stop progressive PAS formation and angle dysfunction. Even with a patent iridotomy, progressive angle closure may occur and therefore serial gonioscopy is imperative. Most patients are adequately treated with medical therapy, though [glaucoma surgery](https://glaucomaassociates.com/incisional-glaucoma-surgery/) may be necessary.

Glaucoma Suspect (Borderline Glaucoma)

Patients who are deemed to be a glaucoma suspect (borderline glaucoma) may show some signs of glaucoma without definitive disease, or may carry risk factors for developing glaucoma. Often, patients are classified as “low risk” or “high risk” glaucoma suspects based on the number of findings or risk factors. Classically, patients described as a glaucoma suspect had one of the following:

* Optic nerve or nerve fiber layer suspicious of glaucoma
* Intraocular pressure of greater than 21 mmHg
* Visual field changes consistent with glaucoma

Several criteria have been associated with an increased risk of glaucoma. Patients may also be considered if they have some of the following:

* Level of intraocular pressure rise
* Cup-to-disc ratio (optic nerve appearance)
* Family history of glaucoma
* Age
* Race
* Central corneal thickness
* Associated diseases (e.g. diabetes, myopia)

Although there is no way to know which glaucoma suspects will ultimately develop glaucoma, your ophthalmologist will discuss your risk factors with you and follow your at regular intervals.

Narrow Angle and Plateau Iris Syndrome

Plateau Iris SyndromeWhile both a narrow angle and plateau iris can be associated with development of angle closure glaucoma, they are not synonymous with glaucoma. Rather, both a narrow angle and plateau iris are anatomic descriptions of the anterior chamber angle between the cornea and iris.

**Narrow Angle**

Patients with a narrow anterior chamber angle are at risk for developing both acute and chronic angle-closure glaucoma. Numerous studies have attempted to predict which patients will go on to develop glaucoma, but none have been consistently validated. Not all patients with narrow angles necessarily need treatment. However, those with intraocular pressure elevation, peripheral anterior synechiae (PAS), appositional angles, history of previous angle closure, positive family history, and/or symptoms of intermittent angle closure should be treated with a [laser iridotomy](https://glaucomaassociates.com/laser-treatment-for-glaucoma/laser-iridotomy-for-glaucoma/). Regardless of a whether a patient is treated or not, all those with a narrow angle should be warned of the signs/symptoms of an angle closure attack or intermittent angle closure, including: eye pain, eye redness, blurry vision, multi-colored halos, headache, nausea, and vomiting.

Several systemic medications including cold and allergy medications, urological medications, and anti-depressants are associated with an increased risk of an acute angle-closure attack. Please let your ophthalmologist know if you take any of these types of medications with any regularity, as their use may influence your doctor’s decision to treat you