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Glaucoma: contributory factors, treatment and prognosis

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Abstract:*Glaucoma is the leading cause of blindness after cataract. Individuals of Asian descent are prone to angle closure glaucoma due to shallower anterior chamber depths. People with family history of glaucoma are at higher risk. The major risk factor for glaucoma is the increased intraocular pressure. Early signs of glaucoma are gradual progressive visual field loss, and optic nerve changes. The diagnosis for glaucoma includes measurement of the intraocular pressure via tonometry. The aims of glaucoma management are to avoid glaucomatous damage and nerve damage, and preserve visual field and quality of life for patients. Poor compliance with medications and follow-up visits is a major reason for vision loss in glaucoma patients. Both laser and conventional surgeries are performed to treat glaucoma. The intraocular pressure can also have an effect, with higher pressure reducing time until blindness.*

Keywords*: Open-angle glaucoma, intraocular pressure, Optic nerve damage, Trabeculactomy.*

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I. Introduction

The word "glaucoma" is from ancient Greek *glaukos* which means blue, green or gray [1].Glaucoma is a group of eye diseases which result in damage to optic nerve and vision loss [2].As of 2010,there were 44.7 million people in the world with open angle glaucoma [3].By 2020,the prevalence is projected to increase to 58.6 million worldwide and 3.4 million in the United States [3].Both internationally and in the United States glaucoma is the second-leading cause of blindness [4].Globally cataract are a more common cause. Glaucoma is also the leading cause of blindness in African Americans, who have high rates of primary open angle glaucoma [5].Bilateral vision loss can negatively affect mobility and interfere with driving [6].The most common type is open angle glaucoma with less common types including closed angle glaucoma and normal tension glaucoma [2].Open angle glaucoma develops slowly over time with no pain [2].Closed – angle glaucoma can present gradually or sudden [4].The sudden presentation may involve severe eye pain, blurred vision,mid-dilated pupil redness of the eye, and nausea [2,4].Vision loss from glaucoma, once it has occurred, is permanent [2].Contributory factors for glaucoma include increased pressure in the eye, a family history of condition, and high blood pressure [4].For eye pressure a value of greater than 21 mmHg or 2.8 kpa is often used with higher pressure leading to greater risk [4].Some individuals with high eye pressure for years and never develop damage [4].Conversely optic nerve damage may occur with normal pressure, known as normal tension glaucoma [7].Laser treatments may be effective in both open and closed-angle glaucoma [4].Treatment of closed-angle glaucoma is a medical emergency [2].The paper reviews the contributory factors, and current treatment of glaucoma.

II. Contributory factors

There are several contributory factors(causes) for glaucoma, ocular hypertension(increased pressure within the eye),is the most important risk factor in most glaucoma, but in some populations, only 50% of people with primary open-angle glaucoma actually have elevated ocular pressure[8].Open-angle glaucoma accounts for 90% of glaucoma cases in the United States. Closed-angle accounts for less than 10% of glaucoma cases in the United States, but many as half of glaucoma cases in other nations(particularly East Asian countries [8].There is no clear evidence indicates vitamin deficiencies cause glaucoma in humans. It follows, then, that oral vitamin supplementation is not a recommended treatment for glaucoma [9].Caffeine increases intraocular pressure in those with glaucoma, but does not appear to affect normal individuals [10].

Role of ethnicity: Many people of East Asian descent are prone to developing angle-closure glaucoma due to shallower anterior chamber depths, with the majority of the cases of glaucoma in this population consisting of some form of angle closure [11].Higher rates of glaucoma have also been reported for **Inuit** (people living in

Siberia,Canada,Alaska, andGreenland(,ref:Macmillan English Dictionary) populations compared to white populations in Canada and Greenland [12]

Roleofgenetics: Individuals with family history of glaucoma are at higher risk.The relative risk of having primary open angle glaucoma (P0AG) is increased about two-to four fold for people with a sibling with glaucoma [13].Glaucoma,particularly primary open angle glaucoma, is associated with mutations in several genes, including MYOC,ASB10,WDR56,NTF4,TBK 1 [14],andRPGRIP1 [15].although most cases of glaucoma do not involve these genetic mutations. Normal tension, which compromise one-third of POAG,is also associated with genetic mutations (including)PA1 and 0PTN genes [16].Various rare congenital/genetic eye malformations are associated with glaucoma. Occasionally, failure of the normal third trimester gestational atrophy of the hyaloid canal and tunicavasculosalentis is associated with other anomalies. Angle closure-induced ocular hypertension and glaucomatous optic neuropathy may also occur with these anomalies [17],and has been modelled in mice [18].

III. Disease progression

The underlying cause of open angle glaucoma remains unclear. Several theories exist on its exact etiology. However, the major risk factor for most glaucoma and the focus of treatment is increased intraocular pressure. Intraocular pressure is a function of production of liquid aqueous humor by the ciliary processes of the eye, and its drainage through the trabecular meshwork. Aqueous humor flows from the ciliary processes into the posterior chamber, bounded by the lens and thezonules of Zinn, and anterior of the iris.It then flows through the pupil of the iris into anterior chamber, bounded posteriorly by the iris and anteriorly the cornea. From here the trabecular meshwork drains aqueous humor via the scleral venous sinus (Schlemm's canal) into scleral plexuses and general blood circulation [19].

In open/wide angle glaucoma, flow is reduced through the trabecular meshwork, due to the degeneration and obstruction of the trabecularmeshwork,those original functions is to absorb the aqueous humor. Loss of aqueous humor absorption leads to increased resistance and thus a chronic, painless buildup of pressure in the eye [20].In close/narrow angle iridocornealangle is completely closed because of forward displacement of the final toll and root of iris against the cornea, resulting in the inability of the aqueous fluid flow from the posterior to the interior chamber and then out of the trabecular network. This accumulation of aqueous humor causes an acute increase in pressure and pain. The inconsistent relationship of glaucomatous optic neuropathy with increased intraocular pressure has provoked hypotheses and studies on anatomic structure, eye development, nerve compression trauma, optic nerve blood flow, excitatory neurotransmitter,trophic factor, retinal ganglion cell/axon degeneration glial support cell, immune system, aging mechanism of neuron loss, and severing of the nerve fibers at the scleral edge [21,22].

IV. Clinical manifestations and Diagnosis

Open angle glaucoma is painless and does not have acute attacks, thus lack of clear symptoms make screening via regular eye check-ups important. The only signs are gradually progressive visual field loss, and optic nerve changes (increased cup-to-disc ratio on funduscopic examination).About 10% of people with closed angles present with acute angle closure 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 is also associated with an oval pupil in some cases. Acute angle closure is an emergency. Opaque specks may occur in the lens in glaucoma, known as glaukomflecken[23].

Leske and associates in an series of 4314 black participants confirmed in the Barbados Eye Study black population, persons most likely to have open-angle glaucoma (OAG) were older men, family history of OAG,high intraocular pressure, lean mass, and cataract history [24].Authors also confirmed in another series of 4601,participants,intraocular pressure(IOP) was higher in the black participants than in the white participants. The results showed that open-angle glaucoma and highIOP alone have a different distribution by sex,although open-angle glaucoma was more frequent in men, ocular hypertension was more frequent in woman[25].

Diagnosis: Screening for glaucoma is usually performed as part of a standard eye examination performed by the optometrists and ophthalmologists. Tests for glaucoma should include measurement of of the intraocular pressure via tonometry [26],anterior angle examination or gonioscopy,and examination of the optic nerve to look for any visible damage to it,or change in the cup-to-disc ratio and also rim appearance and vascular change. A Formal visual field test should be performed. The retinal nerve fiber layer can be asserted with imaging techniques such as optical coherence tomography, scanning laser polarimetry, and/or scanning ophthalmoscopy (Heidelberg retinal tomogram)[27].Examination for glaucoma also should be assessed with more attention given to sex ,race, history of drug use .refraction, inheritance and family history [27].
Frequently used glaucoma tests:[28,29].

i). Tonometry to test inner eye pressure.
ii).Ophthalmoscopy dilated eye pressure to test shape and color of optic nerve.
iii).Visual field orperimetry to test complete field of vision.
Iv).Gonioscopy to test angle in the eye where the iris meets the cornea.
V).Pachymetry to test thickness of cornea.
vi).Nerve fiber analysis to test thickness of nerve fiber layer.
Primary glaucoma and its variants: Primary open angle glaucoma, also known as chronic open-angle glaucoma, chronic simple glaucoma, glaucoma simplex, also include:
* High tension glaucoma,
* Low tension glaucoma,
* Primary angle closure glaucoma,also known as primary closed angle glaucoma, narrow – angle glaucoma, pupil –block glauycoma,acute congestive glaucoma
* Acute angle closure glaucoma (AACG) [30].
*Chronic angle closure glaucoma
*Intermitten angle closure glaucoma
* Superimposed on chronic open-angle closure glaucoma ("combined mechanism"-uncommon).

Primary open-angle glaucoma is when optic never damage in a progressive loss of the visual field [30].This is associated with increased pressure in the eye. Not all people with primary open-angle glaucoma have eye pressure that is elevated beyond normal, but decreasing the eye pressure further has been shown to stop progression even in these cases. The increased pressure is caused by trabecular meshwork blockage. Because the microscopic passageways are blocked, the pressure build up in the eye causes imperceptible very gradual vision loss. Peripheral vision affected first, but eventually the entire vision will be lost if not treated. The diagnosis is made by looking for cupping of the optic nerve.Prosglandlin agonist work by opening uveiscleralpassageways.Bet-blockers, such as timilol,work by decreasing aqueous formation. Carbonic anhydrase inhibitors decrease bicarbonate formation from ciliary process in the eye thus decreasing the formation of Aqueous humor .Parasympathetic analogs are drugs that work on the trabecular outflow by opening up passageway and constricting the pupil. Alpha 2 agonists (bromonidine,aparclonidine)both decrease fluid production (via.inhibition AC) and increase drainage [31].

Developmental glaucoma: Neovascular glaucoma results when new abnormal vessels begin developing in the angle of the eye that begins blocking the drainage. Patients with such condition begin to rapidly loss their eyesight. Sometimes, the disease appears very rapidly especially after cataract surgery procedures. A new treatment for this disease, as first reported by Kahook and colleagues, involves the use of novel group of medications known as anti-VEGF agents. These injectable medications can lead to a dramatic decrease in new vessel formation and, if injected early enough in the disease process, may lead to normalization intraocular pressure. Currently, there are no high quality control trials demonstrating a beneficial effect of anti VEGF treatment in lowering IOP in people with neovascular glaucoma [30].

Toxic glaucoma is open angle glaucoma with an unexplained significant rise of intraocular pressure following unknown pathogenesis.Intraocular pressure can sometimes reach 80mmHg(11kPa).It characteristically manifests as ciliary body inflammation and massive trabecular edema that sometimes extends to Schlemm'scanal.This condition is differentiated from malignant glaucoma by presence of a deep and clear anterior chamber and a lack of aqueous misdirection Also, the corneal appearance is not as hazy.A reduction in visual acuity can secure followed neuroretinalbreakdown.Associated factors include inflammation,drugs,trauma and intraocular surgery,including cataract surgery and vitrectomy procedures.CedePardianto (2005) reported on for patients who had toxic glaucoma,One of them underwent phacoemulsification with small particle nucleus drops.Some cases can be resolved with some medication, vitrectomy procedures or trabeculectomy.Valving procedures can give some relief, butfurther research is required [32].

Different form of glaucoma: Glaucoma is an umbrella term for eye conditions which damage to optic nerve, and which can lead to a loss of vision [33].The main cause of damage to the optic nerve is intraocular pressure(IOP),excessive fluid pressure within the eye, which can be due to various reasons including blockage of drainage ducts, and narrowing or closure of angle between the iris and cornea. The primary division in categorizing different forms(types) of glaucoma is open angle and closed angle (or angle closure) glaucoma. In open angle glaucoma, the iris meets the cornea normally, allowing the fluid from inside the eye to drain, thus relieving the internal pressure. Where this angle is narrowed or closed, pressure increases over time, causing damage to optic nerve, leasing to blindness. Primary open angle glaucoma (also, primary glaucoma, chronic glaucoma) refers to slow clogging of the drainage canals resulting in increased eye pressure which h causes

progressive optic nerve damage. This manifests as a gradual loss of visual fluid, staring with a loss of peripheral vision, but eventually the entire will be lost if not treated [31].This is the most common type of glaucoma, accounting for 90% of the cases in the United States, but fewer in Asian countries. Onset is slow and painless, and loss of vision is gradual and irreversible. Narrow angle glaucoma (also closed angle glaucoma) the iris bows forward, narrowing the angle that drains the eye, increasing pressure within the eye. If untreated, it can lead to the medical emergency of angle closure glaucoma.

Glaucoma screening: The United States Preventive Task Force as of 2013 states there is insufficient evidence to recommend for or against screening glaucoma (34].Therefore there is no national program in the US. Screening however, is recommended starting at age 40 by American Academy of 0phthalmology [4].There is glaucoma program in the UK.Those at risk are advised to have a dilated eye examination at least once a year [35].

V. Treatment and Prognosis

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 [36,37].This requires appropriate diagnostic techniques and follow up examinations, and selection of treatments for the individual patient. Although intraocular pressure is the only 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 therapeutics strategies, including nutritional compounds, some of which may be regarded by the clinicians as safe for use now, while others are on trial,

Intraocular pressure can be lowered with medications, usually eye drops. Several classes of medications are used to treat glaucoma, with several medications in each class. Each of this medicine may have local or 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 be reasonably be started either in one or in both eyes [38].Wiping the eye with an absorbent pad after the administration of eye drops may result in fewer adverse effects, like the growth of eyelashes and hyperpigmentation in the eyelid [39].Poor compliance with medications and follow- up visits is a major reason for vision loss in glaucoma patients. A 2003 study pf 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.Patients education and communication must be ongoing to sustain successful treatment plans for this lifelong disease with no early symptoms [40].

Laser therapy: Argon laser trabeculoplasty (ALT) may be used to treat open angle glaucoma but this is a temporary solution, and not a cure.A50-um argon laser spot is aimed at the trabecular meshwork to stimulate the opening of the mesh to allow more outflow of aqueous fluid. Usually, half of the angle is treated at a time. Traditional laser trabeculoplasty uses a thermal argon laser in an argon laser trabeculoplastyprocedure. A newer type of laser trabeculoplasty uses a ”cold”(nonthermal) laser to stimulate drainage in the trabecular meshwork. This newer procedure, selective laser trabeculoplasty (SLT) uses a 532-nm frequency doubled-switched Nd:YAG laser, which selectively targets melanin pigment in the trabecular meshwork cells. Studies show SLT is as effective as ALT at lowering eye pressure. In addition, SLT may be repeated three to four times, whereas ALT can usually be repeated only once.Nd:YAG laser peripheral iridology (LPI) may be used in patients susceptible to or affected by angle closure glaucoma or pigment dispersion syndrome. During laser iridology, laser energy is used to make a small full-thickness opening, in the iris to equalize the pressure between the front and back of iris, thus correcting any abnormal bulging of the iris.In people with narrow angles, this can uncover the trabecular meshwork. In some cases of intermittent or short- term angle closure, this may lower the eye pressure. Laseriridology reduces the risk of developing an attack of closure. In most cases, it also reduces the risk of developing chronic angle closure. In most cases it also reduces the risk of developing chronic angle closure or adhesions of iris to the trabecular meshwork. Diodelasercycloablation lowers IOP by reducing aqueous secretion by destroying secretory ciliary epithelium [27].

Treatment by surgery::Both laser and conventional surgeries are performed to treat glaucoma. Surgery is the primary therapy for those with congenital glaucoma [.41].Generally these operations are temporary solution, as there is not yet a cure for glaucoma.Canatoplasty is a non-penetrating procedure using micro catheter technology. By opening the canal, the pressure inside the eye may be relived, although the reason is unclear, since the canal (of Schlemm) does not have any fluid resistance in glaucoma or healthy eyes. Long term results are not avail [42,43].Trabeculactomy is the most common conventional surgery performed for glaucoma. Glaucoma drainage implants were developed by Professor Anthony Molteno,as the first glaucoma drainage

implant, in Cape Town in 1966.These (implants) are indicated for glaucoma patients not responding to maximal medical therapy, with previous failed guarded filtering surgery (trabelectomy). [44].Laser-assisted non-penetrating deep sclerectomy:The most common surgical approach currently used for the treatment of glaucoma is turbelectomy,in which sclera is punctured to alleviate intraocular pressure .Nonpenetrating deep sclerectomy(NPDS) surgery is a similar, but modified, procedure, in which instead of puncturing the scleral bed and trabecular meshwork under a scleral flap, a second deep scleral flap is created, excised, with further procedures of deroofing the Schlemm'scanal, uponwhich, percolation of liquid from the inner eye is achieved and thus alleviating pressure, without penetrating the eye.NPDS is demonstrated to have significantly fewer side effects than trabeculactomy [45].

Prognosis: In open angle glaucoma, the typical progression from normal vision to complete blindness takes 25 years to 70 years without treatment, depending on the method of estimation used [46].The intraocular pressure can also have an effect, with higher pressure reducing time until blindness.

VI. Conclusions

Glaucoma a painless, slow progressive vision loss to complete blindness over an extended period.Loss of vision is permanent. Increased eye pressure that leads to optic nerve damage is contributory factor. There isnoknown cure for glaucoma.

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