THE DIAGNOSIS AND TREATMENT OF GLAUCOMA

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Abstract

Background Glaucoma is a group of chronically progressive disorders of the optic nerve. In this article, we present the epidemiology of and risk factors for glaucoma, as well as the diagnostic work-up and treatment options.

Methods

This review is based on pertinent publications retrieved by a selective search in Medline and the Cochrane Library, supplemented by further articles chosen by the authors.

Results

In Central Asia, the prevalence of glaucoma is 2.93% among persons aged 40 to 80 years. The prevalence rises with age, reaching 10% in persons over 90 years old. The available diagnostic methods include ophthalmoscopy, tonometry, perimetry, and imaging techniques. The treatment of glaucoma is focused on lowering the intraocular pressure with topical drugs, laser therapy, and glaucoma surgery. In patients with manifest glaucoma, lowering the intraocular pressure prevents the progression of visual field defects, with a number needed to treat of 7.

Conclusion

The diagnostic evaluation of glaucoma rests on multiple pillars, all of which must be considered for establishing the diagnosis and defining the desired target pressure: these are, among others, the intraocular pressure and ocular function and morphology. Individually tailored pressure-lowering treatment should be evaluated in regularly scheduled follow-up visits for assessment of function and morphology and adjusted as necessary to minimize the risk of progression.

Glaucoma (from the Greek *glaukós,* a nonspecific term for green or light gray is a group of disorders that differ in their pathophysiology, risk factors, manifestations, treatments, and prognoses. Their common feature is progressive degeneration of the optic nerve, with loss of retinal ganglion cells, thinning of the retinal nerve fiber layer, and progressive excavation of the optic disc .

Annotatsiya

Glaukoma - bu ko‘ruv nervining surunkali progressiv kasalliklari guruhi. Ushbu maqolada biz glaukomaning epidemiologiyasi va xavf omillarini, shuningdek, diagnostika va davolash usullarini taqdim etamiz.

Usullar

Ushbu sharh Medline va Cochrane kutubxonasida tanlab olingan tegishli nashrlarga asoslangan bo‘lib, mualliflar tomonidan tanlangan qo‘shimcha maqolalar bilan to‘ldirilgan.

Natijalar

Markaziy Osiyoda glaukomaning tarqalishi 40 yoshdan 80 yoshgacha bo‘lgan odamlar orasida 2,93% ni tashkil qiladi. Yosh ulg‘aygan sari tarqalish darajasi ortib boradi va 90 yoshdan oshgan odamlarda 10% ga yetadi. Oftalmoskopiya, tonometriya, perimetriya va vizualizatsiya usullari mavjud. Glaukomani davolash mahalliy dorilar, lazer terapiyasi va glaukoma jarrohligi yordamida ko‘z ichi bosimini pasaytirishga qaratilgan. Yaqqol namoyon bo‘lgan glaukoma bilan og‘rigan bemorlarda ko‘z ichi bosimini pasaytirish ko‘rish maydoni nuqsonlarining rivojlanishiga to‘sqinlik qiladi, davolash uchun zarur bo‘lgan soni 7 tani tashkil qiladi.

Xulosa

Glaukomaning diagnostik bahosi bir nechta ustunlarga asoslanadi, ularning barchasi tashxis qo‘yish va kerakli maqsadli bosimni aniqlash uchun hisobga olinishi kerak: bular boshqalar qatori ko‘z ichi bosimi va ko‘z funksiyasi va morfologiyasidir. Bosimni pasaytiruvchi individual davolash funksiyasi va morfologiyasini baholash uchun muntazam rejalashtirilgan keyingi tashriflarda baholanishi va rivojlanish xavfini minimallashtirish uchun zarur bo‘lganda tuzatilishi kerak.

Glaukoma (yunoncha glaukós, yashil yoki och kulrangning o‘ziga xos bo‘lmagan atamasi patofiziologiyasi, xavf omillari, namoyon bo‘lishi, davolash va prognozi bilan farq qiladigan kasalliklar guruhidir. Ularning umumiy xususiyati ko‘ruv nervining progressiv degeneratsiyasi, to‘r parda gangliy hujayralarining yo‘qolishi, to‘r parda nerv tolalari qatlamining yupqalashishi va ko‘ruv nervi diskining progressiv ekskavatsiyasidir.

Keywords: glaucoma, glaucomadrainageimplants, surgical , adult.

Kalit so’zlar: glaukoma, glaukomadrainajimplantlar, jarrohlik, katallar.

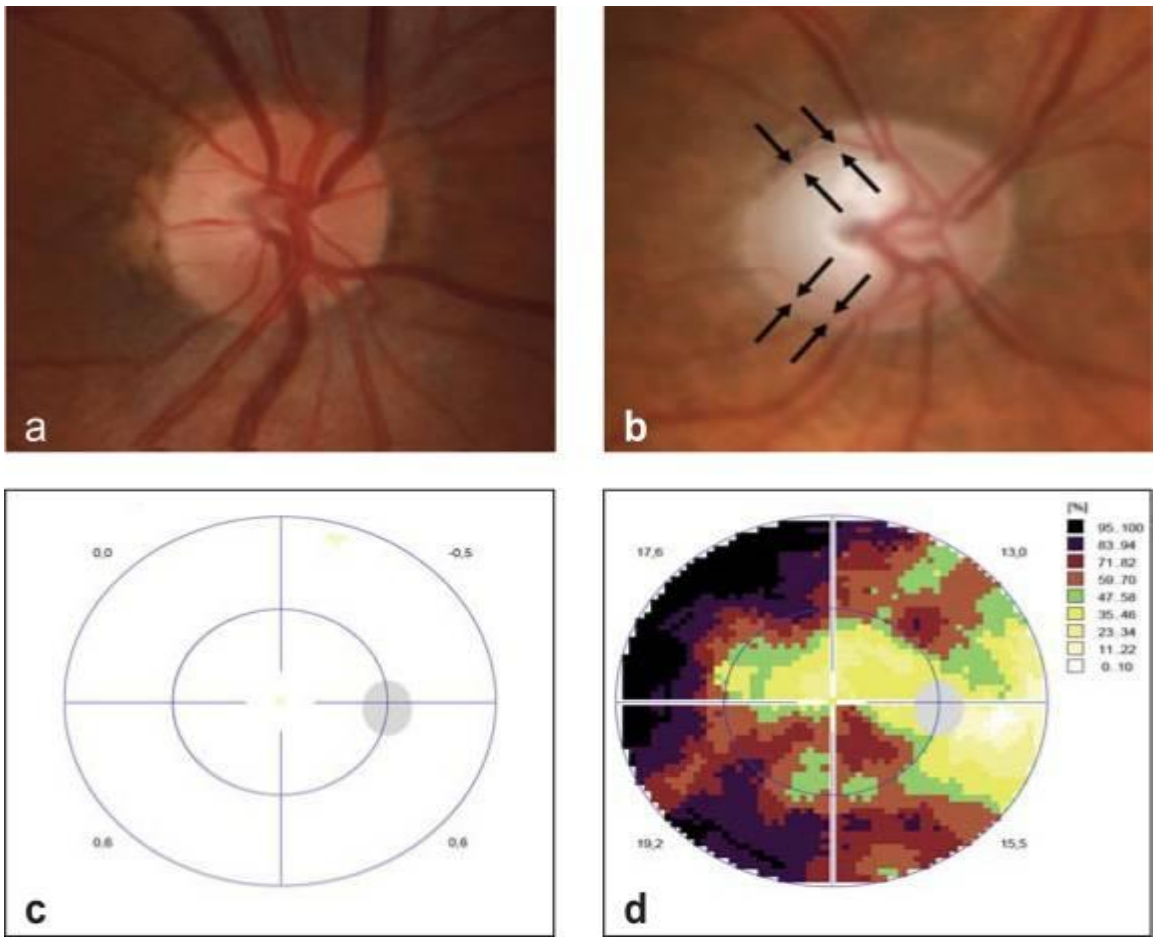


Figure 1.

Optic disc images: a) normal and b) glaucomatous optic disc; c) and d) the respective visual field measurements. The findings from one eye (the right eye) are shown in all images. The optic disc in b) displays, particularly in the upper and lower temporal quadrants, marked thinning of the neuroretinal edge zone (black arrows), with a large excavation. The corresponding visual field examination d) reveals marked defects with central sparing. Darker shading represents the areas in which light is less well perceived; a normal visual field is shown for comparison (c). Visual field measurements depend on the patient's concentration and cooperation, which can be quantified, for example, with automatic fixation detection and trick questions.

Definition.

Glaucoma is a group of disorders whose common feature is progressive degeneration of the optic nerve, with loss of retinal ganglion cells, thinning of the retinal nerve fiber layer, and increasing excavation of the optic disc.

Learning objectives

After reading this article, the reader should know:

- How the various types of glaucoma differ from one another
- How a targeted diagnostic evaluation should be structured
- What treatment options are available for each of the disease entities

Pathophysiology.

Elevated intraocular pressure and low perfusion pressure increase the gradient across the lamina cribrosa and cause papillary hypoperfusion, leading to structural changes and remodeling of the lamina cribrosa and to impaired axonal transport in the optic nerve fibers.

Resulting disturbances.

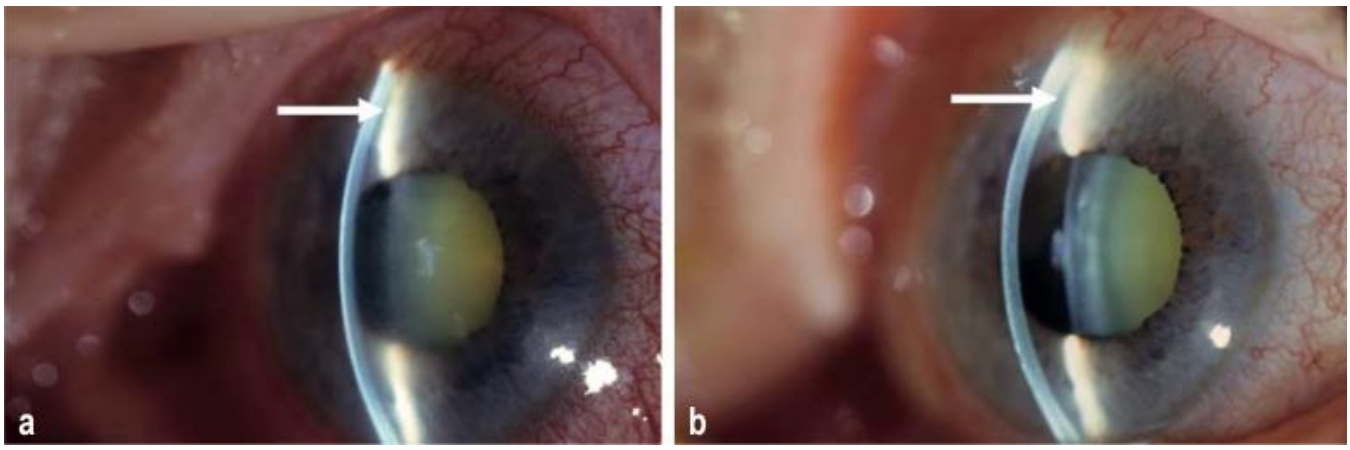
The progressive loss of retinal ganglion cells leads to increasing impairment of the visual field. Further functional disturbances include impaired contrast and color perception and difficulty in reading.

The retinal ganglion cells are neurons of the central nervous system that receive signals from the photoreceptors, process them, and transmit them in axons through the optic nerve to further centers in the brain. These axons run from the ganglion cell nuclei in the retina to the optic disc , and then together with the retinal vessels through the lamina cribrosa, a sieve-like structure composed of collagen. Behind the lamina cribrosa, the axons, surrounded by a myelin sheath, continue as the optic nerve. Elevated intraocular pressure, low perfusion pressure, and/or low cerebrospinal fluid pressure increase the gradient across the lamina cribrosa and cause papillary hypoperfusion, leading to structural changes and remodeling of the lamina cribrosa and to impaired axonal transport in the optic nerve fibers . In particular, the pores in the anterior region of the lamina cribrosa are elongated in open-angle glaucoma .

The increasing loss of retinal ganglion cells leads to progressive impairment of the visual field, generally beginning in the mid-periphery and then advancing until only a central or peripheral island of intact vision remains. Further functional disturbances include impaired contrast and color perception and difficulty in reading . The mechanisms by which retinal ganglion cells are lost are not yet fully understood.

The different types of glaucoma are classified according to the respective structural changes in the anterior segment of the eye. The aqueous humor is mainly drained in the chamber angle via the trabecular meshwork and the canal of Schlemm, and partly via the uveoscleral outflow (root of the iris, ciliary body). The chamber angle lies between the iris and the peripheral posterior surface of the cornea, and at its end the canal of Schlemm lies under the trabecular meshwork. While in open-angle glaucoma the chamber angle is macroscopically open, in acute angle closure it is occluded by the iris (figure 2); this suddenly blocks the outflow of aqueous humor via the trabecular meshwork and the canal of Schlemm, causing a marked elevation of intraocular pressure. In secondary open-angle glaucoma, there are changes of the chamber angle that are visible under the microscope (gonioscope), such as pigment deposition (in pigmentary glaucoma) or protein deposition (in pseudoexfoliation glaucoma) , that elevate the intraocular pressure. The mechanisms leading to elevated intraocular pressure in primary open-angle glaucoma are not fully understood.

Figure 2.



Slit-lamp examination a) of an occluded chamber angle in acute angle closure and b) after successful treatment (laser iridotomy). There is marked enlargement of the chamber angle (white arrow) and deepening of the anterior chamber from a) to b), as well as corneal clearing and regression of conjunctival hyperemia and scleral vascular distention.

Chamber angle.

The chamber angle lies between the iris and the peripheral posterior surface of the cornea, and at its end the canal of Schlemm lies under the trabecular meshwork.

The normal intraocular pressure has an average value of 15.7 mm Hg but displays marked chronobiological and interindividual variation even in healthy persons. It is regulated by the balance between the secretion of aqueous humor by the ciliary body and its outflow. Central Asia intraocular pressure may thus be a consequence of increased outflow resistance. This can be due to gonioscopically visible changes in the chamber angle in secondary open-angle glaucoma, as mentioned above, but it can also arise without any such changes, as in primary open-angle glaucoma. Glaucomatous changes in the optic nerve may arise even when the intraocular pressure is within normal limits (normal-pressure glaucoma). Among persons of European ancestry, the intraocular pressure is normal in 30% of all cases of glaucoma , with regional variation in prevalence. This disorder is apparently caused by an intraocular pressure that, although within normal limits, nonetheless exceeds the pressure sensitivity of the optic disc. The importance of the pressure sensitivity of the optic disc is also indicated by the fact that a 25% pressure reduction lowers the risk of glaucoma progression by 50% . Moreover, vascular changes seem to play a role in the pathophysiology of open-angle glaucoma, and of normal-pressure glaucoma in particular , e.g., an excessive nocturnal drop in blood pressure in otherwise normotensive persons .

Risk factors

- Advanced age
- Elevated intraocular pressure

- High myopia
- A positive family history of glaucoma

The risk also depends on ethnicity . Moreover, excavation of the optic disc is particularly hard to assess in highly myopic eyes. It is thought that enlargement of the optic disc due to myopia, with consequent thinning of the lamina cribrosa, may predispose to glaucoma . Increased shear forces in the lamina cribrosa caused by eye movements in persons with highly myopic (long) eyes have been mentioned as another possible pathogenetic factor .

Elevated intraocular pressure, or an elevated translaminar pressure gradient , is the sole modifiable risk factor for open-angle glaucoma that has been identified so far. The randomized, controlled Ocular Hypertension Treatment Study led to the conclusion that lowering elevated intraocular pressure (21–32 mm Hg) by 22.5% can decrease the 5-year risk of developing open-angle glaucoma from 9.5% to 4.4% .

Diagnostic evaluation

Symptoms

Acute angle closure can manifest itself with pain radiating from the eye, visual impairment, conjunctival hyperemia, and sometimes nausea and vomiting with a tense, rock-hard globe. This is an ophthalmological emergency that demands immediate treatment to prevent severe ocular damage and blindness.

Risk reduction.

Lowering an elevated intraocular pressure (21–32 mm Hg) by 22.5% can lower the 5-year risk of developing open-angle glaucoma from 9.5% to 4.4%.

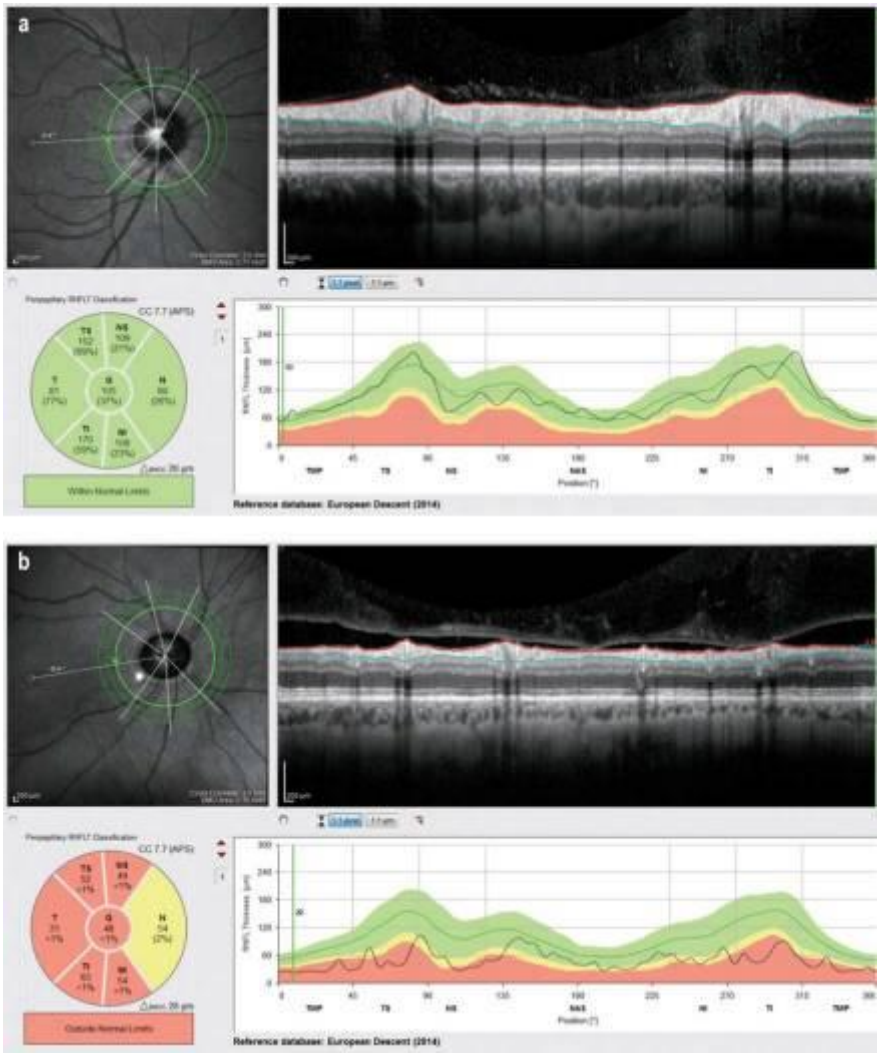
In contrast, open-angle glaucoma usually does not become symptomatic until it has reached an advanced stage. If visual field defects are present, they usually do not lie in the same part of the fields of the two eyes and are therefore well compensated by binocular vision. Thus, persons with open-angle glaucoma generally report no symptoms , and many are completely unaware that they have the condition . One-third of patients already have the condition in an advanced or late stage in at least one eye at the time of diagnosis . Gramer et al. reported that 10–20% of patients were already unable to drive a vehicle at the time of presentation at the clinic because of binocular visual field defects .

Early detection

As this condition only becomes symptomatic when it has reached an advanced stage, the German ophthalmological associations recommend regular screening examinations for early detection from age 40 onward . Because of the low prevalence of the disorder (and the low sensitivity and specificity of the tests , The rate of false positives is high (> 65%, and even higher in younger patients), and thus any positive finding must be followed up by further testing. Regular examination is especially important in risk groups with elevated incidence and prevalence of the disorder, so

that it can be diagnosed and treated early in its course. No randomized, controlled trials on this topic have yet been conducted. The recommended screening examination consists of at least a clinical history, stereoscopic examination of the papilla and peripapillary nerve layer, tonometry, and slit-lamp examination of the eye . Screening examinations for glaucoma are not covered by the statutory health insurance providers in Germany, nor is there any population-wide periodic screening for glaucoma in other European countries such as the UK, France, or the Netherlands.

Figure 4



Examination of peripapillary nerve fiber layer thickness a) in a normal eye and b) in a glaucomatous eye. The retinal nerve fiber layer lies between the internal limiting membrane (red line) and the border between the retinal nerve fiber layer and the ganglion cell layer (turquoise line). Beyond this layer, reflections from the vitreous body can be seen.

Topical treatment

Various substance classes are available for topical use to reduce the intraocular pressure. They differ in their mechanisms of action, in the degree to which they lower

the intraocular pressure , and in their dosing, side effects, and cost. A network meta-analysis on topical first-line drugs showed that the intraocular pressure is lowered to the greatest extent by prostaglandin analogs (bimatoprost by 5.61 mm Hg, latanoprost 4.85 mm Hg, travoprost 4.83 mm Hg, tafluprost 4.37 mm Hg), followed by beta-blockers (levobunolol 4.51 mm Hg, timolol 3.70 mm Hg, carteolol 3.44 mm Hg, levobetaxolol 2.56 mm Hg, betaxolol 2.24 mm Hg), alpha$_2$-adrenergic agonists (brimonidine 3.59 mm Hg, apraclonidine 2.52 mm Hg), and carbonic anhydrase inhibitors (dorzolamide 2.49 mm Hg, brinzolamide 2.42 mm Hg) .

Prostaglandin analogs are usually prescribed for the initial treatment and are applied once daily, in the evening. These drugs improve the uveoscleral and trabecular outflow and thereby lower the intraocular pressure. Their side effects include conjunctival hyperemia, increased growth of the eyelashes, reduction of periorbital fat, and increased pigmentation of the iris and periocular skin . Systemic conditions limiting the use of prostaglandin analogs include bronchial asthma, severe cardiovascular conditions, and diseases of the liver or kidneys.

Target range for intraocular pressure.

The target pressure is set individually and the further course of the intraocular pressure should be regularly checked to determine whether the disease has stabilized as desired.

Topically applied beta-blockers are an alternative. These are usually applied twice per day; they lower the intraocular pressure by diminishing the production of the aqueous humor. Their main local side effect is dry eye disease, or exacerbation of existing dry eye disease. Systemic contraindications include bronchial asthma, sinus bradycardia, second- or third-degree AV block, decompensated congestive heart failure, severe allergic rhinitis, cerebral hypoperfusion, and muscle weakness. Beta-blockers can exacerbate hyperglycemia and mask the symptoms of hypoglycemia in diabetic patients.

Alpha$_2$-adrenergic agonists lessen the secretion of aqueous humor and increase the uveoscleral outflow. The local side effects include initial white discoloration of the conjunctiva after the drops are applied, and, over the long term, topical intolerance in more than one-third of patients. Less commonly, there can be lid retraction, dry mouth, bradycardia, and fatigue . Simultaneous treatment with monoamine oxidase inhibitors, sympathomimetic drugs, or tricyclic antidepressants, which can affect noradrenergic transmission, is a systemic contraindication. Topical treatment with alpha$_2$-adrenergic agonists is contraindicated in children under 12 because of extremely severe side effects (ranging up to coma in toddlers). Conditions requiring special caution include bradycardia, hypotension, arteriosclerosis, and impaired hepatic or renal function. Topical carbonic anhydrase inhibitors likewise function by decreasing the production of aqueous humor; local undesired side effects include tearing, burning, and corneal endothelial decompensation. Miotic drugs can be a

further alternative, but are now hardly ever used as the treatment of first resort. Drugs from these different substance classes may be combined with one another, with due consideration of their side effect profiles and mechanisms of action. It is recommended that the eyes should be kept closed for a few minutes after the local, touch-free application of eyedrops in the lower conjunctival sac; if indicated, the tear ducts should be manually occluded with the index fingers. This lessens outflow of the drug through the tear duct system and resorption through the nasal mucosa, thereby reducing the chance of systemic side effects. Multiple studies have shown that patients tend to display poor compliance with local antiglaucomatous treatment.

Prostaglandin analogs.

These drugs improve the uveoscleral and trabecular outflow and thereby lower the intraocular pressure. Their side effects include conjunctival hyperemia, increased growth of the eyelashes, reduction of periorbital fat, and increased pigmentation of the iris and periocular skin.In the past, most eyedrops contained benzalkonium chloride as preservative, but in recent years multiple eyedrop preparations have been developed and approved without this ingredient (so-called preservative-free eyedrops). These have lessened the undesired side effects, conjunctival hyperemia in particular, and improved local tolerance . Artificial tears (e.g., hyaluronic acid preparations) are used to treat side effects such as dry eye.

Laser therapy

Laser therapy may be considered as a supplementary measure if local treatment does not adequately lower the intraocular pressure or fails to achieve the target pressure (e.g., because of lacking compliance with treatment). Laser therapy, however, generally results in a moderate lowering of the intraocular pressure, by way of increased aqueous humor outflow after laser trabeculoplasty or diminished aqueous humor production after cyclophotocoagulation . The latter lowers the intraocular pressure by at least 20% in 47% of the treated eyes ; its potential complications include inadequate or excessive pressure reduction, inflammation, and pupillary deformity, which may lead to highly bothersome glare. Micropulse laser techniques can be used for both applications as well, but their efficacy has not yet been fully documented.

Glaucoma surgery

Surgery is indicated if nonsurgical treatment options are insufficient to lower the intraocular pressure to the target pressure, or cause intolerable side effects. Minimally invasive, filtering, and non-filtering types of glaucoma surgery are available. For example, in one type of minimally invasive procedure a stent is placed in the canal of Schlemm to lower the outflow resistance through the trabecular meshwork. In general, this operation, which can be performed in combination with cataract surgery, does not lower the intraocular pressure enough, unless the glaucoma is only moderate ; in recent years, the surgical options have expanded markedly. Minimally invasive

glaucoma surgery seems to have fewer side effects than a filtering procedure , but also lowers the intraocular pressure by a lesser amount .

Laser therapy.

Laser therapy may be considered as supplementary treatment if local treatment does not adequately lower the intraocular pressure or fails to achieve the target pressure (e.g., because of non-compliance). In a filtering operation, an accessory pathway is created for the aqueous humor to flow out of the eye under the conjunctiva. Trabeculectomy is now considered the reference standard for this type of procedure. Various antimetabolites are applied intraoperatively and postoperatively to inhibit local conjunctival scarring . Patients with advanced glaucoma have less worsening of their visual fields if they are treated with trabeculectomy than if they undergo laser trabeculoplasty (hazard ratio [HR] = 3.95 in 10 years for Caucasian patients, HR = 1.62 in 10 years for patients with dark skin) . Other methods include deep sclerectomy and canaloplasty; these seem to have a lower risk of complications (cataract, endophthalmitis, etc.) . Treatments for acute angle closure, aside from intraocular pressure reduction with topical agents and systemic drugs (carbonic anhydrase inhibitors), include surgical procedures such as lentectomy with intraocular lens implantation or mechanical opening of the occluded angle (iridotomy, iridectomy) , which can be performed as an emergency procedure for persistent acute angle closure. The other eye should also be treated surgically shortly afterwards, as the risk of acute angle closure in the second eye is 51%, but can be reduced by successful treatment to 2% .

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