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Review Article A Review on Diagnosis, Types and Management of Glaucoma

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Abstract: *Glaucoma is a diverse collection of disorders distinguished by the cupping of the optic nerve head and visual field loss. It is the leading cause of irreversible blindness worldwide. When the intraocular pressure is reduced by 30-50% from the baseline, the progression usually ceases. The global age-standardised prevalence in the population aged 40 years or older is approximately 3.5 percent. Chronic forms of glaucoma are painless, and noticeable visual field abnormalities develop later. Early detection by ophthalmological examination is required. The most prevalent kind of glaucoma, primary open-angle glaucoma, is associated with older age, raised intraocular pressure, sub-Saharan African ethnic origin, a positive family history, and high myopia. The main risk factors for primary angle-closure glaucoma are old age, hyperopia, and East Asian ethnicity. Glaucoma is diagnosed using ophthalmoscopy, tonometry, and perimetry. Topical medications, laser therapy, and surgical intervention are used to treat intraocular pressure when other therapeutic methods fail to prevent progression.*

Keywords: Glaucoma, Open angle Glaucoma, Closed angle Glaucoma, IntraOcular Pressure (IOP) , Optic nerve

I. INTRODUCTION

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.(1) For those over 60, glaucoma is the most common cause of blindness. However, with early intervention, glaucoma-related blindness is frequently avoidable(2l A healthy eye's drainage angle allows fluid to exit the lens while maintaining constant pressure.(2)

II. CLASSIFICATION

To characterise the anatomic state of the anterior chamber angle, glaucomas can be divided into two categories: open-angle glaucomas (OAGs) and angle-closure glaucoma (ACGs)). Each of these is further subdivided into primary and secondary categories, which denote the existence or absence, respectively, of additional clinically recognized systemic or ocular illnesses that could be the cause of the glaucoma.

1. Open Angle Glaucoma :

This kind of glaucoma is the most prevalent kind. The progressive condition where the eye's fluid drainage is impaired (like to a plugged drain) occurs. As a result, the optic nerve begins to suffer damage as eye pressure increases. This kind of glaucoma first affects eyesight without causing any pain.Normal eye pressure might cause optic nerves in certain persons to become sensitive. This indicates that their risk of developing glaucoma is elevated. To detect early indications of optic nerve degeneration, routine eye exams are crucial.(2) A chronic, progressive, multifactorial optic neuropathy, open-angle glaucoma (OAG) is irreversibly caused.

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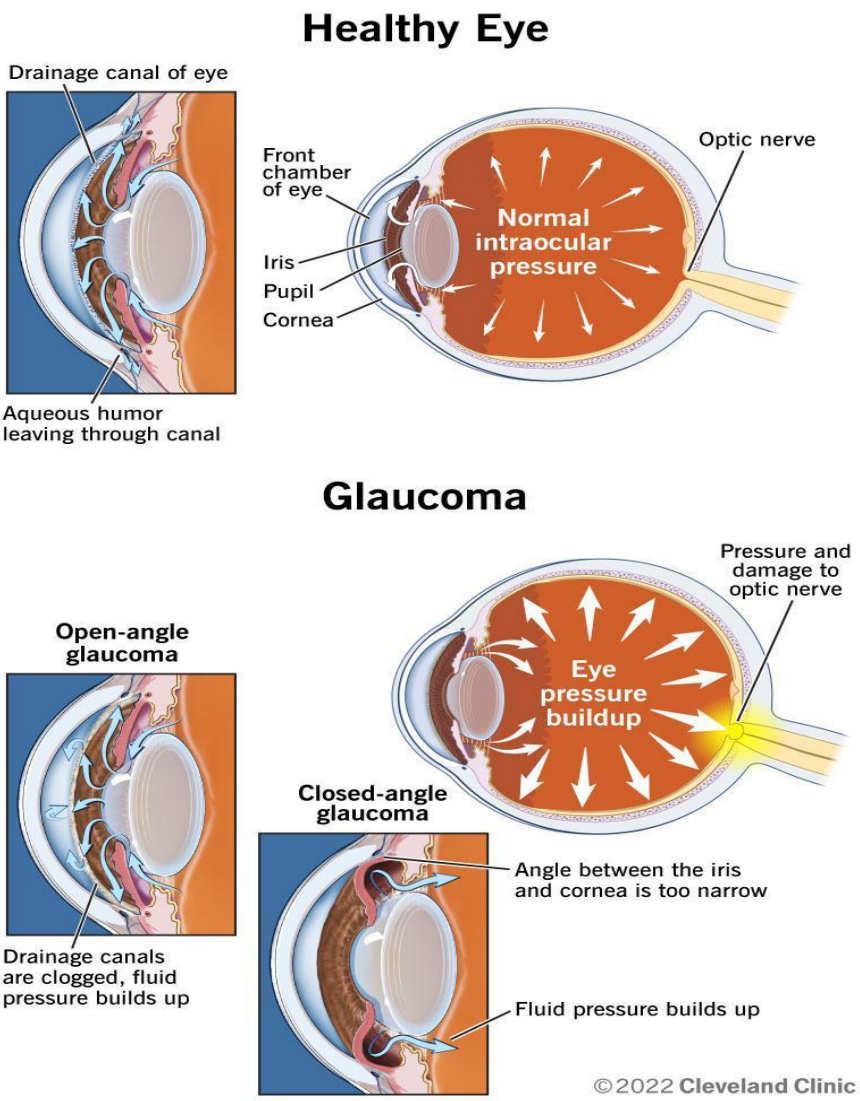

Figure 1. Healthy Eye & Eye With Glaucoma

Until it reaches an advanced stage, OAG is asymptomatic. This highlights the vital need for early detection and treatment. It is characterised by an open angle of the anterior chamber, alterations in the optic nerve head, and a progressive deterioration in peripheral vision followed by loss of central visual field. Whether from main or secondary causes, elevated intraocular pressure is a major risk factor. (3) In addition to describing how early detection and treatment can help prevent irreversible vision loss and maintain patients' quality of life, this course assesses the difficulties of aged eyesight. Together with its pathogenesis, risk factors, and clinical symptoms, participants examine the complex nature of OAG.(3) The ciliary body continuously produces aqueous humour in the posterior chamber of the eye and drains it into the anterior chamber; the majority of aqueous humour drainage occurs through the trabecular meshwork, and a small portion of aqueous outflow drains through the uveoscleral pathway. The most common type of glaucoma is called primary open-angle glaucoma (POAG), which is characterised by increased resistance to drainage in the trabecular meshwork, but the drainage angle between the cornea and iris remains open.[6] As a result of this blockage, the pressure inside the eye gradually increases, causing optic nerve damage and progressive visual loss. Secondary OAG can have multiple etiologies but is far less common than POAG.(3) Both typical and anomalous

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discharge of aqueous humour. (A) Normal outflow via uveoscleral channels (small arrow) and trabecular meshwork (big arrow), together with associated anatomy. Trabecular meshwork is where the majority of water flow occurs. The venous circulation of the eye drains each route. (B) There is less aqueous outflow via these channels in primary open-angle glaucoma. (C) The iris is atypically positioned in angle-closure glaucoma to obstruct aqueous outflow through the anterior chamber (iridocorneal) angle.

2. Closed Angle Glaucoma

Based on the structure of the eyes, angle-closure glaucoma is categorised as either a chronic illness or an acute medical emergency.(6) The acute form of glaucoma is caused by the closure of the angle established between the iris and cornea, which causes an abrupt blockage of the drainage system in the eye.(7) A relative pupillary block that gradually increases and pushes the iris anteriorly is usually the result of age-related lens thickening, which causes this blockage. Blockage of the outflow path is more likely in cases of anteriorly displaced iris combined with anatomical variations such as smaller angles observed in hypermetropia or in particular ethnic groups.(8) A pupillary block is considered the underlying cause in the majority of cases.(9) 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. (10) Inside the eye, the pressure builds up quickly. In the absence of intervention, this quick shift in IOP can result in very high IOP and central and/or optic neuropathy, or glaucomatous optic nerve injury might result from an acute angle closure and a markedly increased IOP. The acute angle-closure type of glaucoma only accounts for roughly 10% of cases, it is vital to remember this.(10) An angle-closure Glaucoma can also develop as a secondary condition for a number of reasons. Lens subluxation in Marfan syndrome , lens dislocation and lens-induced glaucoma are the examples.(11) (12) (13) An acute pupillary block may result from the lens being displaced into the pupil or anterior chamber.(14) Because the ciliary processes in a plateau iris arrangement force the iris edges forward, they can also result in acute pupillary block and chronic angle closure. (15) (16) Uneven corneal endothelium migration into the peripheral iris and trabecular meshwork can result in elevated peripheral anterior synechiae in iridocorneal endothelial syndrome, which closes the angle and prevents outflow.(17) (18) The formation of a fibrovascular membrane during neovascularization can result in angle closure in neovascular glaucoma by flattening and pushing the iris anteriorly. Complete synechial closure of the angle can result from this process, as well as new vessel creation in the iris and angle in rubeosis iridis.(19) The most common etiologies of neovascular glaucoma are central retinal or branch retinal vein occlusion, proliferative diabetic retinopathy, and ocular ischemic syndrome. (20) After ophthalmic surgery, angle-closure glaucoma may also develop as a result of ciliary body edema, the location of the scleral buckle, fibrin deposition, gas, or silicone oil used during retinal surgery.(21) Topiramate and other sulfa medications can also cause ciliochoroidal effusion, which compresses and displaces the lens-iris diaphragm anteriorly, leading to angle closure.(22)

III. EXAMINATION / DIAGNOSIS OF GLAUCOMA

As a result, patient awareness education is crucial for both diagnosis and treatment of glaucoma, as around 50% of people in resource-poor nations do not know they have the condition.(23, 26,27) The diagnosis of glaucoma involves risk assessment, measurement of visual acuity, IOP, and corneal thickness, OCT imaging of the retinal nerve fibre layer (RNFL) and ONH, and visual field testing. Because most patients with glaucoma are asymptomatic for years, it is recommended that those with risk factors (advanced age, family history, non-White race, high IOP, and steroid use) be referred to an eye care provider for a glaucoma assessment.(23, 24,25) Patients who are at an increased risk of developing glaucoma should have their intraocular pressure checked on a frequent basis. Rebound tonometry (iCare ic100; iCare) or Goldmann applanation tonometry, the "gold standard," are frequently used to measure it. While Goldmann applanation tonometry measures the force needed to flatten a piece of the cornea with a diameter of 3.06 mm, the iCare tonometer measures the IOP-dependent rebound velocity following brief corneal contact.(28) The two devices' measurements agree well, although the iCare's dependability declines with increasing intraocular pressure and thicker central corneas.(29, 30,31) The normal range for intraocular pressure (IOP) is 11 to 21 mm Hg(32); however, optic nerve abnormalities and/or high central cornea values should be taken into account when evaluating IOP.(33) IOP

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readings can be normal in up to 50% of glaucomatous eyes, which highlights the significance of obtaining further diagnostic imaging when necessary.(23, 34)

Because there is no universally accepted diagnostic criteria for glaucoma, diagnosing the condition can be challenging, especially in its early stages.(23) Visual field testing can identify functional alterations in advanced glaucoma, while OCT imaging of the optic nerve and macula can reveal structural changes in early glaucomaNormal appearances of the ONH, RNFL, and visual field are shown in Figure 2 . All glaucomas are defined by ONH degeneration with disc excavation and RNFL thinning. (35) The neuroretinal rim thins in optic nerve head injury, mainly in the superior and inferior quadrants, while the remaining portion of the ONH may still be pink with a normal neuroretinal rim.(23, 35) The retinal ganglion cells narrow between the ganglion cell layer and internal limiting membrane on Optical Coherence Tomography (OCT) due to apoptosis brought on by glaucomatous damage.(35) Visual field deficits are caused by anomalies in ONH and RNFL as glaucoma advances (Figure 2F). Due to the preservation of peripheral vision and Snellen visual acuity until an advanced level of RNFL loss, visual field abnormalities are frequently not noticed in the earliest glaucoma.(33)

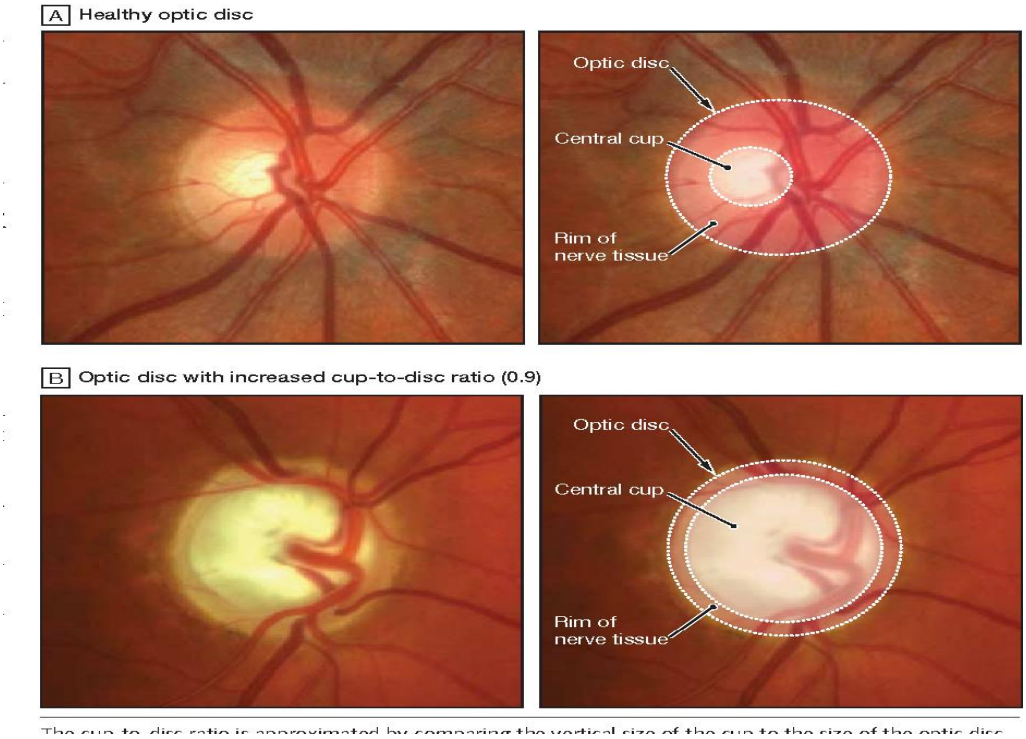

Figure 2. Healthy optic disc & Optic disc with increased cup to disc ratio(0.9)

There is a general correlation between OCT imaging and visual field examination, but there is no widely accepted method for comparing the two (36), and the diagnosis of glaucoma is ultimately left to the physician's judgement. Once glaucoma is diagnosed, it needs to be classified as mild, moderate, or severe. Since all types of glaucoma present with structural damage, most classification systems grade severity based on functional visual field abnormalities. The International Classification of Diseases, Tenth Revision, released a grading system recently (2015) and associates mild glaucoma with a general absence of visual field defects, moderate glaucoma with visual field abnormalities in 1 hemifield (but outside 5° of fixation), and severe glaucoma with abnormalities in both hemifields and visual field loss within 5° of fixation.(37)

IV. TREATMENT & MANAGEMENT

1. Medicinal Therapy :

Even in eyes with normal tension glaucoma, an initial IOP reduction of 20%–30% is a suitable target to delay disease development, according to guidelines from the American Academy of Ophthalmology Preferred Practice Pattern

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(2020).(38) Compared to previous systemic (oral CAIs) and topical (pilocarpine) therapies, these drugs are more efficacious and have better safety profiles.(39) Generally accepted pharmacotherapy guidelines state that the fewest drugs and least amount of side effects are necessary to achieve the intended IOP range.(23) Because of their tendency to induce glaucoma, ocular and systemic corticosteroids should be administered with caution in at-risk patients.

Table 1. US Food and Drug Administration–Approved Medications Used for the Treatment of Glaucoma

Class	Medication	Adverse effect	Contraindications
Prostaglandin analogs	•Bimatoprost •Latanoprost •Tafluprost •Travoprost •Unoprostone	•Eyelash growth •Iris darkening •Keratitis •Uveitis	•Hypersensitivity to ingredients
Cholinergic agonists	•Pilocarpine •Carbachol	•Myopia •Angle closure •Cataract •Retinal detachment	•Miosis •Bradycardia •Retinal detachment •Asthma •Inflammatory eye disease
Carbonic anhydrase inhibitors	First generation (systemic): •Acetazolamide •Methazolamide •Dichlorphenamide Second generation (topical): •Brinzolamide •Dorzolamide	First generation (systemic): •Renal calculi •Stevens-Johnson syndrome •Serum electrolyte imbalance Second generation (topical): •Corneal edema •Metallic taste	•Allergy to sulfa-containing medications (both) •Adrenal insufficiency, metabolic acidosis (systemic inhibitors only) •Sickle cell disease (topical inhibitors only)
Beta adrenergic antagonists	Nonselective: •Carteolol •Levobunolol •Metipranolol •Timolol β1-selective: •Betaxolol	•Congestive heart failure •Exercise intolerance •Hypotension •Bronchospasm •Bradycardia	•Cardiovascular disease •Asthma •Diabetes mellitus •Chronic obstructive pulmonary disease
Alpha adrenergic agonists	•Brimonidine	•Hypotension •Fatigue •Allergic conjunctivitis	•Monoamine oxidase inhibitor therapy
Rho kinase inhibitors	•Netarsudil	•Keratitis •Conjunctival haemorrhage •Corneal verticillata	•None

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Hyperosmotic agents	•Glycerol •Mannitol •Isosorbide	•Congestive heart failure •Renal failure •Nausea •Vomiting •Headache	•Cardiovascular disease •Renal failure

Prostaglandin analogs (PGAs) are the most commonly used medications for the treatment of OAG and ocular hypertension. Prostaglandin analogs compensate for decreased TM outflow by increasing outflow through the uveoscleral pathway,(40) where aqueous humour moves through the ciliary muscle into the supraciliary and suprachoroidal spaces.(41) Prostaglandin analogs are administered once daily, are well tolerated, and have limited systemic adverse effects.(23, 40) The main ocular adverse effects are eyelash growth, iris pigmentation, and uveitis. (38) Both beta blockers and calcium channel inhibitors (CAPIs) decrease intraocular pressure (IOP) by focusing on the ciliary body's production of aqueous humour. After topical application, CAIs enter the ciliary body epithelium and penetrate the cornea, where they decrease the production of bicarbonate ions.(42) The 2% and 1% CAIs are administered twice or three times a day, but their efficacy is generally lower than that of PGAs and beta blockers, which restricts their use as first-line therapy.(42)Systemic CAIs, such as methazolamide and acetazolamide, are very effective and can be used to treat ACG, but their use is constrained by a high rate of side effects—50% of patients develop intolerable within a month.

2. Laser Therapy :

A variety of laser procedures can be performed in glaucomatous eyes, with the procedure of choice depending on the etiology of the disease (Table 2). Laser procedures effectively lower the IOP and minimise the long-term costs that are associated with the long-term use of multiple pressure-lowering medications. When pharmacotherapy fails to achieve the target IOP and prevent vision loss, laser and surgical procedures are indicated.(25) Laser trabeculoplasty and ab-interno excimer trabeculectomy (Glautec AG) are both indicated for OAG that is refractory to pharmacotherapy. Laser trabeculoplasty—multiple spots of thermal laser applied directly to the TM—induces favourable structural changes that increase the aqueous humour outflow.(43) Argon laser trabeculoplasty, developed in 1979, uses a with a blue-green continuous-wave laser (488 and 514 nm) to disrupt the TM, whereas selective laser trabeculoplasty (SLT), developed in 1995, uses low energy, brief duration, large spots from a green, frequency-doubled laser to target melanin-containing cells and spare the TM tissue.(44) Selective laser trabeculoplasty has largely supplanted argon laser trabeculoplasty because of its favourable safety profile, comparable IOP-lowering efficacy, and ability for repeated treatment applications.(45) Patients with ACG require different laser procedures from those with OAG. A laser peripheral iridotomy creates a hole in the peripheral iris and is often performed to eliminate pupillary block,(46)whereas a laser peripheral iridoplasty uses low-power laser burns to relieve appositional angle closure (by shrinking the peripheral iris) in cases where laser peripheral iridotomy is ineffective.(47)When combined, both treatments have been shown to be safe and effective in lowering the IOP in eyes with acute primary ACG refractory to pharmacotherapy.(48)

Table 2. Laser Procedures for the Treatment of Glaucoma

Laser procedure	Preferred use	Pros	Cons
Laser trabeculoplasty •Argon laser trabeculoplasty •Selective laser trabeculoplasty •MicroPulse laser trabeculoplasty •Titanium-sapphire laser trabeculoplasty •Pattern scanning trabeculoplasty	•Open-angle glaucoma	•Performed in-office •Minimally invasive •Newer methods protect the TM tissue	•Decrease in efficacy over time •May cause transient IOP spikes and anterior uveitis

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Excimer laser trabeculostomy	•Open-angle glaucoma	•Minimally invasive •Minimises tissue fibrosis	•Performed in the operating room •Requires incision
Laser peripheral iridotomy	•Angle-closure glaucoma (pupillary block)	Performed in-office •Highly effective in the treatment of pupillary block–induced angle closure	•Not sufficient to relieve the angle closure caused by multiple mechanisms •May promote cataract progression
Laser peripheral iridoplasty	•Angle-closure glaucoma (plateau iris)	•Performed in-office •Can relieve appositional angle closure after an LPI •Effective in the treatment of angle closure caused by multiple mechanisms	•May cause atrophic iris scarring and loss of visual acuity •May develop Urrets-Zavalia syndrome
Cyclodestructive procedures •Endoscopic cyclophotocoagulation •Continuous-wave diode transscleral laser •MicroPulse diode transscleral laser therapy	•Glaucoma refractory to surgical treatment •Secondary glaucoma	•High IOP-reducing efficacy from mechanism targeting ciliary body	•Associated with a series of complications (hyphema, macular edema, mydriasis, decrease in visual acuity, keratitis, etc) •May require multiple treatments •Performed in the operating room

3. Surgical Treatment :

Operations are usually performed when medical and laser treatments have failed to achieve adequate IOP reduction. Surgical options consist of the traditional, bleb-based IOP-lowering operations (trabeculectomy and tube shunt implantation) and the newer, conjunctiva-sparing MIGSs (Table 3). Bleb-based operations can effectively lower IOP; however, they may develop bleb-related complications and may have high reoperation rates. As a result, the current role of traditional procedures in the era of evolving MIGSs is unclear. Surgeons' perspectives are changing(49) ; a recent practice preferences survey from the American Glaucoma Society (2017) found that trabeculectomy has fallen out of favour, with tube shunt implantation reported as the preferred incisional surgical treatment in 7 of 8 surgical centers.(50) When prospective MIGS trials are completed, the pendulum may swing in favour of MIGSs.(49)

Table 3. Surgical Procedures for the Treatment of Glaucoma

Procedure	Type	Pros	Cons
Trabeculectomy	•Incisional operation •Antimetabolite-associated	•Excellent IOP control •Can adjust the rate of fluid flow	•Bleb-related complications

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Ex-PRESS mini shunt operation	•Incisional operation	•Favourable safety profile to trabeculectomy •Minimal IOP fluctuations	•Bleb-related complications •High incidence of erosion, displacement, and hypotony
Valved drainage implants •Ahmed FP7 valve •Ahmed FP8 valve •Pars plana Ahmed	•Incisional operation	•Immediate IOP reduction •Valve reduces hypotony-associated complications during early postoperative period	•Bleb-related complications •Malfunctioning of the valve may result in hypotony
Nonvalved drainage implants •Molteno glaucoma drainage device •Baerveldt glaucoma implant •Ahmed ClearPath drainage device •PAUL glaucoma implant	•Incisional operation	•Greater surface area promotes sustained reduction of IOP	•Bleb-related complications •Delayed encapsulation and high incidence of hypotony in older Molteno and Baerveldt models
Trabecular bypass •iStent •iStent inject •iStent inject W •Hydrus Microstent	•MIGSs targeting the trabecular outflow pathway	•Low risk of hypotony •Favourable safety profile •Effective for mild and moderate glaucoma	•Does not achieve IOP reduction comparable to trabeculectomy •Not suitable for severe glaucoma •High risk of fibrosis
Canaloplasty •Ab-externo canaloplasty without tensioning suture •Ab-externo canaloplasty with tensioning suture •ABiC	•MIGSs targeting the trabecular outflow pathway	•Low complications rates •ABiC: safer and easier than ab-externo approach	•Generally not suitable for severe glaucoma
Ab-interno trabeculotomy; goniotomy •Trabectome •Goniotome •Gonioscopy assisted transluminal trabeculotomy •iAccess (Glaukos) •Kahook Dual Blade goniotomy •Kahook Dual Blade Glide	•MIGSs targeting the trabecular outflow pathway	•Goniotomy: clean excision of TM limits fibrosis and closure	

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Trabeculotomy/viscodilation •OMNI Surgical System	•MIGSs targeting the trabecular outflow pathway	•Targets all 3 points of outflow resistance (TM, Schlemm canal, collector channels)	
Goniotomy/viscodilation •STREAMLINE Surgical System	•MIGSs targeting the trabecular outflow pathway	•Ease of use •Combined TM excision and delivery of viscoelastic promotes high IOP reduction	•No long-term efficacy• Potential risk of fibrosis
Ab-interno subconjunctival implant •XEN45 gel stent •PRESERFLO microshunt	MIGSs targeting the subconjunctival space	•Greater IOP-lowering efficacy than angle-based MIGS •Suitable for severe glaucoma	•Bleb-related complications •Subconjunctival fibrosis

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