

GLAUCOMA “SILENT THIEF OF SIGHT”

Glaucoma is a term describing a group of ocular disorders with multi-factorial etiology united by a clinically characteristic intraocular pressure-associated optic neuropathy. This can permanently damage vision in the affected eye(s) and lead to blindness if left untreated. It is normally associated with increased fluid pressure in the eye (aqueous humour). The term "ocular hypertension" is used for people with consistently raised intraocular pressure (IOP) without any associated optic nerve damage. Conversely, the term 'normal tension' or 'low tension' glaucoma is used for those with optic nerve damage and associated visual field loss, but normal or low IOP.

The nerve damage involves loss of retinal ganglion cells in a characteristic pattern. The many different subtypes of glaucoma can all be considered to be a type of optic neuropathy. Raised intraocular pressure (above 21 mmHg or 2.8 kPa) is the most important and only modifiable risk factor for glaucoma. However, some may have high eye pressure for years and never develop damage, while others can develop nerve damage at a relatively low pressure. Untreated glaucoma can lead to permanent damage of the optic nerve and resultant visual field loss, which over time can progress to blindness.

Glaucoma can be roughly divided into two main categories, "open-angle" and "closed-angle" (or "angle closure") glaucoma. The angle refers to the area between the iris and cornea, through which fluid must flow to escape via the trabecular meshwork. Closed-angle glaucoma can appear suddenly and is often painful; visual loss can progress quickly, but the discomfort often leads patients to seek medical attention before permanent damage occurs. Open-angle, chronic glaucoma tends to progress at a slower rate and patients may not notice they have lost vision until the disease has progressed significantly.

Glaucoma has been called the "silent thief of sight" because the loss of vision often occurs gradually over a long period of time, and symptoms only occur when the disease is quite advanced. Once lost, vision cannot normally be recovered, so treatment is aimed at preventing further loss. Worldwide, glaucoma is the second-leading cause of blindness after cataracts. It is also the leading cause of blindness among African Americans. Glaucoma affects one in 200 people aged 50 and younger, and one in 10 over the age of eighty. If the condition is detected early enough, it is possible to arrest the development or slow the progression with medical and surgical means. Closed-angle glaucoma accounts for less than 10% of glaucoma cases in the United States, but as many as half of glaucoma cases in other nations (particularly Asian countries). About 10% of patients with closed angles present with acute angle closure crises characterized by sudden ocular pain, seeing halos around lights, red eye, very high intraocular pressure (>30 mmHg), nausea and vomiting, suddenly decreased vision, and a fixed, mid-dilated pupil. It is also associated with an oval pupil in some cases. Acute angle closure is an emergency. Many people of East Asian descent are prone to developing angle closure glaucoma due to shallower anterior chamber depths, with the majority of cases of glaucoma in this population consisting of some form of angle closure. Inuit also have a 20 to 40 times higher risk of developing primary angle closure glaucoma. Women are three times more likely than men to develop acute angle closure glaucoma due to their shallower anterior chambers. People of African descent are three times more likely to develop primary open angle glaucoma. Positive family history is a risk factor for glaucoma. The relative risk of having primary open angle glaucoma (POAG) is increased approximately 2–4 fold for individuals who have a sibling with

glaucoma. Glaucoma, particularly primary open angle glaucoma, is associated with mutations in several different genes (including MYOC, ASB10, WDR36, NTF4, TBK1 genes), although most cases of glaucoma do not involve these genetic mutations. Normal tension glaucoma, which comprises one-third of POAG, is also associated with genetic mutations (including OPA1 and OPTN genes). Various rare congenital/genetic eye malformations are associated with glaucoma. Occasionally, failure of the normal third trimester gestational atrophy of the hyaloid canal and the tunica vasculosa lentis is associated with other anomalies. Angle closure-induced ocular hypertension and glaucomatous optic neuropathy may also occur with these anomalies, and has been modelled in mice. Other factors can cause glaucoma, known as "secondary glaucomas", including prolonged use of steroids (steroid-induced glaucoma); conditions that severely restrict blood flow to the eye, such as severe diabetic retinopathy and central retinal vein occlusion (neovascular glaucoma); ocular trauma (angle recession glaucoma); and uveitis (uveitic glaucoma). In a large study in the UK, glaucoma patients had a 29% increased incidence of systemic hypertension compared to age- and sex-matched controls. The modern goals of glaucoma management are to avoid glaucomatous damage and nerve damage, and preserve visual field and total quality of life for patients, with minimal side effects. This requires appropriate diagnostic techniques and follow-up examinations, and judicious selection of treatments for the individual patient. Although intraocular pressure is only one of the major risk factors for glaucoma, lowering it via various pharmaceuticals and/or surgical techniques is currently the mainstay of glaucoma treatment. Vascular flow and neurodegenerative theories of glaucomatous optic neuropathy have prompted studies on various neuroprotective therapeutic strategies, including nutritional compounds, some of which may be regarded by clinicians as safe for use now, while others are on trial. Intraocular pressure can be lowered with medication, usually eye drops. Several different classes of medications are used to treat glaucoma, with several different medications in each class. Each of these medicines may have local and systemic side effects. Adherence to medication protocol can be confusing and expensive; if side effects occur, the patient must be willing either to tolerate them, or to communicate with the treating physician to improve the drug regimen. Initially, glaucoma drops may reasonably be started in either one or in both eyes. Poor compliance with medications and follow-up visits is a major reason for vision loss in glaucoma patients. A 2003 study of patients in an HMO found half failed to fill their prescriptions the first time, and one-fourth failed to refill their prescriptions a second time. Patient education and communication must be ongoing to sustain successful treatment plans for this lifelong disease with no early symptoms. The possible neuroprotective effects of various topical and systemic medications are also being investigated.

- Prostaglandin analogs, such as latanoprost (Xalatan), bimatoprost (Lumigan) and travoprost (Travatan), increase uveoscleral outflow of aqueous humor. Bimatoprost also increases trabecular outflow.
- Topical beta-adrenergic receptor antagonists, such as timolol, levobunolol (Betagan), and betaxolol, decrease aqueous humor production by the ciliary body.
- Alpha2-adrenergic agonists, such as brimonidine (Alphagan) and apraclonidine, work by a dual mechanism, decreasing aqueous humor production and increasing uveoscleral outflow.
- Less-selective alpha agonists, such as epinephrine, decrease aqueous humor production through vasoconstriction of ciliary body blood vessels, useful only in open-angle glaucoma. Epinephrine's mydriatic effect, however, renders it unsuitable for closed-angle glaucoma due to further narrowing of the uveoscleral outflow (i.e. further closure of trabecular meshwork, which is responsible for absorption of aqueous humor).

- Miotic agents (parasympathomimetics), such as pilocarpine, work by contraction of the ciliary muscle, opening the trabecular meshwork and allowing increased outflow of the aqueous humor. Echthiophate, an acetylcholinesterase inhibitor, is used in chronic glaucoma.
- Carbonic anhydrase inhibitors, such as dorzolamide (Trusopt), brinzolamide (Azopt), and acetazolamide (Diamox), lower secretion of aqueous humor by inhibiting carbonic anhydrase in the ciliary body.
- Physostigmine is also used to treat glaucoma.
- Marijuana was found, in the early 1970s, to reduce pressure in the eyes, though how the cannabinoids in marijuana produce this effect remains unknown.

Conventional surgery to treat glaucoma makes a new opening in the meshwork, which helps fluid to leave the eye and lowers intraocular pressure. Both laser and conventional surgeries are performed to treat glaucoma. Surgery is the primary therapy for those with congenital glaucoma. Generally, these operations are a temporary solution, as there is not yet a cure for glaucoma. Canaloplasty is a nonpenetrating procedure using microcatheter technology. Argon laser trabeculoplasty (ALT) may be used to treat open-angle glaucoma. It is a temporary solution, not a cure. A newer type of laser trabeculoplasty uses a "cold" (nonthermal) laser to stimulate drainage in the trabecular meshwork. Nd:YAG laser peripheral iridotomy (LPI) may be used in patients susceptible to or affected by angle closure glaucoma or pigment dispersion syndrome. Diode laser cycloablation lowers IOP by reducing aqueous secretion by destroying secretory ciliary epithelium. Glaucoma drainage implants: Professor Anthony Molteno developed the first glaucoma drainage implant, in Cape Town in 1966. Since then, several different types of implants have followed on from the original, the Baerveldt tube shunt, or the valved implants, such as the Ahmed glaucoma valve implant or the ExPress Mini Shunt and the later generation pressure ridge Molteno implants. These are indicated for glaucoma patients not responding to maximal medical therapy, with previous failed guarded filtering surgery.

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