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AN UPDATE ON THE RISK FACTORS AND CURRENT MANAGEMENT STRATEGIES OF GLAUCOMA

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Abstract: -It is widely acknowledged that glaucoma is the leading cause of irreversible vision loss in the world. The loss of retinal ganglion cells is a characteristic of glaucoma. Excessive increase in Intraocular pressure (IOP) is the primary cause of glaucoma. An overall grasp of the disease is necessary so that the physicians may be able to refer high-risk patients for comprehensive ophthalmologic examinations and participate in patient care more actively if they have a better understanding of the disease's risk factors and treatment options, including pharmacotherapy as well as surgical management. The article presents the risks for glaucoma, along with current treatment options

Keywords: - Glaucoma, POAG, PACG, ganglion cells trabecular meshwork, Aqueous humor, Intraocular pressure

INTRODUCTION

There are more than 70 million cases of irreversible blindness caused by glaucoma around the world, 10% of whom are bilaterally blind [1][2]. An optic neuropathology that can cause cupping of the optic disc with accompanying loss of visual field, glaucoma is a multi-factorial condition marked by progressive structural changes within retinal ganglion cells (RGC) [3]. About half of people who have glaucoma are unaware of their condition until it is severe, leaving it untreated can lead to blindness [4]. There have also been reports of falls and inability to work due to glaucoma. Active lifestyle activities such as reading, walking, and driving can all be affected by the disease [5].

There are several factors that contribute to the development of glaucoma, including inflammation, injury, selective drugs, the aggregation of pigment and pseudo-exfoliating material. Glaucoma is classified by its appearance and obstruction of the drainage pathway. The prevalence of glaucoma is higher among African-Americans and Hispanics. Typically, glaucoma occurs in Europe and the United States as POAG, which involves normal, open anterior chambers and constrained aqueous humor outflows with an elevated IOP [6]. Primary angle-closure glaucoma, more common in Asia (about 70%), is blocked by the iris,

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whereas primary angle-closure glaucoma occurs when the drainage pathways are blocked by the iris.

TYPES:

Glaucoma can be categorized into two types: open angle and angle-closure glaucoma.

Primary open Angle Glaucoma (POAG) : -

The development of primary open-angle glaucoma (POAG), one of the leading causes of irreversible blindness in the world, is greatly affected by demographic and social factors. It is the most prevalent type of glaucoma. POAG is a persistent optic neuropathy that is characterized by the progressive loss of retinal ganglion cells (RGCs), resulting in structural impairment in the optic nerve head (ONH) and retinal nerve fibre layer (RNFL). These changes eventually manifest as visual field abnormalities [7]. POAG accounts for more than 50% of all glaucoma cases. In 2020, it is estimated there were 52.68 million cases of POAG among adults (40–80 years old). In 2040, it is predicted that there will be 79.76 million cases of POAG among adults [8]. POAG is more prevalent among black people than white people [9].The progressive degeneration of the optic nerve is usually accompanied by an elevated intraocular pressure (IOP). As of now, there are no neuroprotective therapies available and the only effective treatment is to lower IOP, which can slow disease progression at the early stages of the disease; however, over 50% of patients with glaucoma are not diagnosed until irreversible optic nerve damage has already taken place.[10]

Angle closure Glaucoma (ACG): -

There are approximately 26% of people with primary angle-closure glaucoma (PACG), but PACG accounts for nearly half of blindness related to glaucoma worldwide [1]. As a result of mechanical obstruction of the trabecular meshwork, PACG consists of elevated intraocular pressure (IOP). This obstruction may be caused by either an iris apposition to the meshwork or a synechial close angle [11].

Risk factors:

Age and Frailty: -

Age is one of the leading risk factors for Glaucoma. There is a greater risk of glaucoma with age. Due to this, glaucoma can be anticipated to lead to other age-associated conditions such as macular degeneration and vascular diseases [12]. Elevated eye pressure is one affront that the aged optic nerve may have difficulty handling. In a study of European-derived populations, PACG prevalence was 0.02% among those aged 40–49, but rose to 0.95% among those aged 70 and older [13]. Anterior chambers gradually decrease in depth and volume with time. The anterior chamber depth and volume decreased over 10 years by 0.21 mm and 19 µl respectively, contributing to angle narrowing and peripheral anterior synechiae (PAS) incidence among the elderly [14]. An individual with frailty is at a higher risk for accelerated physical and cognitive decline, disability, and death [15]. Frailty is recognized clinically as a state of enhanced vulnerability. It is determined whether an individual is frail based on his or her level of health deficits, such as high blood pressure, low blood pressure, diabetes, migraine, obstructive sleep apnea syndrome, cataracts, glaucoma, and medication use [16]. A number of other health problems are associated with frailty, as is the case with glaucoma. This may be an important cause for the concomitance of these health problems in

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some patients. Developing glaucoma is more likely to occur in frail individuals at a young age [17].

Gender: -

While glaucoma incidence and prevalence differ between males and females, it is still unclear whether gender plays a role in its development and progression [18]. There is a significant difference between the proportion of Asian women who have narrow angles compared to those of Asian men at every age, according to several surveys conducted among Asians. According to Rotterdam studies, Women with early menopause are at greater risk of developing primary open angle glaucoma (POAG) [19]. A US database, the 2002-2005 National Health Interview Survey (NHIS), revealed that women had a increased self-reported visual impairment for all causes (OR: 1.31; 95%CI, 1.19–1.45) and glaucoma had a higher prevalence (OR: 1.20; 95%CI, 0.99–1.45) [20]. By univariate analysis, there was a correlation between male gender and POAG in the Ocular Hypertension Treatment (OHT) study [21]. Based on a Bayesian meta-analysis, it has been found that black people had the highest POAG prevalence throughout life, but white people had the steepest POAG prevalence increase with age. Furthermore, Rudnicka et al. concluded that men were still at greater risk of POAG than women. These findings may only be applicable to the populations examined [22].

Genetic and Family history: -

OAG is significantly more anticipatedly to develop in individuals with a family history of the disease. A three-to-six-fold greater prevalence of glaucoma is found in people with close family members suffering from primary open angle glaucoma [23]. Those with a familial predisposition of glaucoma are almost one third more anticipatedly to experience advanced field loss than people without a family history (adjusted odds ratio, 0.29 [0.12, 0.74]) [24]. There is a relative risk of 2.1 times for glaucoma being associated with at least a possible family history [25]. It is important to note, however, that family history has varying significance depending on thethe level of familial or interpersonal proximity between a patient and an affected person of their family (1º,2º or 3º). It is estimated that approximately 50% of the patients with POAG have a $_+$ ve family history, and the risk of developing the disease is approximately nine times greater if their first-degree relatives suffer from the disease (parents, siblings or children) According to a meta-analysis comprising four studies, OAG is strongly associated with family history, with siblings showing the strongest association [26].

RACE: -

A review of 11 population-based studies found that POAG prevalence varied widely among people of the same race [27]. Research has shown that people of African, Hispanic, and Asian descent are more likely to contract several types of glaucoma compared to those of European ancestry. Even though OAG is most prevalent in black populations, white groups showed the sharpest increase in incidence with age [9]. Asian populations, particularly East Asians, are more likely to develop angle-closure glaucoma,

OSA: -

In several investigations, obstructive sleep apnea and glaucoma have been found to be associated. OSA patients had significant 24-hour changes in IOP, with the highest values at night. According to a study, seven patients at baseline and twelve on CPAP therapy both had

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an IOP fluctuation of 8% during the 24-hour period [28]. Those suffering from normal-tension glaucoma are particularly susceptible to sleep apnea [29]. In OSA, severe hypoxia and increased vascular resistance may contribute to ganglion cell loss [30]. Researchers have shown that eye conditions associated with OSA include glaucoma, papillary conjunctivitis, keratoconus, papilledema, optic neuropathy, and filamentary or infectious keratitis [31].

Eye Trauma: -

Traumatic glaucoma can be caused by both injuries to the iris and angle, as well as hyphema in the anterior segment of the eye. Those with blunt ocular trauma are most likely to suffer from iridodialysis, cyclodialysis, and angle recession [32]. Even if there is no hyphema, a blunt eye trauma can harm the drainage angle. As the IOP rises, bleeding and clot formation are reduced. Rebleeding from wounded veins is most likely to occur between 2-5 days after the injury as the clot lysis and retraction are under way [33]. Among the factors that may increase the risk of bleeding again are large hyphemas, youth, and race. The result of a late-onset glaucoma after a hyphema is angle recession accompanied by ghost cell glaucoma. During an injury, normal red blood cells in the vitreous become stiff, khaki-coloured ghost cells and migrate into the anterior chamber of the eye, this process gets completed in a couple of weeks. This cell type may cause an increase in IOP since the trabecular meshwork is blocked [34].Radial ciliary muscle tears caused by angle recession divide the circular and longitudinal fibres. According to one study, 7% of eyes with ≥180º angle recession results in advancement of glaucoma.

Medication induced Glaucoma: -

It is possible to develop glaucoma when certain medications are concomitantly used and instilled directly or systematically into the eye. If left untreated, glaucoma may cause debilitating vision loss.

Anti Glaucoma Drugs: -

Taking systemic and glaucoma medications together to treat co-existing illnesses is likely to cause drug interactions and negative effects. Glaucoma medications most commonly contain preservatives like benzalkonium chloride, a preservative at an average concentration of 0.01%. Various studies concluded that Benzalkonium chloride (BAC) contains numerous cytotoxic compounds that cause corneal damage and conjunctival lymphocytic infiltration both in animal and human models [35] [36]. Some studies have shown that Dorzolamide can cause stinging in the eyes and a metallic taste to some people [37]. In two studies, dry mouth was reported in 16.7 and 30.0% of patients, which is a common side effect associated with brimonidine 0.2% [38].

Antibiotics: -

Some studies have found that Sulphonamides induce ciliary body edema, resulting in mechanical angle closure, by inducing ciliary body rotation along with supraciliary effusion [39]. Case-control studies have shown that patients under age group 50 who use topiramate have a fivefold increased risk of angle closure compared to those who don't [40]. It has been reported that topical application of gentamicin in patients with narrow iridocorneal angles could cause ACAG. Macular infractions and widespread retinal nonperfusion can result from a subconjunctival injection of gentamycin and other aminoglycosides [41].

Steroids: -

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As a medication of choice for treating autoimmune and inflammatory disorders, steroids are the most widely prescribed medication in present era. It is worth emphasizing that steroids, in spite of all their advantages, can also have negative effects on the eyes, especially steroid-induced glaucoma and cataracts [42]. In many cases, intraocular pressure (IOP) rises within three to six weeks after topical steroid treatment. Once steroid eyedrops are stopped, IOP typically returns to normal within two weeks [43]. Corticosteroid ocular injections, however, may result in a sudden increase in IOP. The IOP increases 6 to 15 mmHg in about 30% of people treated with topical cortisone, as per various researches. However, only about 5% experience a significant response (IOP elevation over 16mmHg) [44].

Diabetes and hypertension: -

Researchers have discovered a separate link between type 2 DM and HT and glaucoma development. The blue mountains eye study found that 45.7% and 13% of OAG patients had HT and DM, respectively [45]. POAG risk can be increased by diabetes through vascular constriction caused by hyperglycaemia, which increases intraocular pressure and puts the optic nerve at greater risk [46]. In a study conducted by Sato and Roy, high glucose levels in the aqueous humour may make diabetes patients produce and accumulate more fibronectin in the trabecular meshwork [47]. According to studies, high blood pressure may affect the optic nerve's blood flow and damage it. Vascular abnormalities in the eye can be caused by hypertension, including arteriosclerosis (hardening of arteries) and constriction of blood vessels. POAG and arterial HT also have a favourable relationship in many studies [48].

Management protocols: -

Goal of management: -Vision preservation and slowing down the progression of glaucoma are the ultimate goals for glaucoma management, as well as individualised treatment approaches and a high quality of life for the patient [49]. It has been shown that the only effective method of treating glaucoma is to diminish the IOP, as it is generally accepted to be useful in delaying the progression of the disease [50]. The treatment of PACG differs greatly from the treatment of POAG due to the fundamentally different processes involved. There are many variables that affect treatment modality and intensity, including the glaucoma variant, expected life expectancy, visual prognosis, and ocular and systemic comorbidities. In all cases of glaucoma, IOP reduction remains the foremost treatment [51].

A variety of medications are available to treat glaucoma on a long-term basis, including prostaglandin analogues (PG), carbonic anhydrase inhibitors(CAI), adrenergic agonists, b-adrenergic antagonists, and cholinergic agonists.

Prostaglandin Analogues: _Prostaglandins are commonly considered as the initial preferred treatment option for glaucoma.In addition to prostaglandin analogues (PGAs), which are most effective at lowering IOP, other widely used classes such as b-blockers, carbonic anhydrase inhibitors, or a2-agonists are usually combined with them[52]. There are four prostaglandin analogues that are the most potent and most commonly prescribed: latanoprost, Travoprost, bimatoprost, and Travoprost. These drugs are commonly prescribed with a once daily medication regimen for improved patient compliance.FDA has approved latanoprost in 1996, followed by Travoprost and bimatoprost in 2001[53].

Prostaglandin analogues (PGAs) have demonstrated greater efficacy in reducing intraocular pressure (IOP) compared to β-blockers, while also maintaining a favourable systemic safety profile. The PGAs lower IOP by improving the drainage of aqueous humour into the

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uveoscleral outflow pathway, which is accountable for 10% of aqueous outflow. They also provide superior control over the diurnal IOP fluctuation that affects glaucoma patients frequently and contributes to long-term sight loss [54]. Latanoprost has been shown to be effective during nocturnal times by Lieu's study [55]. Other drugs such as alpha-agonists, β-blockers, and carbonic anhydrase can also be combined with prostaglandin analogues.The combined use of prostaglandin analogues (PGAs) and timolol can be beneficial in cases where either medication alone is insufficient in achieving the desired reduction in intraocular pressure (IOP). A separate meta-analysis reported a pooled decrease of -6.3 mmHg (95% CI -5.5, -7.1) following the addition of latanoprost once daily to 0.5% timolol bd, as well as a relative decrease of -28.3% (95% CI -23.7, -32.8) in the mean Intra-ocular pressure curve [56]. Most commonly, prostaglandins may occasionally result in certain side effects such as hypermia (redness of conjunctiva) and hypertrichosis, where eyelashes grow, thicken, or become pigmented. In darker-skinned individuals, periocular skin pigmentation may be more likely to occur [57].

β-blockers: -

Prior to the introduction of latanoprost in 1996, timolol was the drug of choice for reducing IOP [58].Timolol, Betaxolol, levobunolol, cartelol are included in the list of β-blockers. It was in 1978 that timolol was first drug to be authorised for therapeutic use of glaucoma,which aims to reduces IOP by impeding the synthesis of Aqueous humor, at an average of 20-30% [59][60]. Since b-blockers can cause heart failure, bronchoconstriction, bradyarrhythmia's, and systemic hypotension, the effectiveness of PGs is considered superior to that of b-blockers. Individuals with heart and lung problems should not take b-blockers because of their systemic absorption [61]. By inhibiting the binding and activity of endogenous catecholamines, epinephrine, and nor-epinephrine to ciliary body receptors, beta-blockers tend to decrease the production of aqueous humor [62]. Beta-blockers have been used to reduce IOP by 20–30% from baseline levels [63].

Carbonic Anhydrase Inhibitors(CAI): -

CAI are sulphonamides belongingto metalloenzyme family that decrease IOP by reducing production of Aqueous humor [62]. carbonic anhydrase isoenzyme II undergoes catalysis and results in the generation of HCO3- and H+ from CO2 and H2O, which is necessary for the formation of aqueous humour, so inhibition of this enzyme results in the decrease in the production of Aqueous humour. During peak and trough of IOP reductions after taking dorzolamide and brinzolamide, respectively, 22% and 17% were reported [64][65].Due to their security, widespread acceptability, and safety, they are frequently used as third- or fourth-line agents. The utilisation of these agents is not recommended in patients with sulphonamide allergies [66]. Carbonic anhydrase inhibitors can be used standalone or combination with other antiglaucoma medications (β-blockers and alpha agonists). Researchers have observed a reduction in IOP after prescribing a combination of latanoprost with dorzolamide in at least 30 patients [67]

Alpha -2 agonists: -

Apraclonidine hydrochloride 0.1% and Brimonidine Tartrate 1% are two common alpha-2 agonists used in the treatment of elevated IOP [68]. As a result of the stimulation of alpha-2 receptors in the eye, these medications constrict blood vessels within the ciliary body [69]. As a result, the production of aqueous humor is reduced, leading to reduction in IOP. Based on

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the result of a meta-analysis, which revealed that brimonidine reduces baseline IOP by approximately 17% when administered as an IOP-reducing agent [70]. By studying 15 patients prospectively, Liu and colleagues investigated how brimonidine affected IOP during sleep–wake cycles. An IOP measurement was conducted in sitting and supine positions in a sleep laboratory for patients who received brimonidine tartrate 0.1% tds in both eyes 8 hours apart [71].

According to the result of a meta-analysis, that all the formulations i.e, Brimonidine 0.2% in BAK, Brimonidine 0.1% in purite and Brimonidine 0.15 % in purite has been found to be safe and tolerable and have comparable efficacy [72].Clinical studies testing clonidine's neuroprotective properties show that topical clonidine can lower mean defect, short-term fluctuation, and also corrected pattern standard deviation[73]. Apraclonidine, on the other hand, may result in a substantial worsening of the mean defect (p 0.05), short-term fluctuation (p 0.05), and corrected pattern standard deviation (p 0.01). Apraclonidine may be associated with worse outcomes than clonidine, according to the findings of this study [74]. It is possible that brimonidine has the potential to inhibit retinal ganglion cell death without interacting with pressure-reducing mechanisms by directly interacting with alpha-2 adrenergic receptors in RGCs.

Brimonidine 0.2% is commonly associated with blepharitis, blepharoconjunctivitis, conjunctivitis, hyperaemia, hazy sight, xerostomia, and ocular allergy [75]. In roughly 12% of patients, these events may result in therapy cessation [76].

Laser therapy

Laser and surgical techniques are recommended when medication fails to reach the desired IOP and avoid vision loss. Laser techniques efficiently lower IOP while reducing long-term expenditures associated with the long-term usage of numerous pressure-lowering drugs [77]. A variety of laser operations can be executed in glaucomatous eyes, with the preferred procedure dependent on the aetiology of the disease.

In case of open angle glaucoma, the first laser procedure technique was Argon laser trabeculoplasty (ALT) introduced in 1979.The goal of procedure is to let fluids drain out from the eye and to prevail better visual field, lowering IOP which can cause optic nerve damage and loss of vision. This treatment involves the placement of several (50-100) non-penetrating laser burn spots uniformly around the trabecular meshwork at an angle of 180-360º [78]. Most researchers estimate a 5-year success rate of around 50% [79]. ALT is associated with complications such as persistent IOP elevation, iritis and PAS formation[80]. Laser heat causes coagulative degradation of the uveoscleral meshwork and damage to the adjacent structural collagen fibres [81].To address these issues, selective laser trabeculoplasty (SLT) was created in 1995, which uses a green (532nm), Q-switched, frequency doubled Nd: YAG laser to protect the trabecular meshwork from any damage [82][84]. This laser may specifically target pigmented cells in the trabecular meshwork because they have higher optical absorbance to the laser than surrounding cells, preventing collateral damage [83][84]. SLT had largely succeeded over ALT because of advantageous safety profile, maximum IOP reduction performance, less invasive and can be repeated multiple time. Other newly implemented laser trabeculoplasty procedures are Titanium-sapphire laser trabeculoplasty and pattern scanning trabeculoplasty, studies have found that both of the methods have

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comparable safety and efficacy profile [84]. Patients who have previously undergone failure in ALT either 180ºor 360º, have responded effectively to SLT treatment. Side-effects are less as compared to ALT and has been found to be effective in around 80% of patients, although it has a 50% failure rate at two years [85].

In case of ACG different laser techniques are introduced which include Laser peripheral iridotomy (LPI), which has been considered as first line treatment for ACG in mid 1970s [86].In addition to eliminating the pupillary block component, LPI may continue to widen the anterior-posterior ACA by balancing the pressures between the two chambers [87][88].An appositional angle closure resulting from a mechanism unrelated to pupillary block can be ameliorated with laser peripheral iridoplasty, which uses thermal energy to contract the peripheral iris away from the trabecular meshwork.To prevent pupillary block and reduce PAS formation, a modified LPI technique was developed that combined standard LPI with laser peripheral iridoplasty [89]. So, combination of both of these treatments have great safety profile and results in effective lowering of IOP in ACG patients who have poorly responded to pharmacotherapy [90].

SURGICAL MANAGEMEMNT

Surgical treatment is followed when there is a failure in reduction of IOP with previous medical and laser management. They include bleb based IOP lowering operations such as trabeculectomy and tube shunt, these are less invasive and have enhanced safety profile when compared to typical drainage surgeries [91].

Trabeculectomy – a gold standard procedure that is being performed since mid-1960s [85]. The procedure involves creating a fistula between the anterior chamber and sub-conjunctival space beneath a scleral flap. The aqueous humour exits from the eye and gets collected in a bleb shaped structure, this increased outflow of aqueous humour results in the decrease in the IOP [92]. According to a study of 1240 cases, different early and late complications associated with trabeculectomy were occurred which include hyphaema, shallow anterior chamber, hypotony, wound leak, supra choroidal haemorrhage [93]. A study based on the trabeculectomies in young population was conducted, In the study, 117 trabeculectomies were performed on 108 patients' eyes. Among the operations performed, 39 (33% were for primary non-developmental glaucomas, 50 (43%) were for secondary non-neovascular glaucomas, 17 (15%) were for developmental glaucomas, and II (9%) were performed for neovascular glaucoma. Among primary glaucoma cases, the highest success rate was achieved, while neovascular glaucoma cases showed the lowest success rate [94]. Late complications include vision loss, cataract, encapsulated bleb, staphyloma. Despite these hazards, trabeculectomy is generally considered safe and predictable [95].

Tube shunt: -This drainage device is considered as an alternative to trabeculectomy. Tube shunt are typically recommended when other treatments, such as medications or laser therapy, have failed to adequately control the disease [96]. They have a great advantage over trabeculectomy by preventing conjunctival scarring and formation of permanent bleb. In a tube shunt procedure, a non-decomposable silicone or polypropylene tube is placed in the anterior chamber of the eye where it allows the aqueous humor to bypass the natural drainage pathways and flow out through an attached plate or reservoir. Ahmed Glaucoma Valve (with

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flow restriction) and Baerveldt Glaucoma Implant (without flow restriction) are the two most commonly used shunts. GDDs may be valved (promotes unidirectional flow) or non-valved (passive acting) and are designed in the same manner as early Molteno implants93 [97]. Ahmed Baerveldt Comparison and Ahmed Versus Baerveldt studies compared the safety and efficacy of the valveless Baerveldt 350-mm2 GDD (Johnson & Johnson) with the valved Ahmed-FP7 GDD (New World Medical Inc). Butboth devices were efficient at lowering IOP and the need for IOP-lowering drugs, however the valveless Baerveldt- 350-mm2 GDD had a better IOP decline, medication burden reduction, and safety profile after 5 years [98]. Findings of a retrospective comparative case study suggested that ,3 out of 127 patients implanted with Ahmed glaucoma valves had reported corneal decompensation [99].

Minimally invasive glaucoma surgeries (MIGs): -

Minimally invasive glaucoma surgeries (MIGS) refers to a group of procedures with an ab-interno approach that are designed for their improved efficacy and safety profile, minimal trauma, or with conjunctiva -sparing as well as increased IOP reduction by using less invasive techniques. MIGs are performed as standalone or in concurrence with cataract surgery [100]. MIGs are categorised into three types Trabecular micro bypass stents-these device work by ameliorating trabecular outflow through Schlemm's canal e.g.iStent, Hydrus [101]. Second one is Suprachoroidal shunts enhance the uveoscleral outflow through a connection between the anterior chamber (e.g. CyPass®, iStent® Supra) and the suprachoroidal space and the third one is sub conjunctival MIGs, Using subconjunctival devices, the aqueous humor escapes into the subconjunctival space in an alternative route [102]

CONCLUSION: -

Glaucoma is a complex condition and can lead to irreversible loss of eyesight. The fundamental objective for glaucoma detection is the assessment of risk factors, which will aid in establishing whether patients are either at high risk or low risk of developing glaucoma. Age, family history, ancestral heritage, increased intraocular pressure, and specific medical disorders have all been recognised as important risk factors for glaucoma. Effective glaucoma therapy is critical for preserving eyesight and keeping a high quality of life for those suffering from the condition. Glaucoma management strategies are tailored based on the specific underlying causes of the condition, and there is a increasing tendency towards considering surgical interventions such as trabeculectomy, tube shunt and novel strategies of MIGs, at an earlier stage for both open-angle and angle-closure glaucoma.

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