Recognition and management of acute glaucoma

Acute glaucoma is a medical emergency that often occurs in the evening when the GP is the first port of call. Early assessment, initiation of treatment and appropriate referral will maximise the amount of vision saved.

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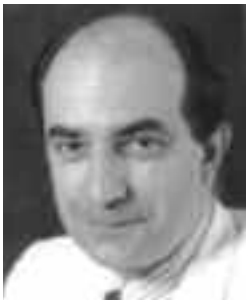

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Acute angle closure glaucoma (referred to as acute glaucoma in this article) is a treatable and preventable cause of blindness that affects about one in 1000 people aged over 40 years. Its prevalence increases with age, and it is more common in Asian than Caucasian people. Early recognition of acute glaucoma and prompt referral will optimise the amount of vision saved.

The GP is frequently the first port of call for an urgent night-time consultation by a patient with acute glaucoma. It is therefore very important to be able to recognise the symptoms and signs of acute glaucoma, to initiate treatment if available and to organise immediate referral to an ophthalmologist – no matter what time of day or night.

Normal anatomy of the angle

The angle of the anterior chamber of the eye refers to the anatomical angle between the cornea and the iris (Figure 1). Contained in the angle is the trabecular meshwork, which is a series of interweaved fibres that communicate with the Canal of Schlemm – a modified vein. Aqueous humour is secreted from the ciliary body and passes through the pupil into the anterior chamber angle, draining through the trabecular meshwork spaces into the Canal of Schlemm. From the Canal, the aqueous seeps by way of a plexus of fine veins (collector channels) through the sclera into the episcleral veins. The constant production, circulation and drainage of aqueous maintain a more or less constant intraocular pressure within the range of 11 to 22 mmHg.

Mechanism of angle closure

The risk of developing acute glaucoma is associated with age as well as hereditary and racial factors. People with a small anterior segment and a shallow anterior chamber have an anatomical predisposition for developing acute glaucoma. Those in whom the anterior chamber tends to be shallow include women, Asian people and those with hypermetropia. As these people age, the lens

IN SUMMARY

- **Acute angle closure glaucoma is a medical emergency that requires immediate referral to an ophthalmologist.**
- **During a subacute glaucoma attack, patients may see rainbow-coloured haloes around lights and have some mistiness of vision and eye discomfort. These symptoms always warrant referral to an ophthalmologist.**
- **Acute glaucoma causes sudden profound reduction of vision, severe pain in and around the eye and often nausea and vomiting. Cardinal signs include a red eye, with a mid-dilated, fixed ovoid pupil and a cloudy cornea.**
- **The differential diagnoses are those of 'the acute red eye', which include iritis, conjunctivitis, keratitis and episcleritis.**
- **Acute glaucoma may be prevented by laser peripheral iridotomy.**

thickens and tends to move forwards in the eye, narrowing the angle further. A precipitating factor, such as mid-pupillary dilation (for example, while watching television in dim lighting or a film at a cinema) or forward lens movement when lying prone, can trigger an acute attack.

An acute attack starts with the aqueous being restricted in its passage from the posterior to the anterior chamber (pupil block). This creates a pressure differential that pushes the peripheral iris forwards into contact with the trabecular meshwork, occluding the angle of the anterior chamber (Figure 2). Aqueous can no longer escape and the intraocular pressure builds up rapidly to very high levels (60 to 70 mmHg).

Warning attacks

About half of the patients presenting with acute glaucoma give a history of prodromal episodic subacute attacks. They report episodes of eye pain, blurring of vision and seeing rainbow-coloured haloes around lights. After one to two hours, these attacks are ended usually by constriction of the pupil from exposure to bright light or sleep. Sooner or later, an episode of raised intraocular pressure does not spontaneously resolve and classic acute glaucoma results.

Prevention

Patients' eyes can be screened for shallow anterior chambers by shining a torch directed from the temporal side of the eye along the plane of the iris (Figure 3). The light casts a shadow nasally if the anterior chamber is shallow. Patients with suspected shallow anterior chambers warrant an ophthalmologist referral for assessment of the angle depth by gonioscopy. This involves placing a diagnostic contact lens (Goldmann or others) on the cornea. The lens refracts light so that the normally hidden angle comes into view. In people with confirmed narrow angles, a prophylactic laser peripheral iridotomy may prevent an acute glaucoma attack (Figures 4 and 5).

Clinical features of acute glaucoma

Acute glaucoma often happens in the evening, when it is precipitated by pupil dilation caused by an emotional upset or dim illumination (for example, in a cinema). It also may occur following general anaesthesia and should be considered in the differential diagnoses of any red or painful eye in a patient who has had a general anaesthetic.

Occasionally, nonspecific symptoms dominate the clinical presentation; hence, acute glaucoma should be considered in patients with headache, nausea or vomiting.

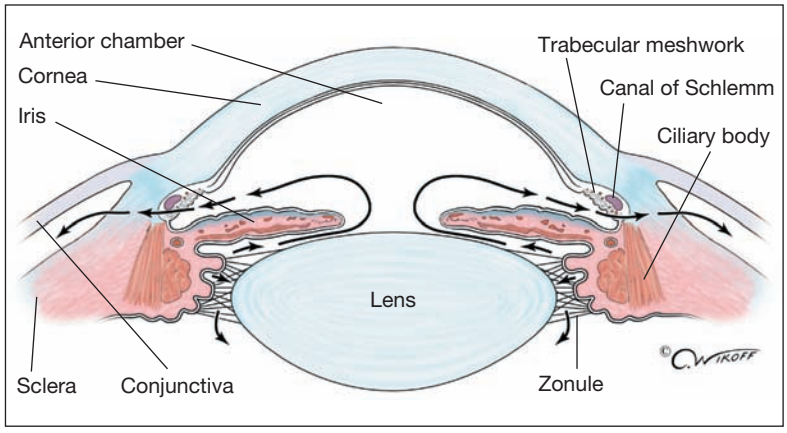

Figure 1. Circulation of the aqueous in a normal eye with an anterior chamber of normal depth.

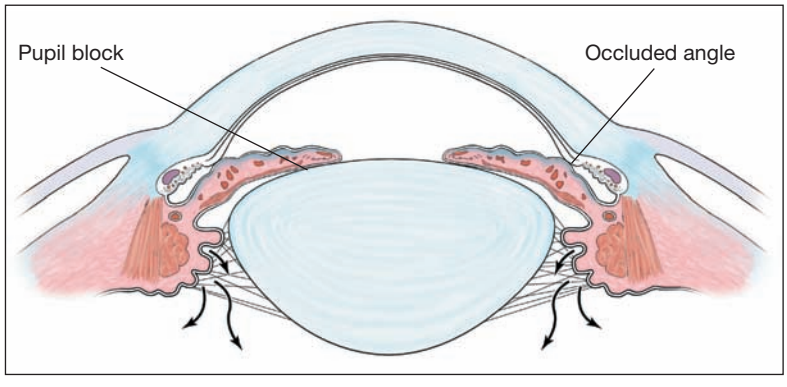

Figure 2. Angle closure in an eye with a narrow angle. As pressure increases in the posterior chamber the (enlarged) lens pushes the iris forward closing the angle.

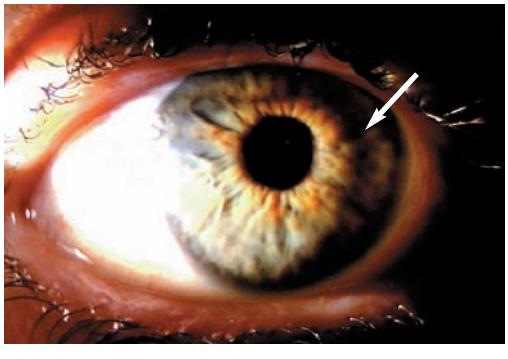

Figure 3. In an eye with a narrow angle, the iris bows forward and a torchlight directed from the temporal side of the eye along the iris plane casts a shadow nasally.

continued

Consultant's comment

Angle closure glaucoma remains a preventable cause of visual loss that is often misdiagnosed in the initial stages, leading to a delay in treatment. The degree of optic nerve, lens and corneal damage is directly related to the duration of elevated intraocular pressure.

Following the miosis precipitated by sleep or bright light, physiological pupil dilation in dim lighting may lead to premonitory symptoms, which are frequently transient. Consequently, they are often not reported by patients. Specific questioning, especially during pre-operative assessments, can help to identify patients at particular risk of angle closure glaucoma from general anaesthesia. All patients complaining of a red eye, headache, or even atypical nausea and vomiting after general anaesthesia should be specifically assessed for angle closure glaucoma, as delayed diagnosis can result in irreversible visual loss and become a medicolegal issue. Knowledge of those at high risk, well described in Dr Attebo and Professor Coroneo's paper, can raise the index of suspicion and facilitate earlier diagnosis.

Recently, there has been an increased understanding of the crystalline lens' role in the pathogenesis of acute glaucoma and there have been significant improvements in cataract microsurgical techniques. Eyes that previously suffered severe visual loss and required complicated glaucoma filtration surgery after unsuccessful laser iridotomy have achieved good pressure control with small-incision phacoemulsification lens surgery.

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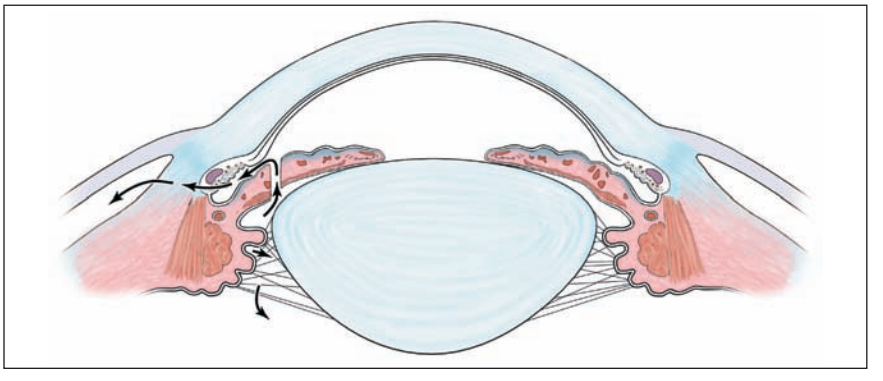


Figure 4. In an eye with a narrow angle, a peripheral laser iridotomy allows the aqueous to circulate thereby reducing the lens' forward force on the iris and opening the angle.

Symptoms

Patients may present with the following symptoms:

- rapid blurring of vision over several hours (due to corneal oedema)
- severe aching pain of the eye and orbit, which patients may describe as a severe headache
- nausea and vomiting, as a result of the pain – some patients may even be suspected of having gastrointestinal pathology
- photophobia and watering of the eye
- seeing rainbow-coloured haloes around artificial lights (such as streetlights or car headlights), due to diffraction in the oedematous cornea.

Signs

The signs of acute glaucoma (see Figures 6a and b) are as follows:

- a red eye due to ciliary vessels dilating, especially around the corneal margin, resulting in 'ciliary injection'
- a cloudy cornea due to corneal oedema
- a semi-dilated, oval pupil that does not react to light
- a rock hard orbit compared to the other or your own eye, detected by digital palpation with the index fingers, indicating the very high intraocular pressure.

Differential diagnoses

The five major causes of ocular pain and redness are conjunctivitis, keratitis, episcleritis, iritis and acute glaucoma. Table 1 shows the features most helpful in distinguishing these conditions. An important

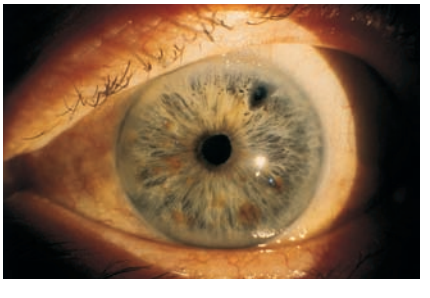
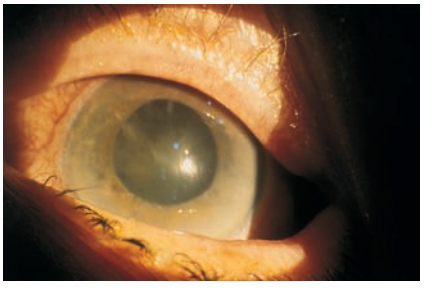
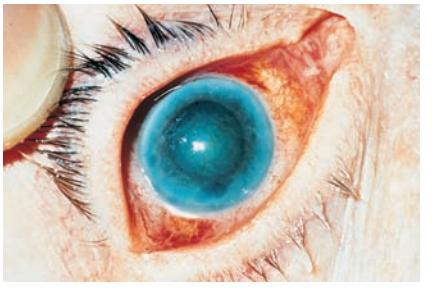


Figure 5. A laser peripheral iridotomy.





Figures 6a and b. Acute glaucoma in two patients: note mid-dilated pupils and cloudy corneas.

Table 1. Clinical features of common causes of red eyes

Symptoms	Signs
Acute glaucoma – usually unilateral	
Watery eye (mild)	Reduced visual acuity
Photophobia	Ciliary injection (may be minimal in early stages)
Blurred vision	Semi-dilated, oval pupil that is nonreactive to light
Seeing haloes	Cloudy cornea
Severe pain	Rock-hard orbit, indicating raised ocular tension
Frontal headache	
Nausea and vomiting	
Acute iritis – usually unilateral	
Watery eye	Reduced visual acuity
Blurred vision	Ciliary injection
Photophobia	Constricted pupil, may be irregular
Severe pain	Normal cornea
Keratitis – usually unilateral	
Watery eye	Reduced visual acuity
Photophobia	Ciliary injection
Blurred vision	Localised corneal opacification
Pain	
Conjunctivitis – usually bilateral	
Gritty sensation	Conjunctival injection
Discharge and stickiness	Swollen eyelids
Normal vision	Mucopurulent discharge
	Normal corneas and pupils
Episcleritis – usually unilateral	
Aching pain	Tenderness (localised to affected area)
Watery eye (mild)	Conjunctival injection (localised to affected area)
Normal vision	Normal cornea and pupil

differential diagnosis of severe headache in the elderly is temporal arteritis.

Treatment of acute glaucoma

Acute glaucoma is a medical emergency. Total and permanent loss of vision can occur within hours. Patients often delay seeking medical care when the onset of symptoms is in the evening (which is frequently the case) because they are reluctant to disturb their GP or ophthalmologist, or are unaware of other available after-hours care. Delay in treatment causes ischaemia of the optic nerve head, death of retinal nerve fibres, visual field loss and subsequent permanent loss of central visual acuity. The patient with suspected acute glaucoma requires immediate referral to an ophthalmologist.

The aims of treatment are to unblock the angle, reduce the volume of aqueous secreted and extract water from the eye. In addition to pilocarpine, the mainstays of medical therapy are osmotic agents, carbonic anhydrase inhibitors and beta blockers; hence, the patient's cardiovascular status, allergies and electrolyte balance must be considered when contemplating treatment. Table 2 shows contraindications, precautions and adverse reactions to the medications used in acute angle closure glaucoma. A summary of the patient's past medical history is useful for the ophthalmologist.

Initiating treatment

While waiting to organise referral and transfer, the GP should initiate treatment with miotic eye drops and acetazolamide.

Miotic drops when used intensively may reopen the angle if they succeed in causing the pupil to constrict. If the attack has lasted too long, the pupil will not respond because the iris sphincter is ischaemic and paralysed when the intra - ocular pressure is above 50 mmHg.

The usual protocol used is 2 to 4% pilocarpine eye drops, one drop into the affected eye every five minutes for half an hour, then every 15 minutes for a further two to three hours, and then four times a day. If the pupil has not constricted after 30 minutes of administering pilocarpine, it is unlikely that any additional pilocarpine will help until the intraocular pressure is reduced. Hence, the GP should stop applying pilocarpine drops after half an hour if the pupil has not constricted.

There is a risk of overdosing on pilocarpine. Patients may develop cholinergic side effects, such as sweating and abdominal pain, and excess pilocarpine may, rarely, make the angle closure worse by increasing the pupil block.

Acetazolamide (Diamox) 500 mg stat, intravenously or orally (if tolerated), should be administered as soon as possible, followed by 250 mg orally four times a day. This inhibits the secretion of aqueous. Acetazolamide thereby often lowers the intraocular pressure sufficiently for the miotic drops to be effective, which occurs when the pressure is below 40 mmHg. In some patients,

analgesics and antiemetics may also be required.

Further treatment

If the intraocular pressure does not decrease with the above treatment, an intravenous infusion of mannitol, 1 to 2 g/kg over 45 minutes, can be given. (A 500 mL bag of 20% mannitol contains 100 g of mannitol.) Mannitol causes extraction of water from the eye by increasing the hypertonicity of the blood.

The ophthalmologist may add topical treatment with a beta blocker (betaxolol [Betoptic, Betoquin]; timolol [Optimol, Tenopt, Timoptol]; levobunolol [Betagan]), an alpha-2 adrenergic agonist (apraclonidine [Iopidine]; brimonidine [Alphagan]) and a high dose corticosteroid such as dexamethasone (Maxidex) to decrease inflammation.

If medical treatment reduces the pressure to normal, it is continued until the cornea clears. This can take from one to five days. A laser peripheral iridotomy is then performed to try to prevent a recurrent attack. Up to two rounds of medication and two attempts at laser peripheral iridotomy may be undertaken to reduce intraocular pressure. If the pressure does not decrease, an urgent surgical removal of the enlarged lens by phacoemulsification and insertion of a thin posterior chamber intraocular lens can help to restore and deepen the anterior chamber of the eye.

Treatment of the other eye

It should be stressed to the patient that often the unaffected eye will undergo an attack of acute glaucoma within a few weeks of the initial episode, because both eyes usually have the same anatomical predisposition. Therefore, at the time of an acute glaucoma attack, it is important to instil 1% pilocarpine four times a day into the unaffected eye. The emotional upset and pain caused by the attack may otherwise dilate this eye and precipitate acute glaucoma.

If the angle in the second eye is also narrow, a prophylactic peripheral laser iridotomy needs to be performed. This is usually done within a week or two of operating on the first eye.

Table 2. Medications in acute glaucoma and some precautions to their use

Medication	Contraindications	Precautions	Adverse reactions
Pilocarpine eye drops (Isopto Carpine, Minims Pilocarpine Nitrate, Neutral Pilocarpine Eye Drops, Pilocarpine, Pilopt, P.V. Carpine)	Conditions where pupillary constriction is undesirable	Retinal disease (retinal tears, previous retinal detachments), pregnancy, lactation, children	Visual blurring, ocular pain, conjunctival injection, retinal detachment, cataract formation
Acetazolamide, intravenous or oral (Diamox)	Sulfur allergy, depressed serum sodium or potassium levels, marked renal or hepatic impairment, cirrhosis, suprarenal gland failure, hyperchloraemic acidosis, pregnancy	Pulmonary obstruction, emphysema, lactation; interacts with oral anticoagulants and antihypertensive agents	Sulfonamide reactions (including rash, Stevens–Johnson syndrome, hepatic necrosis and aplastic anaemia), paraesthesiae, hyperchloraemic metabolic crisis
Beta blocker eye drops (betaxolol [Betoptic, Betoquin]; levobunolol [Betagan]; timolol [Optimol, Tenopt, Timoptol])	Bronchospasm, sinus bradycardia, 2nd and 3rd degree AV block, overt cardiac failure, cardiogenic shock	Cardiac disease, soft contact lenses, pregnancy, lactation, children	Bronchospasm, cardiovascular effects, headache, allergic reactions, gastrointestinal upset
Alpha-2 adrenergic agonist eye drops (apraclonidine [Iopidine]; brimonidine [Alphagan])	Concurrent MAOIs, systemic sympathomimetics or tricyclic antidepressants	Hypertension, severe cardiovascular disease, renal or hepatic impairment, pregnancy, lactation, children	Hypersensitivity, eye irritation
Mannitol, intravenous (Osmitrol Intravenous Infusion)	Anuria, severe pulmonary oedema, severe dehydration, severe renal impairment	Renal impairment, pregnancy, lactation, children	Electrolyte imbalance, dehydration

Follow up

Often after a prolonged episode of acute glaucoma, the patient will need to continue on eye drops for glaucoma or have a trabeculectomy for long term control of the intraocular pressure. This is because the acute attack causes severe inflammation and ischaemia, causing damage to the trabecular meshwork cells and adherence of the peripheral iris to the posterior corneal surface (peripheral anterior synechiae), sealing over the angle in parts.

The patient should be instructed to alert his or her relatives about the occurrence of angle closure attacks. Primary angle closure glaucoma is highly inheritable and 30 to 50% of the first-degree relatives will be expected to have anterior chamber angles that are predisposed to occlusion.

Conclusion

Acute glaucoma is a medical emergency warranting immediate referral to an ophthalmologist. Episodes often occur in the evening when the pupil is mid-dilated due to dim illumination; thus, patients with acute glaucoma may present to their GP. Typical symptoms include a painful eye, blurred vision and seeing haloes around light sources. The eye is usually red, has a nonreactive semi-dilated pupil and feels rock hard compared to the other eye. The GP should initiate treatment with 2 to 4% pilocarpine eye drops and systemic acetazolamide, while organising urgent ophthalmologic review. Treatment of the other eye and screening of family members should not be forgotten. MT

Acknowledgement

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