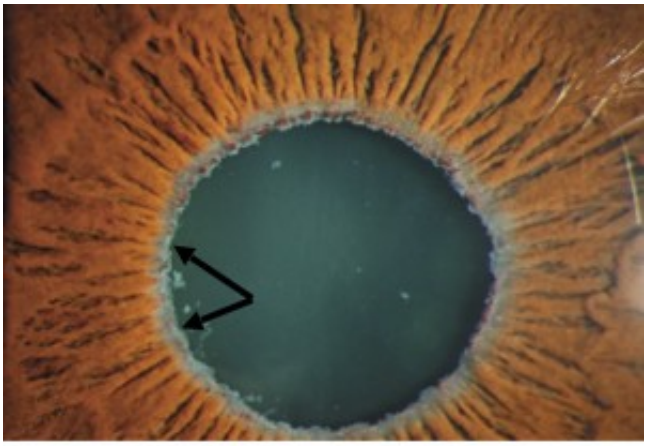
Secondary Open-Angle Glaucoma

Similar to primary open angle glaucoma, patients with secondary open angle glaucoma have an anterior chamber angle which can easily be visualized without obvious obstruction of angle structures. However, in secondary open angle glaucoma, there is an increased resistance to outflow and elevated intraocular pressure associated with an ocular or systemic cause. While the angle can easily be visualized without obstruction, careful gonioscopy often shows subtle changes that your ophthalmologist can detect to give clues as to the cause of the glaucoma.

Pseudoexfoliation Syndrome

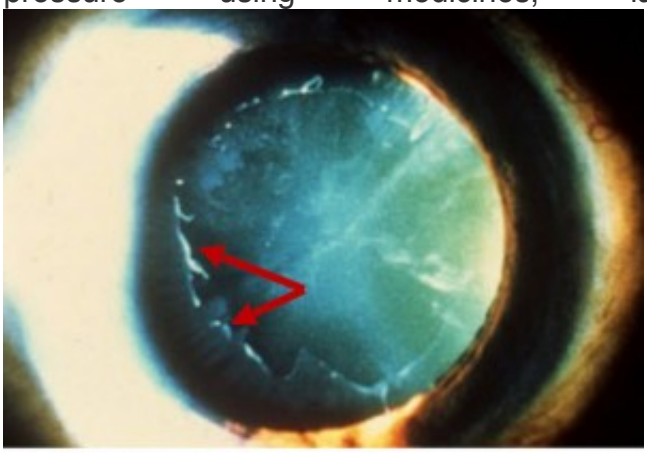


Pseudoexfoliation of Iris Pupillary Border

Pseudoexfoliation Syndrome may lead to a specific type of open angle glaucoma characterized by the deposition of fibrillary material on structures within the anterior chamber, including the iris, lens capsule, zonules, and trabecular meshwork. The disease can be in one or both eyes, and asymmetry of findings is very common. Risk factors for pseudoexfoliation syndrome include age (nearly all patients are older than 50 and most are older than 70), female gender, and Scandinavian descent. **Not all patients with pseudoexfoliation syndrome develop pseudoexfoliation glaucoma.**

Deposition of this material in the trabecular meshwork (part of the anterior chamber angle responsible for drainage), can result in elevated intraocular pressures and subsequent glaucoma. Similar to other types of glaucoma, the treatment for pseudoexfoliation glaucoma is lowering of the intraocular

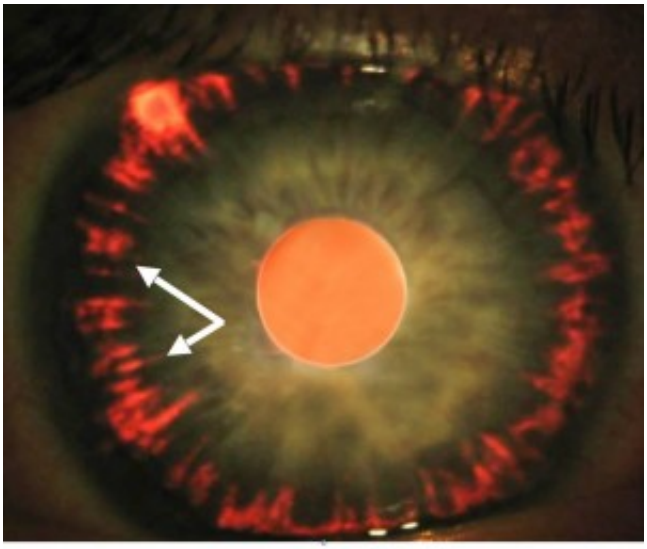
pressure using medicines, lasers, and surgery.



Pseudoexfoliation of Lens Capsule

Other findings associated with pseudoexfoliation syndrome include iris atrophy, floppy iris (iridodonesis), poor pupillary dilation, and zonular weakness with lens instability (phacodonesis). Several of these characteristics can make cataract surgery more complex and can be associated with dramatic eye pressure elevations in the immediate post-operative period. The GAT Doctors have tremendous experience with cataract surgery in patients with Pseudoexfoliation Syndrome as well as appropriately caring for patients in the post-operative period.

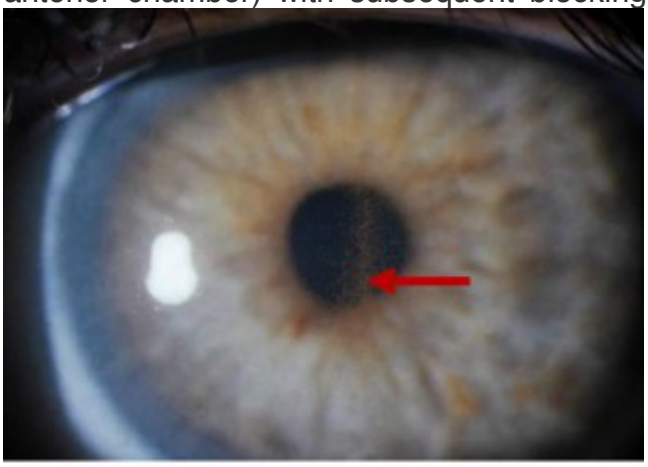
Pigment Dispersion Syndrome



Iris Transillumination Defects 360°

Pigment Dispersion Syndrome is characterized by deposition of pigment on various structures within the eye, including the cornea (**Krukenberg spindle**), trabecular meshwork, and lens capsule (Zentmayer line). It is further characterized by mid-peripheral **iris transillumination defects** and is often associated with a posterior bowing of the iris. Risk factors for pigment dispersion syndrome include Caucasian race, male gender, myopia (near-sightedness), and age somewhat younger than most glaucomas, usually between 20 and 50 years. However, not all patients with pigment dispersion syndrome develop pigmentary glaucoma.

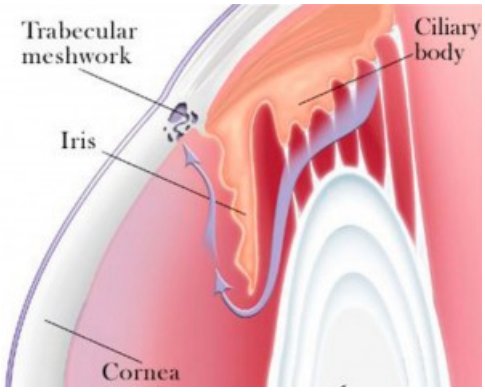
Gonioscopy in patients with pigment dispersion syndrome reveals a densely pigmented trabecular meshwork. The syndrome is often characterized by wide fluctuations in intraocular pressure, felt to be a result of increased pigment release into the aqueous humor (fluid within the anterior chamber) with subsequent blocking of the trabecular meshwork.



Krukenberg Spindle

Similar to other types of glaucoma, the treatment for pigmentary glaucoma is lowering of the intraocular pressure using medicines, lasers, and surgery. Of note, these patients have been found to respond particularly well to Laser Trabeculoplasty.

Steroid (Medication) Induced Glaucoma



A number of medications, most notably corticosteroids ("steroids"), have been associated with elevation of intraocular pressure. Although topical steroid eye drops and intra/peri-ocular steroids (e.g. intravitreal, intracameral, subtenon, retroseptal) are most likely to cause pressure elevations, any form of steroids including oral, topical creams, nasal sprays, and injections can increase intraocular pressure. While prescription steroid medications are often more potent and more likely culprits, numerous over-the-counter creams and nasal sprays can also cause increased eye pressure.

It is estimated that ~30% of all people have a mild-moderate elevation (up to 11 mmHg rise from baseline) with use of ocular steroids (drops or injectable) and 5% have an elevation of greater than 15 mmHg usually requiring treatment. Among patients with preexisting glaucoma, the likelihood of a pressure elevation rises significantly.

More recently, it has been observed that patients receiving various multiple non-steroid intravitreal injections (e.g. bevacizumab/Avastin®, ranibizumab/Lucentis®, aflibercept/Eylea®) can also have sustained intraocular pressure rise. These medications are often used for retinal disease including macular degeneration, diabetes, and macular edema (swelling).

Treatment for steroid (medication) induced glaucoma is similar to other secondary open-angle glaucomas and medications are usually successful. Cases requiring surgical intervention also have a high degree of success.

Other examples of secondary open-angle glaucoma include:

- Inflammatory Glaucoma
- Intraocular Tumor-Induced Glaucoma
- Lens Induced Glaucoma (e.g. phacolytic, lens particle, phacoantigenic)
- Traumatic Glaucoma (e.g. accidental, surgical)
- Elevated Episcleral Venous Pressure (e.g. Sturge-Weber Syndrome, arteriovenous fistula, thyroid-associated orbitopathy)

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 reveals a closed angle without any visible angle structures.

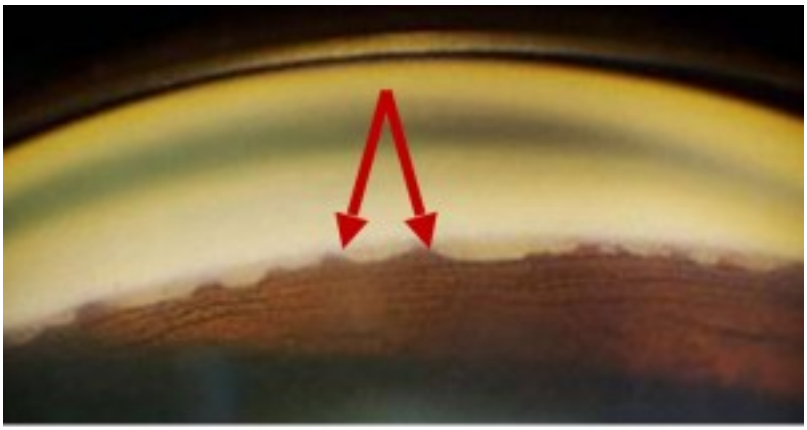
Treatment of an acute angle closure attack is aimed at breaking the attack and lowering the intraocular pressure. Initially, a combination of eye drops and oral medications are used to lower the pressure; miotics (e.g. Pilocarpine) have the additional potential benefit of breaking the attack. A laser iridotomy 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



PAS - Peripheral Anterior Synechiae

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 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 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 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 may be necessary.