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A Review: Drug Induced Angle Closure Glaucoma

[1]Krunal Mali, [2]Shaikh Habiburrahman, [3]Divya Mahajan, [4]Mohammed Sufiyan, [5]Zahid Anwer, [6]Jaydeep Wanare

[1,3,6]U.G. Student, Department of Pharmacy, Dr. Uttamrao Mahajan College of B. Pharmacy Chalisgaon, India
[2]Assistant Professor, Department of Pharmaceutical Chemistry, Dr. Uttamrao Mahajan College of B. Pharmacy Chalisgaon, India

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ABSTRACT:A series of eye conditions known as glaucoma can result in blindness (loss of vision). High intraocular pressure greater than 21 mmHg causes injury to the optic nerve, the nerve that connects the eye to the brain.Primary open-angle glaucoma is thought to affect 57.5 million people worldwide. People over 60, family members of glaucoma patients, diabetics, hypertensives, people with high myopia, people who use steroids, people with central corneal thickness less than 5 mm, and people who have had eye injury are also at higher risk. It is anticipated that 76 million people would have glaucoma by 2020, and that figure will rise to 111.8 million by 2040.Glaucoma comes in a variety eye forms, and they are often categorized as either primary, secondary, open-angle, or angle-closure glaucoma. Numerous ocular or systemic disorders can lead to secondary types of glaucoma. The purpose of this article is to raise awareness of this ophthalmic illness among doctors, especially those outside of the area of ophthalmology who frequently prescribe these drugs or treat these patients before referring them to ophthalmologists.

KEYWORDS: Angle closure, Glaucoma, Drug-induced.

I. INTRODUCTION

One of the main factors contributing to lifelong, irreversible blindness worldwide is glaucoma. There are many clinical manifestations and a complicated etiology for glaucoma.(4) The majority of research agree that age and intraocular pressure (IOP) are the two main risk factors for this condition. Epidemiological research has demonstrated the significance of noting the kind of glaucoma in various groups. Angle-closure glaucoma has a higher incidence of blindness even though open-angle glaucoma has been shown to be more common.(5) Primary angle-closure glaucoma (PACG) and secondary closed-angle glaucoma are two different types of angle-closure glaucoma. Primary angle closure glaucoma (PACG) is further divided into acute and chronic forms. Acute indicates that the anterior chamber angle has closed along with a sudden rise in intraocular pressure.(1) Closure of the anterior chamber angle accompanied by a rise in IOP resulting from known causes is known as secondary closed-angle glaucoma.(1) Despite being less prevalent than open-angle glaucoma, angleclosure glaucoma has a higher incidence of blindness.(1)Glaucoma develops in two stages: early-onset forms with Mendelian heredity (onset before age 40) and adult-onset forms with complicated features (onset beyond age 40).(1) Early-onset glaucoma is caused by gene mutations that are uncommon and have several biological implications, whereas adult-onset glaucoma is more prevalent and has fewer biological effects.(1)

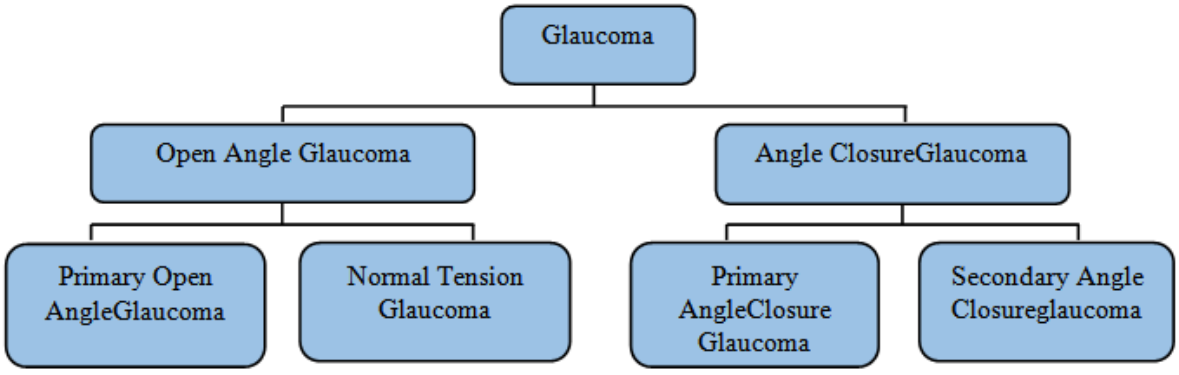

Figure No 1 : Types of Glaucoma

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Should I Be Referred To Hospital?

Damage, eye pain, or those who are at risk for angle closure glaucoma or eye pain should be referred to secondary eye care services.(6) Due to a Regardless of intraocular pressure, patients with signs of optic nerve higher risk of developing glaucoma, some individuals with ocular hypertension and a mildly dilated pupil should be referred.(6)Due to prompt diagnosis, almost one-third of patients with suspected glaucoma who are referred by optometrists are discharged after the first appointment.(6)

Risk factors for acute angle closure

The following are general risk factors for acute angle closure glaucoma: age, gender, a history of the disease in the family, hazy vision, hyperopia, and eye pain. A thick crystalline lens and narrow iridocorneal angles are two physical traits that can further raise the risk.(2) In investigations conducted in clinics, systemic hypertension, abrupt hypotension, and vasospasm have all been identified as significant risk factors for glaucoma. There are correlations between low diastolic pressure, lower ocular perfusion pressure (OPP), and increased prevalence and/or incidence of glaucoma, according to a number of studies.(7)

Pathophysiologic mechanisms of angle closure glaucoma

The most frequent method of angle closure, pupil dilation causing pupillary block, causes a progressive rise in intraocular pressure. The aqueous flow from the posterior chamber to the anterior chamber is blocked when the pupil comes into contact with the lens. This leads to an increase in intraocular pressure (IOP) in the posterior chamber, which causes the peripheral iris to bow anteriorly and adhere to the trabecular meshwork. As a result, the IOP increases and the aqueous cannot drain.

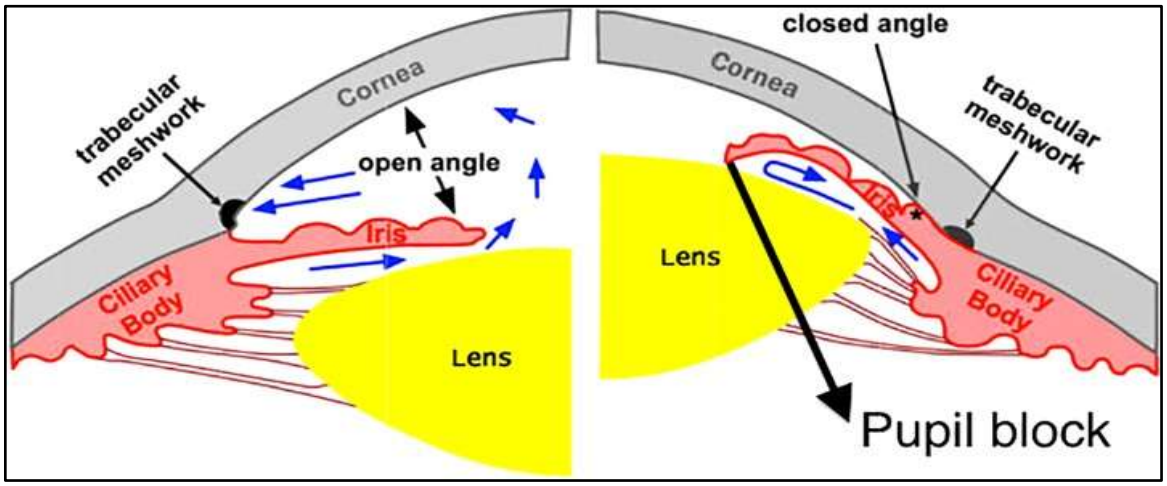

Figure No 2 : Mechanism of acute angle closure by pupil block

Other causes of angle closure besides pupillary block include plateau iris, lens-related, and posterior segment disorders such aqueous misdirection, choroidal effusion, or tumors in the posterior segment behind the lens that cause the lens-iris diaphragm to move forward.

Table 1: Drugs that have the potential to precipitate acute angle closure glaucoma.

Class of drug	Example of drug	Route of administration	Mechanism of angle closure
Adrenergic agonists	Phenylephrine	Eye drops	Pupillary block
	Ephedrine	Intravenous	Pupillary block
Non-catecholamine adrenergic agonists	Naphazoline	Intranasal	Pupillary block
	Salbutamol	Inhalation	Pupillary block

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Anticholinergics	Tropicamide	Eye drops	Pupillary block
	Ipratropium bromide	Inhalation	Pupillary block
	Promethazine (antihistamine)	Oral	Pupillary block
	Botulinum toxin	Periocular (local)	Pupillary block
Cholinergic	Pilocarpine	Eye drops	Forward displacement of lens-iris diaphragm
Medications with anticholinergic side effects	Imipramine (tricyclic antidepressant)	Oral	Pupillary block
	Fluoxetine (serotonin reuptake inhibitor)	Oral	Pupillary block
Sulfa-based agents	Topiramate	Oral	Ciliochoroidal effusion leading to forward displacement of lens-iris diaphragm
	Acetazolamide	Oral	Ciliochoroidal effusion leading to forward displacement of lens-iris diaphragm
Anticoagulants	Heparin	Subcutaneous	Forward displacement of lens-iris diaphragm

Adrenergic agonists:

In people with intrinsically small iridocorneal angles, alpha-adrenergic agonists can cause pupillary block from dilated pupils, which in turn can trigger acute angle closure glaucoma. Iatrogenicity is best demonstrated by the frequent use of phenylephrine (an alpha-adrenergic agonist) eyed drops by opticians and ophthalmologists to dilate the pupil for fundus examination. Acute angle closure glaucoma has been estimated to occur 0.03 percent of the time after diagnostic pupil dilation.(8) In the context of general anesthesia, systemic ephedrine is frequently used to treat hypotension. Despite being a diagnostic problem (because doctors are more likely to have intraoperative corneal abrasions on the top of their differentials for an uncomfortable postoperative eye)(9). A case of postoperative acute angle closure glaucoma caused by intraoperative ephedrine usage after non-ophthalmic surgery was actually recorded. This should serve as a warning because stress might exacerbate the effects of intraoperative sympathomimetic medications, both positive and negative, increasing the risk for acute angle closure glaucoma. A patient who has recently undergone anesthesia is more likely to mistake or ignore signs of acute angle closure glaucoma. Clinicians should therefore have a low threshold for referring any patient complaining of vision loss or having red eyes after surgery for an ophthalmic assessment.

Non-catecholamine adrenergic agonists:

Epistaxis has been treated with nasal ephedrine and naphazoline, but as a result, cases of acute angle closure glaucoma have been reported. It was believed that reflux through the ipsilateral nasolacrimal duct was what caused angle closure. In one case, it was even hypothesized that a nasal decongestant absorbed through the nasal mucosa for a week caused a patient's bilateral acute angle closure glaucoma.(10,11) Adrenergic medications, including salbutamol, are frequently present in nebulizers and inhalers used to treat asthma, as well as in over-the-counter flu medications and rectal suppositories. Salbutamol activates beta-2-adrenergic receptors in the ciliary body to encourage aqueous humor secretion. Salbutamol is a beta-2-adrenergic receptor agonist. Angle closure is made worse by pupil dilation brought on by ipratropium's parasympathetic inhibitory action, which is frequently paired with salbutamol in inhalers or nebulizers. Salbutamol does not, however, cause angle closure when used alone, it should be mentioned. As a result, care should be used when prescribing or administering these

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medications simultaneously, especially because many of them are available over-the-counter.

Anticholinergic agents:

Tropicamide drops have been linked to acute angle closure glaucoma and are frequently used to perform ocular fundus examinations. Angle closure has also been observed to occur when longer duration anticholinergic eye drops, such as atropine and cyclopentolate, are used to relax the ciliary muscle and dilate the pupil.(12)

An antimuscarinic medication known as nebulized ipratropium bromide is frequently given along with salbutamol to patients who have acute asthma or a flare-up of chronic obstructive pulmonary disease. Acute angle closure glaucoma has been linked to it in several case reports.(13,14) According to the proposed mechanism of angle closure, ipratropium bromide diffuses into the cornea after exiting the oxygen mask, causing pupil dilation and pupillary block. Therefore, in order to lower the danger of contact between the cornea and medication, clinicians should make sure that the oxygen masks are properly fitted and positioned. Bone and Kalra.(18) examined how nebulized bronchodilator medication affected glaucoma patients' intraocular pressures. They came to the conclusion that while using salbutamol and nebulized ipratropium bromide individually in individuals with narrow-angle glaucoma is generally safe, doing so increases the chance of causing an acute angle closure glaucoma attack in those who are vulnerable.

Due to their anticholinergic characteristics, antihistamines (H1 and H2 receptor blockers) can also cause acute angle closure glaucoma. According to reports, promethazine can enlarge the lens, which can lead to pupillary obstruction. It has also been demonstrated that the antacids ranitidine and cimetidine, which are used to treat gastroesophageal reflux disease and peptic ulcers, raise intraocular pressure in glaucoma patients.(21,15)

Botulinum toxin:

Over the past few decades, the usage of botulinum toxin has been increasingly widespread in the cosmetics business. There is little information available on the precise pathophysiology of acute angle closure glaucoma brought on by periorbital injections of botulinum toxin. Because of its anticholinergic impact on the sympathetic ganglia, preganglionic nerve terminals, and postganglionic nerve terminals of the parasympathetic nervous system, botulinum toxin is hypothesized to cause acute angle closure glaucoma. As a result, the pupillary sphincter is inhibited, causing pupil dilation and pupillary block. Before beginning therapy, patients receiving injections of the botulinum toxin for aesthetic purposes should undergo ocular tests, including gonioscopy, to assess their risk profile for angle closure and establish whether laser iridotomy might be beneficial.(19) To refer every patient receiving botulinum injections to an ophthalmologist for gonioscopy, however, is not always possible for doctors. Utilizing the oblique penlight illumination test is a simpler way to gauge the depth of the anterior chamber. To shine the light nasally, the penlight is oriented temporally and perpendicular to the iris plane. An open anterior chamber is indicated by an iris that is completely lighted. The iris of people who have shallow anterior chambers is convex and bows forward over the lens. A shallow anterior chamber is therefore indicated by the existence of a nasal shadow.(16) An instance of acute angle closure glaucoma caused by periorbital botulinum toxin injections used to treat blepharospasm therapy.. The toxin's diffusion from the injection site to the ciliary ganglion, which caused pupil dilation, was the proposed mechanism of angle closure.

Cholinergic agents:

The parasympathetic nerve system is agonistby pilocarpine, which causes pupil constriction (miosis). Due to its miotic action, pilocarpine eye drops are used to treat angle closure glaucoma. Additionally, it aids in lowering intraocular pressure by increasing aqueous outflow. However, pilocarpine can cause acute angle closure glaucoma on its own due to the lens-iris diaphragm’s forward displacement.(20)

Antidepressants:

Acute angle closure glaucoma has been linked to tricyclic antidepressants like clomipramine, imipramine, and amitriptyline as well as selective serotonin reuptake inhibitors (SSRI) like venlafaxine, citalopram, escitalopram, fluoxetine, and paroxetine. Due to the strong anticholinergic and serotonergic adversereactions of these antidepressants, pupillary block brought on by dilation of the pupils is the underlying mechanism.(17,22) However, it is unknown how serotonin affects the physiology of the human eyes. Before prescribing SSRIs, doctors should think about referring patients with a higher risk of developing acute angle closure glaucoma for an eye exam. Monoamine oxidase inhibitor-containing

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antidepressants such tranylcypromine sulphate or phenelzine sulphate have negligible anticholinergic effects. However, when combined with other anticholinergic medications, they have been shown to accelerate the development of acute angle closure glaucoma.(25) Trifluoperazine, perphenazine, and fluphenazine are antipsychotics that have been documented to cause acute angle closure glaucoma as well, but they have fewer anticholinergic effects on ocular smooth muscle than tricyclic antidepressants do.(21,15)

Sulfa-based agents:

Topiramate, acetazolamide, and hydrochlorothiazide are only a few of the few medications that can cause acute angle closure glaucoma, also known as glaucoma with "non-pupillary block." Sulfa-based medications have been linked to shallowing of the frontal chamber, choroidal effusion, enhance intraocular pressure, lens swelling, and retinal oedema, according to Lee et al.(23)The typical symptom is blurred vision as a result of the lens-iris diaphragm moving forward (myopia).It is unclear exactly how sulfa-based substances produce acute angle closure glaucoma. The iris-lens diaphragm can move forward due to ciliary body edema or anterior choroidal effusion, which can both impede aqueous flow and lead to acute angle closure glaucoma. Both of these mechanisms have the potential to diminish the ciliary body ring's diameter, which can result in zonular laxity, which promotes lens thickening and further reduces anterior chamber depth.(24)

Topiramate is frequently used for weight loss and migraine prevention. Several documents have actually recorded cases of mutual acute angle closure glaucoma caused by topiramate in patients with narrow angles, despite the fact that some literature refers to topiramate-induced reactions as type B reactions, or idiosyncratic reactions.(27,29)

Choroidal effusion is the primary mechanism of angle closure induced by topiramate. According to ultrasound scans, topiramate-induced ciliary oedema causes the zonules to relax, which in turn causes the lens thickness to grow. The ciliary body rotates anterolaterally as a result, the lens-iris diaphragm moves forward, and the anterior chamber shallows as a result.(30) More than 100 previously known cases of topiramate-induced angle closure described bilateral non-pupillary block acute angle closure glaucoma following the first doses of the medication (only three cases of unilateral involvement). The episodes normally began between days 1 and 49 and stopped after the causative agent was stopped.(21)It is crucial to stress that topiramate primarily causes simultaneous bilateral angle closure glaucoma attacks in both eyes. Bilaterality is a blatant indication that a drug-induced acute attack has occurred.(26) described a case of acute angle closure glaucoma caused by sulfamethoxazole-trimethoprim that resulted in permanent bilateral blindness despite quitting the medication. Acute angle closure glaucoma caused by sulfa drugs is managed by discontinuing the medicine and reducing intraocular pressure with cycloplegics and topical or systemic steroids. Topical miotics ought to be avoided since they might result in pupillary obstruction. Additionally, laser iridotomy is useless in these circumstances.(23)

In order to lower intraocular and intracranial pressure, acetazolamide is frequently utilized in ophthalmic and neurologic treatment. Interestingly, oral acetazolamide is normally given for a day after cataract surgery to minimize unwarranted rises in intraocular pressure. Acute mutual angle-closure glaucoma and choroidal effusion linked to acetazolamide treatment after cataract surgery have been described in a few case reports.(31,33) Therefore, while treating patients who experience acute angle-closure glaucoma following cataract surgery, doctors should take into account this significant acetazolamide side effect.

Anticoagulants:

Acute angle-closure glaucoma can be brought on by uncommon side effects of anticoagulant medication, such as severe vitreous, choroidal, or subretinal hemorrhage. The mechanism for angle closure is the abrupt forward displacement of the lens-iris diaphragm caused by the detached retina or choroid. Risk elements include excessive anticoagulation, nonophthalmic conditions, and exudative age-related macular degeneration.(32,34) Peripheral iridotomy is ineffective in these situations because non-pupillary block is the mechanism of angle closure. To stop additional bleeding, especially in the opposite eye, it is advised that the doctor think about stopping the anticoagulant.(34)

II. CONCLUSION:

Due to the non-specific nature of the presenting symptoms of individuals with acute angle-closure glaucoma, they might be deceiving. These symptoms include headache, blurred vision, nausea, and vomiting. A mid-dilated pupil and abrupt beginning of visual impairment, however,

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point to acute angle-closure glaucoma. When patients with these symptoms arise, hyperopic glasses (for longsightedness) are a surefire marker of acute angle-closure glaucoma. Clinical professionals who provide these medications are unable to refer every patient for eye examinations. Furthermore, because the majority of patients at risk for acute angle-closure glaucoma are asymptomatic and unaware of the elevated risk brought on by innately narrow iridocorneal angles, it is challenging to identify all of these people.

To stop attacks of acute angle-closure glaucoma, patients with angle closure glaucoma are often treated with laser iridotomy, filtering surgery, or cataract removal. It should be safe for these people who have had laser iridotomy to take drugs that can only cause acute angle-closure glaucoma by pupillary block. This educational review demonstrates how crucial it is for clinicians to be aware of drugs that may cause acute angle-closure glaucoma. A complete history should be taken of patients who have acute angle-closure glaucoma, and a full list of both prescribed and over-the-counter medications should be obtained, including discontinued ones, can use to identify the causal substances. Once the diagnosis is suspected, immediate medical attention is needed since proper and fast treatment may be able to save your sight.

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