

EVALUATION AND DIAGNOSIS OF GLAUCOMA IN PRIMARY HEALTH CARE, LITERATURE REVIEW

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Abstract

Background: Glaucoma is the most common cause of permanent blindness in the globe. Many patients have glaucoma early in the disease and have no symptoms. Primary care physicians should know which patients to refer to an eye care expert in order to check for signs of glaucoma and determine whether systemic diseases or medications can increase a patient's risk of developing glaucoma.

Methods: The Medline, Pubmed, Embase, NCBI, and Cochrane databases were searched for studies of patients with Glucome evaluated and diagnosed in a primary health care.

Conclusion: If glaucoma is not treated, it can cause irreversible visual loss and is not a benign condition. The likelihood of harming the optic nerve increases with increased pressure and length of excessive IOP. Prompt diagnosis is essential for minimizing the effects of glaucoma, and prompt treatment is key for halting and reducing the progression of visual loss. Good treatment can frequently result in favorable outcomes, maintaining visual field integrity and slowing the progression of the disease, particularly when it comes to maintaining low IOP levels.

Keywords: Advanced glaucoma, Collaborative initial glaucoma, Closed angle glaucoma, Glaucoma, Glaucoma scrutility scale, Open-angle glaucoma.

Introduction

A complicated eye disease called glaucoma is marked by high intraocular pressure (IOP), which can eventually lead to visual loss. The most common cause of irreversible blindness in the US is glaucoma, which primarily affects elderly persons (1). Within each of the two main categories of glaucoma—primary or secondary—there are open-angle and closed-angle variations. Primary open-angle glaucoma (POAG), angle-closure glaucoma, and secondary open and angle-

closure glaucoma are all considered forms of adult glaucoma, with a focus on POAG, which is the most common kind (2). The condition known as glaucoma is caused by an acquired loss of retinal ganglion cells and axons within the optic nerve, also known as optic neuropathy. This condition causes a progressive loss of vision along with the distinctive optic nerve head appearance. It is this distinct pattern of peripheral vision loss that sets it apart from other forms of visual impairment (3). Unless early indicators of glaucoma are detected during normal eye exams, patients with POAG are frequently asymptomatic until extensive optic nerve damage occurs. Conversely, acute angle-closure glaucoma can occur rapidly, resulting in a sharp reduction in vision along with symptoms like headache, nausea, emesis, corneal edema, and eye pain (4). A prior eye injury or underlying medical disorders are common causes of secondary glaucoma, which raises IOP and causes optic neuropathy as a result. Subtypes such as congenital, pigmentary, neovascular, exfoliative, traumatic, and uveitic glaucoma are included in this category. Even with normal or unremarkable IOP values, glaucoma of the normal or low-tension type manifests as an optic neuropathy with glaucomatous vision loss (5). While a juvenile version of POAG and congenital, infantile, and developmental glaucoma typically afflict younger people, most glaucoma diagnoses occur in people 40 years of age and older. Even though glaucoma and IOP are frequently linked, a clear causal link has not been proven. Scholars are examining the role of genetic and environmental variables in the development of glaucoma. Studies on monozygotic twin pairs, who have a greater concordance rate than dizygotic couples, provide evidence that environmental factors play a major role in the development of the condition. While there is currently no cure for optic nerve damage or way to reverse loss of visual field, treatments such as medication, laser therapy, or incisional glaucoma surgery can slow the disease's progression and stop more vision loss. The goal of all therapy approaches is to reduce IOP and lessen the effects of this illness that could cause blindness. This strategy attempts to keep individuals with risk factors from developing glaucoma and to successfully manage the illness to slow its course in those who already have it (6).

Epidemiology:

Glaucoma is a serious public health issue because it causes irreversible damage to the optic nerve and retinal ganglion cells, as well as a progressive loss of peripheral vision. Its etiology is complicated, involving immunological, vascular, genetic, and anatomical components. Given that glaucoma is the second leading cause of irreversible blindness after cataracts, it is an important public health concern. Already impacting more than 60 million people globally, this figure is projected to rise to over 110 million by 2040 (7). The most common kind, known as POAG, affects between 2% to 4% of people 40 years of age and older and about 10% of people 75 years of age and older. Open-angle glaucoma is most prevalent in the African population. Compared to other population groups, people of African heritage have a 15-fold higher chance of becoming blind from open-angle glaucoma. On the other side, angle-closure glaucoma is more common in the Inuit community. In this group, women are more likely than men to be impacted, and people of Asian heritage are also more likely to be affected. These people also typically have a shallower anterior chamber, which adds to the greater rates of angle closure (8). Japanese inhabitants are most commonly affected by the normal-tension kind of glaucoma. In all forms of glaucoma, age is a major risk factor for the gradual loss of retinal ganglion cells. A family history of the condition in a key relative (mother, father, brother, sister, or children) as well as physical disorders like diabetes, high blood pressure, and heart disease are additional risk factors for developing glaucoma. An higher risk of glaucoma is also linked to ocular damage, anatomical variations such

as thinner corneas, a history of retinal detachment, inflammation or tumors in the eyes, and long-term use of corticosteroids (9).

Evaluation:

A fundoscopic examination, gonioscopy, tonometry, optical coherence tomography (OCT), and visual field tests are all included in the assessment process. Tonometry is crucial because of the highest risk factor among them, which is IOP (10). While there are other tonometer types available, Goldmann applanation tonometry is considered the gold standard for patients with glaucoma, increased IOP, and risk factors. In situations where Goldmann applanation tonometry is not feasible, such as in the case of bedridden patients, noncompliant individuals, minors, or those allergic to anesthetic drops, alternative tonometers may be taken into consideration (11). Assessment of visual acuity to ascertain any influence on vision, pachymetry to quantify corneal thickness, and retinal scans to track progressive changes in the retinal nerve fiber layer are additional useful tests in the examination of glaucoma. It is essential for people with risk factors, those receiving treatment for glaucoma, and those with ocular hypertension to conduct routine visual field testing utilizing full-threshold techniques (11). OCT is useful in the monitoring of morphological changes in the retinal nerve fiber layer and optic nerve, particularly in patients with early-to-moderate glaucoma and ocular hypertension (12). Diagnosing glaucoma is dependent on the presence of high intraocular pressure (IOP) and/or progressive optic neuropathy and/or visual field abnormalities. When an individual's IOP is higher than 21 mmHg and they do not exhibit functional visual field deficits or indications of glaucomatous optic neuropathy, they are classified with ocular hypertension. Studies reveal that approximately 20% of individuals with ocular hypertension go on to develop glaucoma. This emphasizes the significance of routine testing, tonometry, and thorough eye exams to start appropriate treatment targeted at lowering IOP when glaucomatous damage is present (13). There isn't a single gold standard test available for glaucoma diagnosis. Since glaucoma frequently manifests as asymptomatic vision loss, it is typically discovered during routine eye exams. To accurately diagnose and stage glaucoma, clinicians rely on identifying the typical look of the optic nerve, evaluating risk factors, and interpreting the results of ancillary tests. For those with glaucoma risk factors, the American Academy of Ophthalmology currently advises routine comprehensive eye exams. The frequency of these tests should be determined by criteria such as age, race, family history, and specific risk factors (14).

Symptoms and sign:

Acute angle closure can cause nausea, vomiting, and a stiff, rock-hard globe. It can also cause vision impairment, conjunctival hyperemia, and radiating discomfort from the eye. This is an ophthalmological emergency that needs to be treated right once in order to avoid blindness and severe damage to the eyes. On the other hand, open-angle glaucoma typically does not show symptoms until it has progressed to a severe level. If there are visual field defects, binocular vision usually makes up for them as they often do not occur in the same area of the fields of the two eyes. Therefore, many people with open-angle glaucoma are utterly ignorant that they have the condition (15), and most people with the condition report having no symptoms. At the time of diagnosis, one-third of patients already had the disease in one or more eyes at an advanced or late stage (16). According to Gramer et al., binocular visual field abnormalities prevented 10–20% of patients from being able to operate a vehicle when they first arrived at the clinic.

Treatment:

Personalized management plans for glaucoma must take into account the nature and severity of the condition. Although current therapies cannot stop vision loss entirely, they do try to reduce IOP, a major risk factor, in order to stop additional harm and vision loss. The goal of treatment alternatives such as eye drops, laser treatments, and surgery is to lower IOP. Utilizing instruments such as tonometry, visual field testing, OCT, and vision loss mapping is part of tracking the course of the disease. Initially, ocular pressure-lowering medicines are usually used to treat open-angle glaucoma. Prostaglandin analogs, β -blockers, α -2 agonists, carbonic anhydrase inhibitors, miotic agents, and, more recently, rho-kinase inhibitors and nitric oxide-donating drugs are common drug classes (17). Certain situations may also warrant consideration of laser trabeculoplasty, including argon laser trabeculoplasty, selective laser trabeculoplasty, and multipulse laser trabeculoplasty. Nonetheless, retreatments are sometimes required because the advantages of laser trabeculoplasty for decreasing IOP can continue for several months (18). Drugs to lower IOP and treat any underlying medical issues can be used to treat normal-tension glaucoma. Prostaglandin analogs, α -2 agonists, carbonic anhydrase inhibitors, and miotics are among the available treatment options. The use of β -blockers is controversial because of worries about decreased perfusion of the optic nerve head, especially in light of the possibility of aggravating the blood pressure nadir in the morning. Laser trabeculoplasty or filtration surgery may be required if medicinal therapy is shown to be inadequate, particularly in situations of progressive vision loss. After attaining a 30% reduction in IOP, patients with normal-tension glaucoma can halt or stabilize their field loss, according to a collaborative study (19). Because angle-closure glaucoma can cause increased pressures that result in ischemic nerve injury, glaucomatous optic nerve damage, or retinal vascular blockage, the condition is regarded as a medical emergency. In order to lower eye pressure as soon as possible, patients can take drugs, however laser peripheral iridotomy is typically necessary. In order to relieve pupillary blockage, a tiny hole is made in the iris during this treatment. Laser iridotomy resolves iris bombe and opens up the anterior chamber drainage angle, alleviating the condition by equalizing the pressure gradient between the posterior and anterior chambers. Laser iridoplasty and, less frequently, laser pupilloplasty can be used to flatten the peripheral iris. The angle may not necessarily have closed even after the IOP decreases. Acute harm to the ciliary body during an attack can cause a weeks-long reduction in the production of aqueous humor. Thus, in order to verify angle patency, a follow-up gonioscopy is essential. The percentage of the angle with peripheral anterior synechia from acute or previous subacute assaults is also determined by this evaluation. Patients are at a significant chance of suffering an attack in the opposing eye following the resolution of the acute crisis. Therefore, if the angle is narrow, patients should consider preventive iridotomy in the other eye and undergo gonioscopy to evaluate the angle. In addition to treating the underlying cause, treating secondary glaucoma may involve taking medicine to lower intraocular pressure (20).

Conclusion:

A complicated eye disease called glaucoma is marked by high intraocular pressure (IOP), which can eventually lead to visual loss. There are two classifications for this eye condition: primary or secondary types, and then open-angle or closed-angle variants. Primary open-angle glaucoma (POAG), angle-closure glaucoma, and secondary open and angle-closure glaucoma are all considered forms of adult glaucoma, with a focus on POAG, which is the most common kind. Scholars are examining the role of genetic and environmental variables in the development of glaucoma.

References:

- 1- Ezinne NE, Shittu O, Ekemiri KK, Kwarteng MA, Tagoh S, Ogbonna G, Mashige KP. Visual Impairment and Blindness among Patients at Nigeria Army Eye Centre, Bonny Cantonment Lagos, Nigeria. *Healthcare (Basel)*. 2022 Nov 18;10(11).
- 2- Cook C, Foster P. Epidemiology of glaucoma: what's new? *Can J Ophthalmol*. 2012 Jun;47(3):223-6.
- 3- Jonas JB, Aung T, Bourne RR, Bron AM, Ritch R, Panda-Jonas S. Glaucoma. *Lancet*. 2017 Nov 11;390(10108):2183-2193
- 4- Khazaeni B, Zeppieri M, Khazaeni L. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Nov 26, 2023. Acute Angle-Closure Glaucoma.
- 5- Gosling D, Meyer JJ. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Dec 12, 2022. Normal Tension Glaucoma.
- 6- Bailey JN, Loomis SJ, Kang JH, Allingham RR, Gharahkhani P, Khor CC, et al. Genome-wide association analysis identifies TXNRD2, ATXN2 and FOXC1 as susceptibility loci for primary open-angle glaucoma. *Nat Genet*. 2016 Feb;48(2):189-94
- 7- Allison K, Patel D, Alabi O. Epidemiology of Glaucoma: The Past, Present, and Predictions for the Future. *Cureus*. 2020 Nov 24;12(11):e11686
- 8- Bourne RR, Stevens GA, White RA, Smith JL, Flaxman SR, Price H, Jonas JB, Keeffe J, Leasher J, Naidoo K, Pesudovs K, Resnikoff S, Taylor HR., Vision Loss Expert Group. Causes of vision loss worldwide, 1990-2010: a systematic analysis. *Lancet Glob Health*. 2013 Dec;1(6):e339-49
- 9- Leung DY, Tham CC. Normal-tension glaucoma: Current concepts and approaches-A review. *Clin Exp Ophthalmol*. 2022 Mar;50(2):247-259
- 10- Bader J, Zeppieri M, Havens SJ. StatPearls [Internet]. StatPearls Publishing; Treasure Island (FL): Dec 12, 2023. Tonometry
- 11- Salvetat ML, Zeppieri M, Tosoni C, Brusini P. Repeatability and accuracy of applanation resonance tonometry in healthy subjects and patients with glaucoma. *Acta Ophthalmol*. 2014 Feb;92(1):e66-73
- 12- Mahmoudinezhad G, Moghimi S, Proudfoot JA, Brye N, Nishida T, Yarmohammadi A, Kamalipour A, Zangwill LM, Weinreb RN. Effect of Testing Frequency on the Time to Detect Glaucoma Progression With Optical Coherence Tomography (OCT) and OCT Angiography. *Am J Ophthalmol*. 2023 Jan;245:184-192.
- 13- Kelly SR, Khawaja AP, Bryan SR, Azuara-Blanco A, Sparrow JM, Crabb DP. Progression from ocular hypertension to visual field loss in the English hospital eye service. *Br J Ophthalmol*. 2020 Oct;104(10):1406-1411.
- 14- Salvetat ML, Zeppieri M, Tosoni C, Brusini P., Medscape. Baseline factors predicting the risk of conversion from ocular hypertension to primary open-angle glaucoma during a 10-year follow-up. *Eye (Lond)*. 2016 Jun;30(6):784-95.
- 15- Kim KE, Kim MJ, Park KH, et al.: Prevalence, awareness, and risk factors of primary open-angle glaucoma: Korea National Health and Nutrition Examination Survey 2008–2011. *Ophthalmology* 2016; 123: 532–41.
- 16- Heijl A, Bengtsson B, Oskarsdottir SE: Prevalence and severity of undetected manifest glaucoma: results from the early manifest glaucoma trial screening. *Ophthalmology* 2013; 120: 1541–5.
- 17- Buffault J, Brignole-Baudouin F, Reboussin É, Kessal K, Labbé A, Mélik Parsadaniantz S, Baudouin C. The Dual Effect of Rho-Kinase Inhibition on Trabecular Meshwork Cells

- Cytoskeleton and Extracellular Matrix in an In Vitro Model of Glaucoma. *J Clin Med.* 2022 Feb 15;11(4)
- 18- Zhou R, Sun Y, Chen H, Sha S, He M, Wang W. Laser Trabeculoplasty for Open-Angle Glaucoma: A Systematic Review and Network Meta-Analysis. *Am J Ophthalmol.* 2021 Sep;229:301-313.
- 19- Anderson DR., Normal Tension Glaucoma Study. Collaborative normal tension glaucoma study. *Curr Opin Ophthalmol.* 2003 Apr;14(2):86-90.
- 20- Bai HQ, Yao L, Wang DB, Jin R, Wang YX. Causes and treatments of traumatic secondary glaucoma. *Eur J Ophthalmol.* 2009 Mar-Apr;19(2):201-6