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What You Need To Know: Acute Primary Angle Closure Glaucoma

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INTRODUCTION

Acute angle closure glaucoma (AACG) is an ophthalmological emergency that is an uncommon cause of the acute red eye in general practice. It is a form of glaucoma in which the intra-ocular pressure (IOP) rises suddenly and markedly when the peripheral iris closes the anterior chamber angle, resulting in a physical barrier to the outflow of aqueous humour.

Epidemiology

Glaucoma is the second most common cause of blindness in the world after cataracts. It is estimated that 6.7 million people worldwide suffer from bilateral blindness due to primary glaucoma[1]. While sight can be restored in cataracts, glaucoma usually results in irreversible visual loss[2]. Unlike Europeans, Latin Americans and Africans, angle closure glaucoma predominates among Asian people where its prevalence outnumbers that of open angle glaucoma by three-fold[2,3].

The incidence of AACG in Singapore is 12.2/100,000/year, with the elderly Chinese female at greatest risk. This is significantly higher than that of the Finns, Thais, Israelis and Japanese[4]. Asian, particularly Chinese eyes are predisposed to angle closure glaucoma because the anterior chamber is shallower. It is still unknown what makes one predisposed person present with an attack of AACG and yet another slowly loses her or his vision with chronic angle closure.

Evolution of an Attack

In the normal eye, aqueous humour flows from the posterior chamber, through the pupil to the anterior chamber and is drained mainly via the trabecular meshwork into the episcleral veins[5].

In the predisposed eye, pupillary block occurs when the dilator muscle of the iris contracts, increasing the amount of apposition between the iris and lens, thus increasing the pressure in the posterior chamber. The simultaneous dilatation of the iris makes it more flaccid, allowing it to increasingly bow anteriorly with the rise in posterior chamber pressure. Eventually, there is complete apposition of the peripheral iris to the trabecular meshwork, resulting in a rapid rise in the IOP.

The elderly eye is predisposed because the lens, being the only structure in the eye that grows even in adulthood, is in a relatively more anterior position. Primary AACG rarely ever occurs below the age of 40. Other predisposing factors include a short eyeball, hypermetropia and autonomic neuropathy.

Attacks are most commonly triggered in the mid-dilated position. It has been proposed that at that position, the posteriorly directed forces of the dilator muscles during contraction are the greatest[6].

Clinical presentation (Table I)

The symptoms of an attack result from a sudden marked elevation of IOP to as high as 80 mmHg; the normal IOP being 16±5 mmHg. The patient experiences intense pain, blurred vision, tearing and lid oedema. These may be accompanied by significant systemic symptoms such as nausea, vomiting and sweating. Some may give a history of previous intermittent attacks which had resolved spontaneously.

Common precipitating events include illness, pharmacologic dilatation, intense concentration, trauma and emotional stress. Acute attacks have also been reported after panretinal photocoagulation and as first presentation of AIDS.

Table I - Clinical presentation of acute angle closure glaucoma

Symptoms	Signs
painful red eye	circumcorneal injection
headache	hazy cornea
blurred vision	decreased visual acuity
nausea & vomiting	eclipse test
	increased intra-ocular pressure

Establishing the diagnosis

On examination, there is circumcorneal injection and conjunctival oedema. The cornea is oedematous, the pupil is mid-dilated and may be sluggishly reactive. Visual acuity is also reduced and the globe feels stony hard. If a torchlight is shone obliquely across the cornea, the anterior chamber may be obviously shallow with only the temporal iris being illuminated (see Fig 1). Also known as the eclipse test or the oblique flashlight test, this test is useful in the GP practice as it does not require special instruments.

Other causes of the acute red eye include corneal abrasions, foreign bodies (conjunctival, subtarsal, corneal), infection, iritis etc. However, AACG is distinguished by its visual loss, hazy cornea and severe pain.

Management of AACG

The principle of treatment is to rapidly lower the IOP in order to relieve pain as well as to allow the cornea to clear so that a laser peripheral iridotomy can be performed. Although AACG is an emergency that

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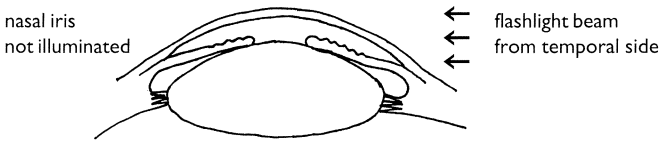

Fig 1

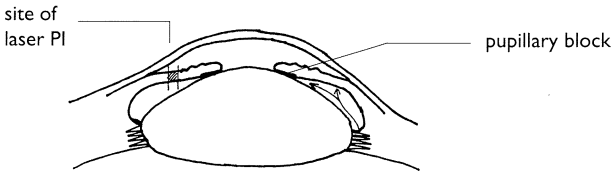

Fig 2

requires hospitalisation, treatment should begin in the clinic with timolol (0.5% stat and bd) and pilocarpine (2% stat and qds) eyedrops. Oral or intravenous acetazolamide 250-500 mg, depending on the patient's ability to take orally, should also be given if there is no history of sulphur allergy (Table II). Timolol and acetazolamide rapidly decrease aqueous humour production while pilocarpine constricts the pupil (provided the iris is not paralysed as a result of ischaemia).

Although pilocarpine is available in 2% and 4% strengths, the higher concentration is not recommended because it causes the ciliary muscles to contract, further displacing the lens diaphragm anteriorly, worsening the pupillary block. Overdose of pilocarpine, 60 drops or 100 mg, will result in systemic cholinergic side effects such as bronchospasm vomiting and bradycardia[7].

Timolol is a non-selective β-blocker and thus may exacerbate pre-existing respiratory and cardiac disease. As it crosses the blood-brain barrier, it may also cause confusion and disorientation[8].

To minimise systemic absorption of eyedrops, not more than one drop at a time should be applied, decreasing the amount of drug that overflows into the lacrimal drainage. Eyelid closure for 5 minutes after instillation will reduce the blink-induced action of the naso-lacrimal pump and thus also reduce the systemic effects of the drugs[8].

As acetazolamide is a sulphur drug, there is a risk of cross-sensitivity in patients with a history of sulphur allergy. It should also be used with caution in patients who are significantly dehydrated, have electrolyte abnormalities, on digoxin or have chronic obstructive pulmonary disease or renal failure[9].

In 90% of cases, medical therapy with or without a laser procedure is sufficient to abort the attack[10].

In the in-patient setting, IOP and corneal clarity are monitored frequently, and once the cornea is clear, laser peripheral iridotomy (PI) is performed. Peripheral iridotomy provides an alternative bypass between the anterior and posterior chambers for the flow of aqueous humour, thus relieving the pupillary block (Fig 2).

If medical therapy fails to break the attack, argon laser peripheral iridoplasty is used to open the closed angle. This consists of placing a ring of contraction burns circumferentially on the peripheral iris in order to contract the iris stroma between the site of the burn and the angle, thus widening the angle itself.

As AACG is an anatomical disorder that occurs in anatomically predisposed eyes, the fellow eye has a 50%-75% probability of being affected within 5 years[11]. Laser PI, which is fairly simple and safe, is routinely and prophylactically performed in the fellow eye.

Sequelae of an attack of AACG

About a third of patients go on to develop chronic glaucoma within the first 4 months to 3 years. This tends to occur if there was a delay of more than 24 hours in initiation of treatment and those in which the attack could not be aborted with medical treatment alone[10]. Unfortunately, only a third of our patients seek medical attention on day one of the attack[4].

It is thought that chronic glaucoma develops as a result of trabecular meshwork damage. Such patients require a filtration procedure such as trabeculectomy.

Ischaemia and inflammation of the iris occurring during the attack may result in a permanently dilated pupil, posterior synechias and segmental iris atrophy. The cornea may decompensate due to endothelial cell loss. Visual field defects similar to those found in other forms of glaucoma may develop in those patients who have recovered from an attack[12].

Table II - Medical treatment of acute angle closure glaucoma

Pilocarpine eyedrops 2% stat, 15 minutes later and qds
Timolol eyedrops 0.5% stat and bd
Acetazolamide iv 250-500 mg stat and tab 250 mg qds
Analgesics
Anti-emetics
Hyperosmotic agents

CONCLUSION

AACG is an ophthalmological emergency to which our population is predisposed. In our ageing population, its incidence, and therefore morbidity, is expected to rise. It should be recognised and empirical treatment initiated before referral to a tertiary centre. The response to non-invasive treatment is good.

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