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GLAUCOMA

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Introduction

Glaucoma is a complex eye condition characterized by elevated intraocular pressure (IOP) that may progress to vision loss over time. Glaucoma is the second leading cause of permanent blindness in the United States and occurs most often in older adults. Glaucoma can be categorized into either primary or secondary types and further into open-angle or closed-angle variants within each type of glaucoma. Adult glaucoma includes primary open-angle glaucoma (POAG) and angle-closure glaucoma, as well as secondary open and angle-closure glaucoma, with a specific focus on the most prevalent type, POAG.

Glaucoma is an acquired loss of retinal ganglion cells and axons within the optic nerve or optic neuropathy that results in a characteristic optic nerve head appearance and a corresponding progressive loss of vision. This unique pattern of peripheral vision loss serves as a distinguishing feature from other types of vision impairment. Patients with POAG are often asymptomatic until significant optic nerve damage occurs unless early signs of glaucoma are identified during routine eye examinations. On the other hand, acute angle-closure glaucoma can develop suddenly and lead to a rapid decline in vision, accompanied by symptoms such as corneal edema, eye pain, headache, nausea, and emesis. Secondary glaucoma often arises due to a previous eye injury or underlying medical conditions, resulting in elevated IOP and subsequent optic neuropathy. This category

encompasses various subtypes, including congenital, pigmentary, neovascular, exfoliative,
traumatic, and uveitic glaucoma. Normal or low-tension type of glaucoma presents as an optic neuropathy with glaucomatous visual loss despite normal or unremarkable IOP readings.

Although congenital, infantile, and developmental glaucoma, along with a juvenile variant of POAG, primarily affect younger individuals, most types of glaucoma are commonly diagnosedin individuals aged 40 and older. While IOP is often associated with glaucoma, a direct causal relationship has not been definitively established. Researchers are investigating genetic and environmental factors contributing to glaucoma development. Evidence from studies involving monozygotic twin pairs, who exhibit a higher concordance rate compared to dizygotic pairs, suggests that environmental factors also have a significant role in the disease's development. Although the available treatments cannot cure existing optic nerve damage or reverse visual field loss, they can help control the disease progression through medication, laser treatment, or incisional glaucoma surgeries to prevent further vision loss. All therapeutic interventions are focused on lowering IOP and minimizing the impact of this vision-threatening condition. This approach aims to prevent the onset of glaucoma in patients with risk factors and to manage the condition effectively to limit its progression in affected individuals.

Etiology

The exact etiology of glaucoma is unknown, but a clear correlation with elevated eye pressure
exists in most cases. High IOP is the primary risk factor for developing glaucoma and for the
progression of the disease, and it is also the sole factor that current treatments can effectively
address.

Primary Open-Angle Glaucoma

POAG typically manifests as slow, painless damage to the optic nerve due to an ineffective

drainage system in the eye. In glaucoma, the resistance to drainage of aqueous humor most commonly starts at the inner wall of Schlemm canal at the juxtacanalicular trabecular meshwork.

This decreased outflow facility or increased resistance to aqueous outflow results in a gradual rise in IOP, leading to characteristic damage patterns in the visual field and the optic nerve ganglion cell nerve fiber layer. Recent studies have highlighted that elevated IOP can also reduce blood flow to the optic nerve fibers, resulting in subtle ischemic damage (see Image. Glaucomatous Optic Nerve Head Showing Inferotemporal Retinal Nerve Fiber Layer Defect).

POAG patients often have elevated IOP readings correlating with characteristic optic nerve damage and visual field defect patterns (see Image. Glaucoma Visual Field Changes in the Left Eye). As the disease progresses, a slow loss of peripheral vision in one or typically both eyes eventually leads to loss of central vision. Because of this loss pattern, affected persons do not notice a change in their vision until their loss is advanced and affects the central vision, in which case damage is permanent and irreversible.

Glaucoma can manifest at different ages, with the age of onset often characterizing its presentation. Although POAG is typically associated with adulthood, it can also affect younger individuals and children, suggesting a significant genetic component. Primary congenital

glaucoma is diagnosed in newborns aged up to 1 month, often suspected when there is eye
enlargement at birth. Infantile glaucoma affects individuals between the ages of 1 and 36
months, while juvenile glaucoma is used to indicate individuals diagnosed with glaucoma
between the ages of 3 and 40. Juvenile open-angle glaucoma shares similarities with POAG in terms of IOP leading to optic nerve damage, but it occurs in a younger age group with higher
IOP levels and potentially more severe visual field defects.

Low-Tension or Normal-Tension Glaucoma

This type of glaucoma resembles POAG in terms of characteristic optic disc cupping and
peripheral visual-field loss findings. However, what sets it apart is that IOP readings are
consistently normal, typically measuring less than 21 mmHg. Theories suggest that patients
with this type of glaucoma may have an optic nerve that is abnormally sensitive to pressure or
may experience intermittent ischemic changes due to atherosclerosis or vascular insufficiency.
These patients often exhibit a higher prevalence of migraines, Raynaud phenomenon,
autoimmune diseases, ischemic vascular diseases, and coagulopathies. This observation may
suggest the involvement of a vascular autoregulatory defect in the pathogenesis of the
disease. In addition, these patients tend to have a greater frequency of nerve
fiber layer hemorrhages and a neuroretinal rim that is thinner inferiorly and inferotemporal than
those with POAG. Visual field defects in this type of glaucoma are typically more focal, deeper,

and closer to fixation rather than following the classic arcuate scotoma pattern seen in open-
angle glaucoma.

Angle-Closure Glaucoma

Angle-closure glaucoma is classified based on ocular anatomy and can manifest as a medical
emergency in the acute setting or as a chronic condition. In the acute form, this type of
glaucoma occurs when the eye's drainage system is abruptly blocked due to the closure of the
angle formed between the cornea and the iris. Typically, this blockage arises from age-
related lens thickening, leading to a gradual increase in a relative pupillary block that pushes the
iris anteriorly. This anteriorly displaced iris, coupled with a natural anatomical variation such as
a smaller angle seen in hypermetropia or specific ethnic groups, predisposes easier blockage of
the outflow tract.

A pupillary block is considered the underlying cause in the majority of cases. When sudden
pupil dilation occurs due to certain stimuli, darkness, or drugs, the iris is thick enough in its
contracted state or anteriorly displaced by pupillary block to block fluid drainage via the
trabecular meshwork. The pressure rapidly increases within the eye. This rapid change in IOP
can cause central and/or peripheral vision loss within a few days of onset without intervention
and very high IOP. A significantly elevated IOP and acute angle closure can lead to

complications such as retinal vascular occlusion, ischemic optic neuropathy, or glaucomatous
optic nerve damage. However, it is important to note that only about 10% of glaucoma cases fall
into the acute angle-closure type category.

Angle-closure glaucoma can also occur as a secondary condition due to various causes.
Examples include lens subluxation in Marfan syndrome, lens dislocation, and lens-induced glaucoma. The displacement of the lens into the pupil or anterior chamber can lead
to an acute pupillary block. Plateau iris configuration can also cause an acute pupillary block
and chronic angle closure, attributed to elongated or anteriorly positioned ciliary processes
pushing the iris edges forward. In iridocorneal endothelial syndrome, irregular corne endothelium migration onto the trabecular meshwork and peripheral iris can lead to high peripheral anterior synechiae, closing the angle and hindering outflow.

Neovascularization can cause angle closure in neovascular glaucoma by forming a fibrovascular
membrane that flattens and displaces the iris anteriorly. This process, along with new vessel
formation in the iris and angle in rubeosis iridis, can lead to total synechial closure of the
angle.The most common etiologies of neovascular glaucoma are central retinal or branch
retinal vein occlusion, proliferative diabetic retinopathy, and ocular ischemic
syndrome. Angle-closure glaucoma can also occur post-ophthalmic surgery due to factors
such as ciliary body edema, scleral buckle placement, fibrin deposition, gas, or silicone oil used
in retinal surgery.Additionally, sulfa drugs such as topiramate can induce angle closure by

causing ciliochoroidal effusion, which compresses the lens-iris diaphragm and anteriorly displaces it, resulting in angle closure.

Pathophysiology

The optic nerve carries over 1 million nerve fibers that transmit visual signals from the photoreceptors in the outer retina to the visual processing areas of the occipital lobe. Damage to the retinal nerve fiber layer occurs in various types of glaucoma. Aqueous humor, the fluid in the anterior chamber of the eye, is crucial in maintaining intraocular pressure and nourishing ocular tissues. Aqueous humor is produced by the non-pigmented epithelial cells of the ciliary body processes and follows a circadian production pattern. Aqueous humor drains continuously through the pupil, then via the trabecular meshwork anterior to the scleral spur and iris insertion, into Schlemm canal, and finally into the episcleral venous system, larger orbital venous system, and systemic venous circulation. The trabecular meshwork consists of multiple layers of connective tissue and the endothelium of Schlemm canal, forming the primary drainage pathway for aqueous humor.

The conventional outflow pathway regulates fluid drainage from the eye in a pressure-dependent manner, acting as a one-way valve for aqueous humor drainage. In contrast, the uveoscleral outflow pathway allows pressure-independent passage of aqueous humor through the ciliary

muscle and iris root into the supraciliary and suprachoroidal space. This pathway is believed
to experience reduced outflow with age. Over time, there is a decline in aqueous humor drainage
through the trabecular meshwork, while the production of aqueous humor by the ciliary body
decreases slightly. This imbalance between outflow and aqueous production leads to elevated
average IOP and larger diurnal fluctuations in IOP, which are common features in patients with
glaucoma.

Prolonged elevation of IOP leads to the death and atrophy of nerve fibers, resulting in a "cupped"
or curved appearance of the optic nerve head observed during fundoscopy. The normal
range of IOP is approximately 16±3 mmHg, with values typically falling between 12 and 21
mmHg. However, IOP fluctuates throughout the day due to various factors including heart
rate, respiration, exercise, hydration status, systemic medications, time of day, alcohol intake,
patient posture, and use of topical medications.

Elevated pressure readings during screening, surpassing 21 mmHg, indicate pressures beyond
normal physiological levels and raise concerns about potential future damage to the optic nerve
due to glaucoma. However, it is challenging to determine if patients experience transient spikes
in pressure during the day, leading to damage that remains undetected during screening.

Consequently, elevated screening pressure serves only as a risk factor for developing glaucoma

and is not sufficient for a glaucoma diagnosis. Continuous monitoring of IOP throughout the day (diurnal pressure monitoring) may aid in identifying individuals at risk in this patient
population.

Patients diagnosed with normal-tension glaucoma often exhibit systemic vascular conditions
such as the Raynaud phenomenon, migraines, sleep apnea, carotid artery disease, and significant
nocturnal blood pressure fluctuations. In acute angle-closure glaucoma, the trabecular
meshwork drainage pathway becomes obstructed due to the iris being pushed forward from
pressure, such as an anteriorly displaced lens, or by fibrous tissue pulling the iris forward. The
most common cause is a pupillary block, where the iris dilates to a mid-position, bows anteriorly
upon contact with the lens, and obstructs the trabecular meshwork, thereby impeding aqueous
outflow. Secondary glaucoma, as mentioned earlier, can arise from various causes such as
surgery or neovascularization, leading to blockage of the outflow tracts and subsequent elevation
of IOP, potentially resulting in glaucomatous optic nerve damage if left untreated.

Evaluation

Assessment includes a fundoscopic examination, visual field testing, tonometry, optical
coherence tomography (OCT), and gonioscopy. Of these, IOP is the greatest risk factor, thereby
making tonometry of utmost importance. Goldmann applanation tonometry is the gold
standard for patients with risk factors, elevated IOP, and glaucoma, although several other types

of tonometers are available. Alternative tonometers can be considered when Goldmann applanation tonometry is impractical, including bedridden patients, noncollaborative individuals, children, or those allergic to anesthetic drops.

Additional helpful tests in glaucoma evaluation include assessing visual acuity to determine any impact on vision, pachymetry to measure corneal thickness, and retinal scans to monitor progressive changes in the retinal nerve fiber layer. Regular visual field testing using full-threshold strategies is crucial for individuals with risk factors, ocular hypertension, and those undergoing glaucoma treatment. OCT is valuable for monitoring morphological changes in the optic nerve and retinal nerve fiber layer, especially in patients with ocular hypertension and early-to-moderate glaucoma.

Glaucoma diagnosis relies on identifying progressive optic neuropathy and/or visual field defects, often in conjunction with elevated IOP. Ocular hypertension is diagnosed in individuals with IOP levels exceeding 21 mmHg without signs of glaucomatous optic neuropathy or functional visual field defects. Research indicates that around 20% of people with ocular hypertension may progress to glaucoma, highlighting the importance of regular testing, tonometry, and comprehensive eye examinations to initiate appropriate treatment aimed at reducing IOP in the presence of initial glaucomatous damage.

A single gold standard test does not exist for diagnosing glaucoma. Typically, glaucoma is

diagnosed during routine eye examinations, as the disease is often asymptomatic without noticeable vision loss. Clinicians rely on recognizing the characteristic appearance of the optic nerve, assessing risk factors, and interpreting ancillary test results to establish an accurate diagnosis and stage of glaucoma.Currently, the American Academy of Ophthalmology recommends routine comprehensive eye examinations for individuals with glaucoma risk factors, with the examination frequency tailored to factors such as age, race, family history, and specific risk factors.

Treatment / Management

Managing glaucoma involves personalized strategies based on the type and severity of the condition. Available treatments cannot reverse vision loss; they aim to lower IOP, a key risk factor, to prevent further damage and vision loss. Therapeutic options such as eye drops, laser procedures, and surgeries are focused on reducing IOP. Monitoring disease progression involves using tools like tonometry, visual field tests, OCT, and vision loss mapping.

Open-angle glaucoma is typically managed initially with medications aimed at reducing eye pressure. Common medication classes include prostaglandin analogs, β-blockers, carbonic anhydrase inhibitors, α-2 agonists, miotic agents, and more recently, Rho-kinase inhibitors and nitric oxide-donating medications. Laser trabeculoplasty, such as argon laser

trabeculoplasty, selective laser trabeculoplasty, and multipulse laser trabeculoplasty, may also be considered in certain cases. However, the benefits of lowering IOP with laser trabeculoplasty often last several months, and retreatments are commonly necessary.

If medical and/or laser management is unsuccessful, procedures such as trabeculectomy, deep sclerectomy, canaloplasty, insertion of a drainage valve/tube shunt, and laser treatment to the ciliary body to reduce aqueous production can help achieve better control of IOP. Minimally invasive glaucoma surgery (MIGS) is an emerging option for individuals with mild-to-moderate glaucoma. Compared to traditional trabeculectomy and tube shunts, MIGS offers a more favorable safety profile, quicker recovery time, and effective reduction of IOP to the mid-high teens. Studies also indicate that MIGS placement can decrease the number of pressure-lowering medications needed to maintain target IOP levels.

Normal-tension glaucoma can be managed with medications to reduce IOP and address any underlying medical conditions. Treatment options include prostaglandin analogs, α-2 agonists, carbonic anhydrase inhibitors, and miotics. The use of β-blockers is debated due to concerns about reduced optic nerve head perfusion, particularly regarding the potential exacerbation of the early morning nadir in blood pressure. If medical therapy proves ineffective, laser trabeculoplasty or filtration surgery may be necessary, especially in cases of progressive vision loss. The collaborative normal-tension glaucoma study demonstrated that patients with this

condition can slow or stabilize their field loss after achieving a 30% reduction in IOP.

Angle-closure glaucoma is considered a medical emergency due to the potential for elevated pressures leading to glaucomatous optic nerve damage, ischemic nerve damage, or retinal vascular occlusion. Patients can take medications to reduce eye pressure as quickly as possible but usually require a laser procedure called laser peripheral iridotomy. This procedure involves creating a small hole in the iris to alleviate pupillary blockage. By equalizing the pressure gradient between the posterior and anterior chambers, laser iridotomy resolves the iris bombe and opens up the drainage angle in the anterior chamber, relieving the condition. The peripheral iris can be flattened with laser iridoplasty and, less commonly, with laser pupilloplasty.

A decrease in IOP does not always indicate that the angle has reopened. Ischemic damage to the ciliary body during an attack can reduce aqueous humor production for several weeks. Therefore, it is crucial to perform a follow-up gonioscopy to confirm angle patency. This evaluation also helps assess the percentage of the angle with peripheral anterior synechia from acute or prior subacute attacks. After resolving the acute crisis, patients are at a high risk of experiencing an attack in the contralateral eye. Therefore, they should undergo gonioscopy to assess the angle and consider prophylactic iridotomy in the other eye if the angle is narrow. Treatment for

secondary glaucoma should focus on addressing the underlying cause along with the possible
inclusion of medications to reduce IOP.

REFERENCES

1. Andryev S. et al. Experience with the use of memantine in the treatment of cognitive disorders //Science and innovation. – 2023. – T. 2. – №. D11. – C. 282-288.
2. Antsiborov S. et al. Association of dopaminergic receptors of peripheral blood lymphocytes with a risk of developing antipsychotic extrapyramidal diseases //Science and innovation. – 2023. – T. 2. – №. D11. – C. 29-35. 3. Asanova R. et al. Features of the treatment of patients with mental disorders and cardiovascular pathology //Science and innovation. – 2023. – T. 2. – №. D12. – C. 545-550.
4. Begbudiyev M. et al. Integration of psychiatric care into primary care //Science and innovation. – 2023. – T. 2. – №. D12. – C. 551-557.
5. Bo'Riyev B. et al. Features of clinical and psychopathological examination of young children //Science and innovation. – 2023. – T. 2. – №. D12. – C. 558-563.
6. Borisova Y. et al. Concomitant mental disorders and social functioning of adults with high functioning autism/asperger syndrome //Science and innovation. – 2023. – T. 2. – №. D11. – C. 36-41.
7. Ivanovich U. A. et al. Efficacy and tolerance of pharmacotherapy with antidepressants in non-psychotic depressions in combination with chronic brain ischemia //Science and Innovation. – 2023. – T. 2. – №. 12. – C. 409-414.
8. Nikolaevich R. A. et al. Comparative effectiveness of treatment of somatoform diseases in psychotherapeutic practice //Science and Innovation. – 2023. – T. 2. – №. 12. – C. 898-903.
9. Novikov A. et al. Alcohol dependence and manifestation of autoagressive behavior in patients of different types //Science and innovation. – 2023. – T. 2. – №. D11. – C. 413-419.
10. Pachulia Y. et al. Assessment of the effect of psychopathic disorders on the dynamics of withdrawal syndrome in synthetic cannabinoid addiction //Science and innovation. – 2023. – T. 2. – №. D12. – C. 240-244.