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REVIEW

Non-steroidal drug-induced glaucoma

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Abstract

Numerous systemically used drugs are involved in drug-induced glaucoma. Most reported cases of non-steroidal drug-induced glaucoma are closed-angle glaucoma (CAG). Indeed, many routinely used drugs that have sympathomimetic or parasympatholytic properties can cause pupillary block CAG in individuals with narrow iridocorneal angle. The resulting acute glaucoma occurs much more commonly unilaterally and only rarely bilaterally. CAG secondary to sulfa drugs is a bilateral non-pupillary block type and is due to forward movement of iris–lens diaphragm, which occurs in individuals with narrow or open iridocorneal angle. A few agents, including antineoplastics, may induce open-angle glaucoma. In conclusion, the majority of cases with glaucoma secondary to non-steroidal medications are of the pupillary block closed-angle type and preventable if the at-risk patients are recognized and treated prophylactically.
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Introduction

Glaucoma is a form of optic neuropathy with specific visual field loss. It is usually associated with raised intraocular pressure (IOP) and may be divided into primary and secondary forms.[1,2] All types of glaucoma are categorized as open angle or closed angle based on the status of iridocorneal angle. If the cause of glaucoma can be identified, then the closed-angle glaucoma (CAG) or open-angle glaucoma is secondary.

Glaucoma can occur as a consequence of some medications administered directly to the eye or systemically within the body. Untreated drug-induced glaucoma can lead to loss of vision. The majority of drugs listed as contraindicated in glaucoma are concerned with CAG. These medications may incite an attack in those individuals with narrow iridocorneal angle.[3] At least one-third of acute closed-angle glaucoma (ACAG) cases are related to an over-the-counter or prescription drug.[1] Prevalence of narrow angles in whites from the Framingham study was 3.8%. Narrow angles are more common in the Asian population. A study of a Vietnamese population estimated a prevalence of occludable angles at 8.5%.[4] The reported prevalence of elevated IOP months to years after controlling ACAG with laser iridotomy ranges from 24 to 72%.[5,6] Additionally, a significant decrease in retinal nerve fiber layer thickness and an increase in the cup/disc ratio occurs after ACAG, which mandates lifelong care even when iridotomy has apparently alleviated the glaucoma attack.[7–10] Although drug-induced ACAG is relatively uncommon, it is a serious adverse reaction which, if not recognized in a timely manner, may result in severe morbidity and even permanent visual loss.

Medications have a direct or secondary effect, either to stimulate sympathetic or inhibit parasympathetic activation causing pupillary dilation, which can precipitate ACAG in predisposed patients (pupillary block CAG). In eyes predisposed to angle closure by their characteristic biometric features (short axial length and small anterior chamber), choroidal volume expansion also is considered as a contributing factor in inducing angle closure by slight forward movement of the lens and then narrowing and increasing resistance in the iris–lens channel.[11] The other mechanism for CAG is thickening and forward movement of lens, ciliary body rotation, and choroidal effusion, which occur in patients with open or narrow iridocorneal angle (non-pupillary block CAG). This process seems to be an idiosyncratic reaction to certain systemic medications.

Most attacks of drug-induced pupillary block CAG occur in individuals that are unaware that they have narrow iridocorneal angles.

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Physicians using these drugs cannot practically send each and every patient to an ophthalmologist for gonioscopy, but they should be informed that patients wearing thick hyperopic glasses are at risk. They can perform the lateral penlight test to estimate anterior chamber depth and send the suspicious patients to an ophthalmologist for gonioscopy.[12] The at-risk patients who are going to use the CAG inciting drugs should be offered a prophylactic iridotomy to prevent acute attacks. These patients need to be followed for any possible synechia formation and chronic CAG as long as they use the drugs.[13]

Numerous systemic medications have been implicated in the cause of drug-induced glaucoma and the most common medications that induce glaucoma are corticosteroids. In our previous review,[14] all drugs causing glaucoma, including steroids, have been discussed; the present review is more comprehensive with recent references and discusses the treatments in detail. This review will discuss the non-steroidal medications that are involved with glaucoma induction.

Ocular agents

Antiglaucoma drugs

Cholinergic agents, topical anticholinesterase, pilocarpine, and intraocular acetylcholine and carbachol are commonly used cholinergic agents in ophthalmic practice. These agents can, however, induce ACAG due to anterior movement of the iris–lens diaphragm.[3] In addition to narrow angle, the eyes with zonular weakness or exfoliation syndrome seem to be particularly prone to developing miotic-induced CAG.[15] Surprisingly, pilocarpine may also precipitate ACAG by inducing mydriasis. This is due to an impurity of the stereoisomer of pilocarpine, jaborine, an atropine-like drug that even today, with improved manufacturing, occasionally is reported.[16] Additionally, these agents can induce non-pupillary block CAG with inducing aqueous misdirection (malignant glaucoma).[17]

Sympathomimetics, especially those with α-1agonistic activity, cause mydriasis and can precipitate ACAG in predisposed individuals. Topical phenylephrine and its prodrugs dipivefrin and apraclonidine have been documented to induce ACAG.[3,18–20]

Latanoprost has been associated with ACAG in two cases.[21] Sakai *et al*[22] had reported about ciliochoroidal effusion induced by latanoprost in a patient with Sturge–Weber syndrome. Enhanced uveoscleral outflow is the mechanism of latanoprost IOP reduction. Yalvac *et al*[21] speculated that the increased flow through the ciliary muscles caused swelling of the ciliary body and pushed the iris–lens diaphragm anteriorly, initiating an ACAG.

Acetazolamide, an oral carbonic anhydrase inhibitor, can induce non-pupillary block CAG similar to other sulfa agents.[23]

Mydriatics

Topical anticholinergic/cycloplegics agents used for pupillary dilation for fundus examination should be used with caution in the susceptible patients. It has been shown that the iris volume increases after pupil dilation in narrow-angle eyes even in those with open iridotomies, which augments the possibility of CAG.[24] Pandit and Taylor[25] in a systematic review stated that the risk of inducing ACAG following mydriasis with tropicamide alone is close to zero and no case being identified. The risk with long-acting or combined agents was between 1 in 3380 and 1 in 20 000. However, Mapstone[26] reported sever IOP elevation, which necessitated antihypertensive treatment in 19 out of 58 patients with narrow iridocorneal angle who underwent tropicamide pupil dilation.

Topical cycloplegics have been shown to cause significant IOP elevation in 2% of the apparently normal population, increasing to 23% of patients with known primary open-angle glaucoma.[27,28] It is recommended that the IOP be rechecked after dilation in glaucoma patients with significantly damaged optic nerve heads. The rise in IOP may be related to decreased aqueous outflow, resulting from decreased pull on the trabecular meshwork due to ciliary muscle paralysis.[29] Valle[30] reported an increase in aqueous inflow in patients who experienced a rise in IOP following pupillary dilation and also suggested a decrease in aqueous outflow in the same patients.

Ointments

Ointments are commonly used after ophthalmic surgery. Intraocular penetration may cause inflammation and open-angle glaucoma. One of the reported complications of intraocular ointments is toxic anterior segment shock syndrome that may lead to glaucoma due to trabeculitis and fibrin membranes.[31] In 1972, Sugar and Airala[32] proposed that ointments should be avoided on the first day or so postoperatively.

Botulinum toxin

Botulinum neurotoxins act primarily at peripheral cholinergic synapses, including the skeletal muscle neuromuscular junction, causing transient muscle paresis by inhibiting the release of acetylcholine. However, botulinum toxins could cause pupillary dilation when injected periocularly. It diffuses toward the ciliary

ganglion and blocks its activity or acts at the pupillary sphincter muscle of the iris. Corridan *et al*[33] reported a case of ACAG after receiving a series of botulinum toxin injections for blepharospasm.

Silicone oil

One of the reported complications of silicone oil is secondary open-angle glaucoma that can occur at any time in the postoperative period.[34] The IOP elevation may range from mild and transient to severe and sustained. The early IOP spikes may be due to pupillary block, mechanical-angle obstruction by oil, or inflammation and the late-onset spikes are proposed to be due to synechial-angle closure, rubeosis iridis, oil droplet emulsification, and migration of silicone oil into the anterior chamber and clogging the outflow. Presence of silicone oil in the anterior chamber may induce a chronic inflammation and trabecular meshwork fibrosis. Treatments of elevated IOP include silicone oil removal, medical therapy, and laser or incisional surgeries.[35–37]

Viscoelastic materials

These agents have been associated with an elevated IOP and open angle in the early postoperative period.[38] The IOP rise seems to be due to obstruction of the trabecular meshwork outflow channels and is usually seen within the first postoperative day.[39] Kocak-Altintas *et al*[40] also reported a higher incidence of postoperative IOP increases associated with the use of viscoelastic agents with higher viscosity. If these agents are carefully washed out of the eye at the end of surgery, the risk of an IOP spike will be significantly reduced.[38,39]

Antibiotics

Sulfa drugs have been reported to induce ACAG without pupillary block. Mechanisms in this type of angle closure include lenticular swelling, retinal edema, choroidal effusion, and secondary shallowing of the anterior chamber associated with elevated IOP.[41–43] The lens thickening has been confirmed with A-scan ultrasound in acute attacks.[44] The increase in the anteroposterior lens thickness may be due to marked zonular relaxation secondary to swelling of ciliary body. Moreover, the mechanism may involve anterior rotation of the ciliary body, choroidal expansion secondary to accumulation of serous fluid in the extravascular choroidal space.[43] This type of CAG has been reported with the following drugs: topiramate, hydrochlorthiazide, acetazolamide, quinine, and tetracycline.[23,45–47] Table 1 shows the list of medications containing sulfa component in their formulations.

Table 1 List of sulfa drugs

Antibiotics	*Rheumatologic drugs*
Trimethoprim-sulfamethoxazole	Sulfasalazine
Sulfadizine	Probenecid
Sulfisoxazole	Celecoxib
Dapsone	Valdecoxib
Topical sulfa antibiotics	
Sulfonylurea	*Diuretics*
Glyburide	Acetazolamide
Chlorpropamide	Furosemide
Gliclazide	Bumetanide
Glimepiride	Hydrochlorothiazide
Tolbutamide	Chlorothiazide
	Chlorthalidone
	Indapamide
	Metolazone
Other drugs	
Sumatriptan	
Naratriptan	
Topiramate	
Ibutilide	
Sotalol	
Zonisamide	

The typical presentation is a bilateral ACAG that occurs within the first several doses of the sulfonamide-containing medication. There are cases reported after a single dose in patients who had taken the medication weeks or months previously without notable visual side effects. The most common presenting symptom is blurred vision. It is due to forward movement of the lens and increased lens thickness, which produces myopia. The forward position of the lens could explain about half of the myopic shift.[48] Fan *et al*[23] reported a case of ACAG in an individual who took oral acetazolamide and subsequently developed myopia in both the phakic and pseudophakic eyes. The induced myopia was attributed to forward movement of intraocular lens.

It has been documented that human immunodeficiency virus-infected individuals have a high risk for hypersensitivity reactions to sulfamethoxazol.[49] A 49-year-old man with acquired immunodeficiency syndrome who started pneumocystis carini prophylaxis with sulfamethoxazole trimethoprim (cotrimoxazole) developed bilateral ACAG and 360 degree choroidal effusions in both eyes after just 4 days of drug use. Although the medication was stopped, the patient developed bilateral cataracts and phthisis bulbi in 2 months.[42]

In order to treat these patients, the sulfa-containing drug should be discontinued and IOP should be controlled by antihypertensive medicines. Topical steroid, cycloplegic agents, and aqueous suppressants

are effective in treating this condition. Although cross-reactivity between sulfa drugs is rare, treatment with topical or systemic carbonic anhydrase inhibitors is contraindicated to control IOP in these patients.[50,51] Rhee *et al*[52] reported the beneficial effect of intravenous methylprednisolone and mannitol in controlling the acute glaucoma induced by topiramate within 4 h in one patient. Topical miotics should be avoided as they may precipitate a relative pupillary block. Laser iridotomies or peripheral iridectomy would alleviate the CAG only if a pupillary block component is present, but otherwise are of no benefit.[48] In those who do not respond to medical therapy, the possibility of supracilliary fluid accumulation should be taken into account.[53] Parikh *et al*[54] reported the successful outcome for ciliochoroidal effusion drainage in a case of bilateral ACAG with topiramate. After choroidal drainage, the anterior chamber deepened, corneal edema resolved, and IOP was controlled without medication.

Gentamicine has been reported to induce mydriasis after topical use in a patient, which theoretically can cause ACAG in individuals with narrow iridocorneal angle.[55] However, no case of gentamicine-induced glaucoma has been reported yet.

Central nervous system agents

Antidepressants

Antidepressants are used in the treatment of psychoneurotic anxiety or depressive reactions. Selective serotonin reuptake inhibitors (SSRI) are increasingly the first line choice of antidepressant because of their tolerable side-effect profile and low rate of lethality, if taken in an overdose. They act by producing a gradual increase in postsynaptic levels of serotonin. The pathophysiological mechanism of SSRI-induced ACAG remains unclear; although anticholinergic effects or increased levels of serotonin, which cause partial pupillary dilatation, and increased aqueous production secondary to increased ciliary body blood flow have been implicated.[56,57] The cause of ACAG in other groups of antidepressants is induced mydriasis by anticholinergic action of these medications in patients with narrow iridocorneal angle. Of tricyclic agents, amitryptiline (the most commonly reported agent) and imipramine[58] and of the non-tricyclic drugs, mianserin hydrochloride,[59] paroxetine,[57,60] fluoxetine,[61] maprotiline,[62,63] fluvoxamine,[64] venlafaxine,[65,66] citalopram,[67] and escitalopram[68] have been documented to be associated with ACAG attacks. Ritch *et al*[58] reported development of ACAG in four patients with occludable angles who received routinely prescribed doses of imipramine.

Monoamine oxidase inhibitors, another group of antidepressant agents, have weak anticholinergic effect. Phenelzine sulfate and tranylcypromine sulfate have been reported to produce CAG.[69]

Antipsychotics

Compared with tricyclic antidepressants, antipsychotics have a relatively weaker anticholinergic action and lower possibility of inducing CAG.[70] Among the available antipsychotic agents, perphenazine, trifluperazine, and fluphenazine have been reported to induce ACAG.[37,69,71] The anticholinergic effects—blurred vision, mydriasis, and decreased accommodation are more likely to occur with concomitant use of anti-Parkinsonian's agents and care in the use of this drug combination should be taken in patients with narrow angles.[16]

Benzodiazepines

The benzodiazepines are a group of compounds with related chemical structure that exert a therapeutic effect via binding to a site on the γ-aminobutyric acid (GABA) receptor complex and enhancing the inhibition that occurs when GABA binds to its receptor.[72] They are effective in the management of psychoneurotic states manifested by anxiety, tension, or agitation. They are also used as adjunctive therapy in the relief of skeletal muscle spasms and as preoperative medications. Theoretically, these agents can induce ACAG, because they induce relaxation of the sphincter muscle of the iris and have a mild anticholinergic effect.[73] Diazepam was suspected as having caused ACAG in a case report, also clotiazepam and alprazolam in another case, but there is no other report of glaucoma induction by these agents.[63,74]

Anti-Parkinsonians

Cabergoline, a dopamine D_2 receptor agonist, is also prescribed in prolactin producing pituitary gland tumors, and dysfunctions associated with hyperprolactinemia has been reported to induce non-pupillary block ACAG associated with choroidal effusion 5 h after ingestion of a single tablet.[75]

Orphenadrine citrate, an anticholinergic agent used also for the treatment of muscle spasm, has been documented to precipitate ACAG.[37]

Trihexyphenidyl, also known as benzhexol, is an antimuscarinic, which has peripheral and central anticholineric activity. It has been shown to precipitate CAG in patients with occludable angles.[76] Trihexyphenidyl has a mydriatic effect, which is about one-third as strong as that of an equal dose of atropine systemically. In addition to precipitating an ACAG attack, its prolonged

cumulative effect may cause chronic CAG in susceptible individuals. Owing to cognitive deficits in some patients with Parkinson's disease, the diagnosis of the glaucoma may be delayed. Friedman and Neumann[13] reported total blindness of one eye in two patients and legal blindness with tubular vision in one eye of a third patient that occurred as a result of CAG due to long-term treatment with trihexyphenidyl. It is, therefore, advisable to have gonioscopic evaluation in all patients who are to be put on trihexyphenidyl.[13]

Anticonvulsants

Topiramate, a sulfamate-substituted monosaccharide antiseizure medication, is also used in the management of migraine, depression, and neuropathic pain. It is also used off-label as a weight-reduction medication. More than 114 cases of topiramate-induced CAG have been reported in the literature. These usually occur within the first 2 weeks after starting topiramate therapy. In some cases, attacks develop within hours after doubling the dosage. Almost all cases are bilateral ACAG. In only three patients, attacks have been reported as unilateral. Glaucoma occurred between days 1 and 49, after drug initiation, with an average of 7 days. In a pilot study by Leung *et al*,[77] it was shown that short-term use of topiramate did not induce asymptomatic angle narrowing. Therefore, it was suggested that topiramate-induced secondary CAG may be an all-or-none phenomenon. The most prevalent presenting symptom was blurred vision due to myopia. Acute myopia up to 6–8.5 diopters may occur in a matter of hours after starting this drug; however, it may take a number of weeks to resolve once the drug is discontinued. Seven patients sustained permanent vision loss.[48,49,78,79]

Ecstasy (3, 4-methylenedioxymethamphetamine)

Ecstasy, a synthetic amphetamine derivate, and marijuana was implicated as the cause of recurrent bilateral ACAG in a 29-year-old woman.[80] Ecstasy increases the release of monoamine neurotransmitters and inhibits the uptake of serotonin from the synaptic gap that can lead to mydriasis and ACAG in predisposed persons.

Respiratory system agents

Epinephrine used for asthmatics with severe symptoms, for epistaxis, and in cold remedies may cause mydriasis and precipitate CAG in predisposed individuals.[20,81–83]

Ipratropium bromide, an anticholineric agent used to relive bronchoconstriction causes mydriasis and might precipitate an ACAG attack in susceptible individuals.[3,84–86] Three of five patients who developed ACAG after receiving ipratropium and salbutamol experienced bilateral ocular involvement.[84]

Tiotropium bromide, another anticholinergic agent, has weaker anticholinergic activity, but has been reported to induce ACAG.[87] Ipratropium and tiotropium can be absorbed through the cornea and the conjunctiva in the nebulized form when the mask is not fitted properly.

The β-2 agonists relieve reversible bronchospasm in patients with asthma or chronic obstructive pulmonary disease by relaxing the smooth muscles of the bronchi. The β-2 adnergics can compound the problem of CAG secondary to anticholigergic agents' mydriasis by increasing the production of aqueous humor.

Cocaine has indirect sympathomimetic activity and causes mydriasis. ACAG has been reported following therapeutic or abuse intranasal application of cocaine.[88,89]

Cardiac agents

Disopyramide, an anticholinergic agent, is indicated for suppression and prevention of recurrence of cardiac arrhythmias. Owing to its anticholinergic activity, it may induce ACAG up to 3 weeks after the patient starts taking the medication.[90,91]

Calcium channel blockers, though may be regarded as a neuroprotective and IOP reducing agent, have been reported to have incremental effect on the IOP.[92,93]

Hematologic agents

Anticoagulants have been described as inducing massive spontaneous choroidal hemorrhages. This is an extremely rare event that cause ACAG in older patients (65–87 years old) receiving anticoagulants or thrombolytic agents. Systemic hypertension and generalized atherosclerosis are additional risk factors. In addition to overtreatment with anticoagulants, some ocular issues such as age-related macular degeneration with neovascularization and nanophthalmos are risk factors for this uncommon complication.[94–98] Four patients with age-related macular degeneration who were on phenprocomoun or heparin developed unilateral ACAG after massive intraocular bleeding. Three of the four eyes were blind within a few months even with surgical evacuation of blood.[99] Chandra *et al*[98] reported a patient with history of atrophic age-related macular degeneration that was on warfarin with a therapeutic international normalized ratio and presented with CAG due to spontaneous suprachoroidal hemorrhage.

To manage increased IOP, anticoagulative agents should be discontinued (if the patient's medical condition allows) and treatment of CAG started. Consultation with patient's physician for alternative

anticoagulative medicine may be necessary.[100] Peripheral iridotomy is not effective because the mechanism is non-pupillary block CAG. Surgery may be needed to drain the choroidal effusion or evacuate the hemorrhage.[97,98] Evacuative sclerotomies may have value in the relief of pain and elevated IOP, but has not been shown to be beneficial in visual and anatomic outcomes.[100,101]

Docetaxel and paclitaxel belong to a generation of anticancer agents used in the treatment of a variety of neoplastic diseases, primarily in the advanced stages. There is a report of a patient with breast cancer and diffuse bone metastasis who developed IOP elevation with docetaxel.[102] She received docetaxel every 21 days. Before each cycle, she received premedication with 50 mg prednisolone 3 and 12 h before docetaxel. After the first cycle, she developed fluid retention and after the fifth cycle, she complained about loss of vision. She had an IOP of 44 mm Hg, but without any glaucomatous optic nerve or visual field abnormality. The mechanism of this IOP elevation is not determined. Although the authors stated that fluid retention could have been responsible for IOP elevation, the possibility of steroid-induced IOP elevation cannot be ruled out.

Imatinib mesylate is a selective inhibitor of the bcr-abl, c-kit, and platelet-derived growth factor receptor tryrosine kinases. This drug is an effective targeted therapy for patients with chronic myelogenous leukemia and gastrointestinal stromal tumors. In a case series on 104 patients, only one patient developed IOP elevation.[103]

Gastrointestinal agents

Spasmolytics, anticholinergic agents, are effective in the management of gastrointestinal tract spasticity and peptic ulcers. Although no case of CAG has been reported with these agents, dicyclomine and propantheline have been reported to increase the IOP in patients with open-angle glaucoma. The mechanism for this is not understood.[104]

Antihemmorhoidal suppositories may contain epinephrine compounds, which can precipitate ACAG in patients with narrow iridocorneal angle.[3,37]

Scopolamine, an anticholinergic drug, used for treatment of intestinal cramping and its time-release discs dermatologic application for the treatment of motion sickness should be prescribed with caution in the susceptible patients.[105,106] In a double-masked randomized, placebo-controlled study on 40 patients with open angle who were on treatment regimens that included or excluded pilocarpine, transdermal scopolamine patches were prescribed. No statistically significant differences in IOP were detected for either group.[107]

Cimetidine and ranitidine, H_2-blocker agents, have weak anticholinergic adverse effects, which may induce CAG in susceptible individuals. These have been documented to raise the IOP in one patient with glaucoma being treated for a duodenal ulcer.[108] However, Cohen *et al*[109] documented no effect on the IOP in any of four medically controlled glaucoma patients treated with intravenous infusion of cimetidine compared with the effect of a control infusion of normal saline.

Immune system agents

Antiallergic agents

Promethazine, an H1-blocker agent, has been shown to produce an idiopathic swelling of the lens that can induce ACAG.[110] Although the possibility of inducing CAG with these agents is low because of their miniscule anticholinergic activity, they should be used with caution in at-risk patients.

Diphenhyramine used in the intravenous form has been regarded as a causative agent for ACAG and fexofenadine has been suggested as an alternative in patients at risk of CAG.[111]

Antiinflammatory agents

Mefenamic acid, a non-steroidal antiinflammatory agent, has been documented to induce secondary non-pupillary block CAG and −9.00 diopter myopia in a 30-year-old patient. This is the same clinical picture that occurs with sulfa drugs.[112]

Anesthetic agents

The majority of drugs used for general anesthesia cause a decrease in IOP.[113] However, the induction of general anesthesia may be associated with an elevated IOP from laryngeal spasm and coughing associated with endotracheal intubation.[114] Additionally, succinylcholine[115] and ketamine[116] have been documented to elevate IOP. This effect seems to be due to increased extraocular muscle tone from these drugs. The IOP elevation is temporary and has no harmful effect on the eye except in patients with ruptured globe. Avoidance of using these agents in this condition is the best preventive method.

Postoperative ACAG after non-ocular surgery has been reported after various procedures, including abdominal, orthopedic, facial, gynecologic, and endoscopic surgery.[117–121] Several factors are likely to induce postoperative ACAG in predisposed individuals such as the use of anticholinergics (atropine, scopolamine, and muscle relaxants) and adrenergics (ephedrine and epinephrine). Moreover, the perioperative period carries the risk of psychological stress and darkness-induced mydriasis may increase the

risk of glaucoma attacks.[3,118,122] Owing to the patient's systemic early postoperative condition, the classic signs of ACAG may not be detected and the diagnosis can be delayed.

Conclusion

There are numerous reports of drug-induced glaucoma; however, controlled, comparison trials of agents involved in this subject are virtually non-existent and most data are obtained from case reports and case series. Although the results from these types of studies may be useful in developing instructions for the optimal use of these agents, controlled trials are required for establishing standard treatment instructions.

Drug-induced glaucoma is an issue that is preventable in many cases. The main mechanism is pupillary block CAG caused by the anticholinergic or adrenergic activity of systemic or topical drugs in patients with narrow iridocorneal angle. For this mechanism, prophylactic laser iridotomy in high-risk individuals offers protection against drug-induced glaucoma. The other mechanism is non-pupillary block CAG that occurs because of ciliary or suprachoroidal effusion, or even vitreous hemorrhage with forward movement of the iris–lens diaphragm. In contrast to the first mechanism where laser iridotomy is curative, in the latter group laser iridotomy is ineffective.

In the case of ACAG, the problem is to know which eye is at risk as the patients are unaware that they have narrow iridocorneal angles and the practitioner prescribing systemic medication are not able to detect at-risk patients. It is wise to send high-risk patients (hyperopic, having family history of glaucoma, being from a high-risk ethnicity such as Asian) for ophthalmic examination before commencing agents that have potential of inducing CAG. All in all, systemic and local drug effects may lead to open or more commonly CAG. Prompt recognition and appropriate treatment may help protect the vision of these susceptible patients and the clinician must be watchful of the potential to cause glaucoma associated with systemic medications that are necessary in the treatment of various systemic diseases.

Conflict of interest

The authors declare no conflict of interest.

Acknowledgements

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