Angle closure glaucoma: an overview

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Recognition of the symptoms of acute angle closure glaucoma, prompt intervention and appropriate referral are essential to reduce intraocular pressure and prevent permanent visual loss. Progressive circumferential closure of the drainage network of the eye is prevalent in Asians and is being increasingly recognised as a major cause of visual loss.

Glaucoma is the second leading cause of visual impairment worldwide.[1] Angle closure glaucoma accounts for almost half of these cases, and is emerging as a leading cause of blindness in Asian countries. With the significant and growing population of Asian origin in Australia, there is an increasing need for awareness of this potentially blinding disease in this country.

Acute angle closure is an ocular emergency that requires rapid diagnosis, prompt intervention and referral to prevent progression and consequent loss of vision. Prophylactic measures are essential to prevent an attack in the fellow eye.

Epidemiology

It has been estimated that the prevalence of people with angle closure glaucoma worldwide in 2010 will be 15.7 million.[2] This figure is expected to increase to 21 million by 2020, and of these affected people 5.3 million will be bilaterally blind from the disease.[2] Angle closure glaucoma is more common among South-East Asian and Chinese peoples and Inuits than among Caucasians and Africans. Older people are generally affected (in the sixth to seventh decade of life) and women are affected two to four times more commonly than men.

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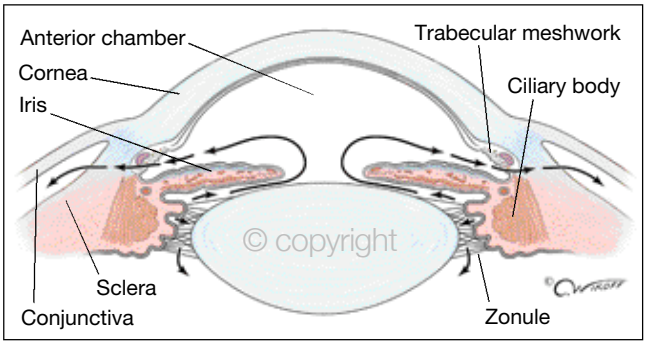

Figure 1. Aqueous circulation in a normal eye (with an anterior chamber of normal depth). Aqueous is secreted by the ciliary body, passes through the pupil into the anterior chamber and drains through the trabecular meshwork contained in the angle between the cornea and the iris. (Arrows indicate direction of aqueous flow.)

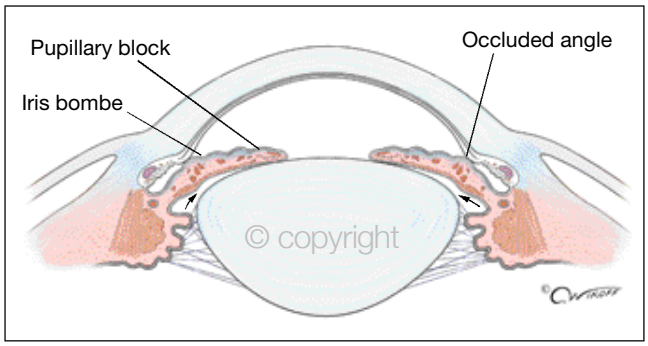

Figure 2. Angle closure due to pupillary block in an older eye with a shallow anterior chamber and a narrow angle. Pupil dilation can cause the iris to catch on the lens, restricting the normal flow of aqueous from the ciliary body around the lens to the anterior chamber, leading to a pressure differential between the posterior and anterior chambers. As aqueous builds up behind the iris, the peripheral iris bows forward (iris bombe) and occludes the angle, blocking access of the aqueous to the trabecular meshwork and causing the pressure to rise further. The lens (which thickens with increasing age) may also push the peripheral iris forward. (Arrows indicate direction of aqueous flow.)

Terminology

Angle closure glaucoma refers to a disorder of raised intraocular pressure (IOP) due to mechanical blockade, usually by the peripheral iris, of the access to the drainage mechanism of the eye (the trabecular meshwork), with subsequent optic disc and visual field changes.

Conceptually, there are five stages in the progression of angle closure glaucoma of unknown cause (primary angle closure glaucoma). These stages range from latent, through subacute, acute and chronic to absolute, the latter referring to the end-stage of untreated disease – an irreversibly blind (no perception of light)

Table 1. Drugs that may precipitate angle closure

- Adrenergic agents – phenylephrine drops, nebulised salbutamol, systemic adrenaline
- Anticholinergics – atropine and homatropine drops, periocular botulinum toxin
- Antihistamines – chlorpheniramine, diphenhydramine
- Antidepressants – amitriptyline, fluoxetine, imipramine, paroxetine, phenelzine
- Selective serotonin reuptake inhibitors – citalopram, venlafaxine
- Sulfa-based drugs – hydrochlorothiazide, sulfamethoxazole, topiramate

Table 2. Symptoms and signs of primary angle closure

Symptoms

Periocular pain
Headache
Blurred vision
Coloured haloes around lights
Nausea, vomiting, sweating and bradycardia

Signs

Red eye – ciliary injection
Cloudy cornea
Mid-dilated pupil, not responsive to light
Hard orbit

eye. In reality, the subacute and acute closure stages merge, and it is this stage that is most commonly encountered in a primary care setting.

Previously, with a lack of standard definitions and criteria, the terms primary angle closure, primary angle closure glaucoma, acute angle closure and acute angle closure glaucoma were used interchangeably. With greater understanding of the epidemiology and revision of the classification, the term primary angle closure glaucoma is now being reserved to indicate the presence of glaucomatous optic neuropathy, with its characteristic visual field defects, in eyes that have signs of primary angle closure.[3] Patients with occludable angles are referred to as primary angle closure suspects.

Chronic angle closure glaucoma refers to the progressive permanent closing of access to the trabecular meshwork, with a gradual rise in IOP and subsequent optic and visual field defects. It is discussed later in this article.

Mechanisms of angle closure

In the normal healthy eye, a constant pressure (normal range, 10 to 21 mmHg) is maintained by the continuous production, circulation and drainage of aqueous humour. Aqueous is secreted by the ciliary body, passes through the pupil into the anterior chamber and then drains through the trabecular meshwork contained in the angle between the cornea and the iris (Figure 1). Trabecular obstruction consequent to closure (occlusion) of this angle because of iris–lens apposition results in raised IOP and characteristic symptoms and signs – the condition of angle closure.

Racial and hereditary factors and increasing age are important risk factors for angle closure. Several anatomical features lead to anterior chamber crowding and predispose individuals to angle closure. These include shallower anterior chambers, thinner ciliary bodies, anteriorly situated iris–lens diaphragms and shorter axial eye lengths. Women and Asian people tend to have shallow anterior chambers, as do older people because of the increasing thickness and forward movement of the lens with increasing age. First-degree relatives of people with angle closure are at increased risk because eye shape is often inherited.[4]

Pupillary block (pathologic iris–lens apposition) is probably the most frequent underlying cause of angle closure (Figure 2). In pupillary block, the resistance to aqueous flow from the posterior chamber through the pupil increases and leads to a pressure differential between the posterior and anterior chambers. This pressure gradient causes the peripheral iris to bow forward (iris bombe) and obstruct the trabecular meshwork, with a consequent elevation of IOP. This is most likely to occur when the pupil is mid-dilated. The severe pain in cases of acute angle closure is caused by the very high IOP, as is also the potentially devastating damage to the optic nerve.

Less common mechanisms of angle closure include plateau iris, lens swelling and ciliary block. Plateau iris is due to anterior insertion of the iris on the ciliary body face, with the superfluous and crowded iris tissue directly blocking the trabecular meshwork and subsequently increasing IOP. Phacomorphic (or lens shape-related) glaucoma and ciliary block (or malignant) glaucoma are relatively rare. Phacomorphic glaucoma is due to the lens intumescence that may occur in advanced cataracts, with subsequent crowding of the anterior chamber. Ciliary block glaucoma is due to posterior misdirection of aqueous to the vitreous cavity, with increasing vitreous pressure and forward movement of the iris–lens diaphragm leading to angle closure. This has been described after intraocular surgery, in panretinal photocoagulation, after scleral buckling surgery for retinal detachment and in uveitis.

In anatomically predisposed eyes, the natural response of pupillary dilation to various environmental and chemical stimuli can result in pathologic iris–lens apposition precipitating acute angle closure. Precipitants of an attack include dim illumination, as at night or in cinemas or theatres, and stress or excitement. Several local and systemic drugs may also be precipitants because of their adrenergic, anticholinergic or sometimes idiosyncratic (causing ciliochoroidal effusion) effects.[5] Practitioners prescribing these drugs need to be aware of their potentially blinding side effects (Table 1).

The mechanisms for chronic angle closure are discussed separately later in this article.

Presentation of primary angle closure

Patients with primary angle closure in the acute stage present with periocular pain and headache that is often severe, is usually not relieved by topical anaesthetics and may radiate in the trigeminal nerve distribution (Table 2). However, it is important to note variability of symptoms in an acute attack; some patients, especially Africans, may have very little pain despite very high IOPs.

Blurred vision and coloured haloes around lights may progress to total visual loss. There may be a past history of transient blurring of vision and haloes around lights, suggesting mild, subacute attacks. In severe cases, systemic symptoms such as nausea, vomiting, sweating and bradycardia may be the main presenting features. The patient is generally unwell and visual acuity may, although rarely, be reduced to hand movements.

Eye examination of a patient with primary angle closure shows ciliary injection due to congestion of limbal blood vessels. The cornea is usually cloudy due to oedema, and this may limit visualisation of the anterior segment. The pupil is mid-dilated and nonreactive (Figure 3). The orbit is hard on digital palpation, compared with the other eye, and tonometry demonstrates raised IOP, which may be as high as 70 mmHg in some instances.

Examination of the fellow eye is crucial, and usually reveals a shallow anterior chamber, with gonioscopy demonstrating narrow angles.

Differential diagnosis

The clinical features of primary angle closure in the acute stage are generally classic, and the condition should be at the forefront of the list of differential diagnoses because it is sight-threatening and usually reversible. If some features are not typical of primary angle closure, the following differential diagnoses should be considered:

- other causes of acutely raised IOP – traumatic glaucoma, pigmentary glaucoma and inflammatory glaucomas
- other causes of acute, severe ocular pain, red eye and visual loss – keratitis, anterior uveitis, scleritis, endophthalmitis and optic neuritis
- other causes of headache – migraine and tension headache.

Primary angle closure is a clinical diagnosis based on a history of ocular pain, blurring of vision, haloes and systemic symptoms (mainly vagal) and the findings of ocular examination. In expert hands, gonioscopy can determine if trabecular blockage is present and gonioscopy itself may break an acute attack. During indentation gonioscopy, the central corneal curvature is altered, pushing aqueous peripherally into the angle recess, and possibly opening the appositionally closed angles.

No definitive laboratory tests or imaging studies are available for angle closure. Ultrasound biomicroscopy and new techniques such as anterior segment optical coherence tomography and use of the scanning peripheral anterior chamber depth analyser may play a role in the screening and management of angle closure.[6]

Management

With prompt diagnosis and treatment, the prognosis for a patient with primary angle closure is excellent. Delays can result in progression to glaucoma, and possibly the devastating consequence of permanent loss of vision.

The early management of primary angle closure is aimed at reducing IOP (and therefore pain) and breaking the acute attack.[7] Once the attack is broken, definitive treatment is performed, usually within 24 to 48 hours, to prevent further attacks.

It is important that patients are aware that first-degree relatives are at increased risk of the condition. Prophylactic laser iridotomy should be considered for relatives found to have shallow anterior chambers.

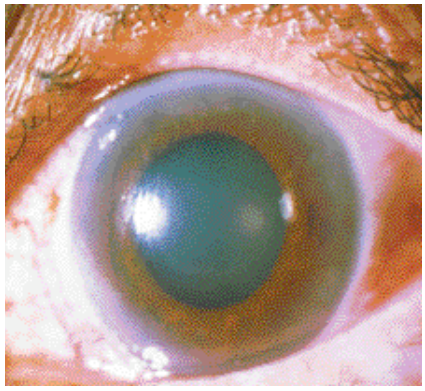

Figure 3. Mid-dilated pupil and cloudy cornea in primary angle closure.

Early management

Medical management

The principles of medical management of a patient with an acute attack of primary angle closure are outlined below. Patients should be assessed hourly until the acute attack is broken and then remain under close observation until surgical treatment.

- Urgent referral, day or night, for emergency care – immediate treatment is needed to reduce IOP and save the patient's vision. In the pre-hospital care, the patient should be in the supine position if possible, and the affected eye should not be covered with an eye patch or blindfold (darkness would maintain the pupillary dilation and help perpetuate the attack).
- Posturing of the patient in a supine position – this may allow the lens to fall posteriorly, away from the iris, and may reduce pupillary block.
- To lower IOP, systemic acetazolamide should be given as soon as possible, preferably intravenously (250 to 500 mg over 10 minutes) but otherwise orally (two 250 mg tablets in one dose). Sulfonamide allergy and sickle cell disease or trait should be checked for when using acetazolamide, and urea

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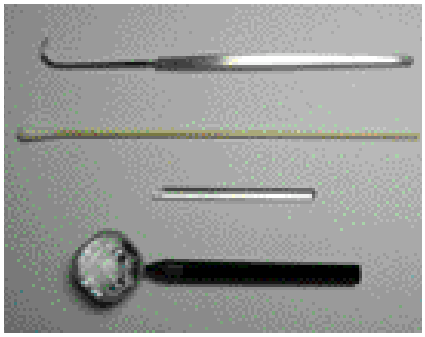

Figure 4. Corneal indenting instruments (from top to bottom): squint hook, cotton applicator, glass rod and gonioscope.

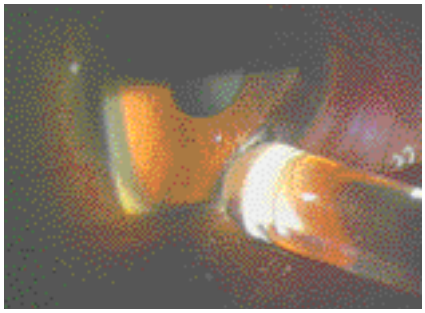

Figure 5. Method of corneal indentation.

and electrolytes levels should be monitored. Acetazolamide lowers IOP by reducing the production of aqueous.

- Use of eye drops to lower IOP – eye drops used to lower IOP in cases of open angle glaucoma are often used to help lower IOP in cases of angle closure (off-label use). Timolol, apraclonidine, brimonidine and latanoprost are usually readily available in eye clinics. Topical beta blockers such as timolol 0.5% lower IOP by reducing aqueous production (caution should be taken using timolol in patients with COPD or asthma). The topical alpha agonists apraclonidine 0.5% and brimonidine 2 mg/mL lower IOP by both reducing aqueous production and increasing its drainage. The prostaglandin analogue latanoprost lowers IOP by increasing the drainage of aqueous from the eye.
- Use of a miotic – topical pilocarpine can be used to induce pupillary miosis and reverse any pupillary block component (off-label use). Topical pilocarpine 2% is usually stocked and available; pilocarpine 4% can be used. Pilocarpine may be ineffective in inducing miosis in the initial stages of an acute attack because the IOP may be sufficiently high to cause pressure-induced ischaemic paralysis of the iris. Once iris ischaemia is relieved, which is usually when the IOP drops below 50 mmHg, it is usually effective.
- To reduce the associated ocular inflammation, topical corticosteroids (e.g. prednisolone acetate, preferably 1%) can be used.
- Systemic analgesics may be required, as may also antiemetics (vomiting can drastically increase IOP).
- If the IOP does not reduce with the above treatment, the topical medications may be repeated and a systemic hyperosmotic may be added (mannitol 1 to 2 g/kg intravenously over a 45-minute period – a 500 mL bag of mannitol 20% contains 100 g of mannitol; it is essential to consider the patient's cardiovascular and electrolyte status before use). Oral glycerol may be considered in patients who do not have diabetes.

Corneal indentation

Corneal indentation has been historically described and recently validated as a rapid and effective method in the early management of primary angle closure.[8] This is a simple and noninvasive technique of compressing the inferior cornea using a squint hook, cotton applicator (or the proprietary cotton bud), glass rod or gonioscope (Figures 4 and 5).

The process of applying pressure to the cornea forces the aqueous into the peripheral anterior chamber and opens the drainage angle. A response is usually seen in three or four cycles of 30 seconds of pressure on and 30 seconds of pressure off the cornea.

Corneal indentation can be performed easily as first-line management in rural settings that have limited access to resources. Not infrequently, corneal indentation without medical treatment can break the acute attack.

Anterior chamber paracentesis

Anterior chamber paracentesis has been proposed in the immediate management of primary angle closure because it can dramatically lower IOP and relieve pain.[9] The procedure is, however, invasive and has the potential for iridolenticular trauma, further shallowing of the anterior chamber, iridocorneal touch, corneal endothelial damage and even intraocular infection. It is, therefore, not the procedure of choice, especially in uncooperative patients.

Surgical treatment

Laser peripheral iridotomy

The procedure of laser peripheral iridotomy involves creating an opening in the superior peripheral iris using an Nd:YAG laser (Figure 6). This has the effect of establishing an alternate communication between the posterior and anterior chambers, thus eliminating any pressure differential and thereby preventing further pupillary block. It is usually carried out once the corneal oedema has cleared enough to provide a good view of the iris.

Laser peripheral iridotomy is usually successful in cases where at least 180 degrees of angle is not closed by peripheral anterior synechiae (adhesions of the peripheral iris to the cornea in the anterior chamber angle). Successful laser peripheral iridotomy is creation of a full thickness opening in the peripheral iris; in some instances, the opening may not be full thickness due to iris oedema or poor visibility (corneal haze). If the angle has already closed completely due to synechiae, creation of peripheral iridotomy may not be sufficient.

Prophylactic peripheral iridotomy should be performed in the fellow eye as the risk of acute angle closure if not

treated is 40 to 80% in five to 10 years.

The procedure is relatively safe but has a few significant complications, the most important being a post-laser spike of IOP (which can be controlled with adequate premedication), iris haemorrhage and corneal burn. In the long term, there is a small risk of cataract formation.

Lensectomy

Lensectomy is known to deepen the anterior chamber, and phacoemulsification with intraocular lens implantation has been advocated for treatment of persistent angle closure and uncontrolled IOP.[10,11] Although it works very effectively, it may not be ideal as an immediate option in eyes with very high IOP.

Chronic angle closure glaucoma

As mentioned earlier, chronic angle closure glaucoma refers to progressive permanent closing of the angle that results in raised IOP and subsequent optic disc and visual field changes.

Usually a prolonged acute angle closure attack or intermittent subacute attacks lead to progressive formation of peripheral anterior synechiae.

In dark irides, however, a different mechanism of angle closure has been recognised where there is bilateral, progressive, circumferential synechial closure of the trabecular meshwork resulting in a gradual rise in IOP. This has been described previously as creeping angle closure glaucoma. It is prevalent in Asians and is being increasingly recognised as a major cause of visual loss.

Other less common types of chronic angle closure glaucoma are:

- combined mechanism glaucoma – combined open angle and angle closure glaucoma
- mixed mechanism – angle closure due to more than one mechanism
- plateau iris
- miotic-induced – long-term use of miotics resulting in pupillary block.

Laser peripheral iridotomy must be attempted in all cases of chronic angle closure glaucoma because the procedure may open the portion of the angle not closed by synechiae. Argon laser peripheral iridoplasty or trabeculoplasty may be attempted in selected cases. In addition, some patients may need topical antiglaucoma drugs, goniosynechiolysis (surgical stripping of peripheral anterior synechiae) or trabeculectomy.

Conclusion

The advent of indentation gonioscopy and laser peripheral iridotomy has led to better understanding of the mechanisms of primary angle closure and considerable advances in its management. Recognition of the symptoms of acute angle closure and urgent referral for treatment is essential to prevent permanent visual loss. Prophylactic laser peripheral iridotomy has emerged as a quick, effective and relatively safe treatment in preventing acute angle closure in eyes at risk.

With the growing Asian population, Australia may see an increasing trend of primary angle closure in the near future and hence there is a greater need for awareness, education and research in this direction. MT

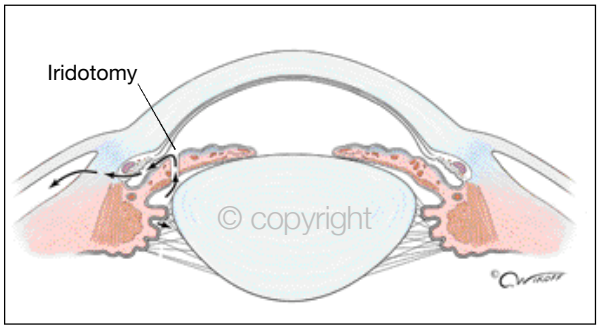

Figure 6. Laser peripheral iridotomy. In an eye with a narrow angle, creating an opening in the superior peripheral iris allows the aqueous to circulate, thereby eliminating any pressure differential and preventing further occlusion of the angle.

References

1. Quigley HA. Number of people with glaucoma worldwide. Br J Ophthalmol 1996; 80: 389-393.
2. Quigley HA, Broman AT. The number of people with glaucoma worldwide in 2010 and 2020. Br J Ophthalmol 2006; 90: 262-267.
3. Foster PJ. Advances in understanding primary angle-closure as a cause of glaucomatous optic neuropathy. Community Eye Health 2001; 14(39): 37-39.
4. Pavlin CJ, Harasiewicz K, Foster FS. Ultrasound biomicroscopy of anterior segment structures in normal and glaucomatous eyes. Am J Ophthalmol 1992; 113: 381-389.
5. Lachkar Y, Bouassida W. Drug-induced acute angle closure glaucoma. Curr Opin Ophthalmol 2007; 18: 129-133.
6. Tsai CJ, Tello C, Ritch R. Angle-closure glaucoma update. Focal points. Clinical modules for ophthalmologists, American Academy of Ophthalmology; 2009; 27(6).
7. Ehlers JP, Shah CP, Fenton GL, Hoskins EN, Shelsta HN. The Wills eye manual. 5th ed. Philadelphia: Lippincott Williams and Wilkins; 2008.
8. Masselos K, Bank A, Francis IC, Stapleton F. Corneal indentation in the early management of acute angle closure. Ophthalmology 2009; 116: 25-29.
9. Lam DS, Chua JK, Tham CC, Lai JS. Efficacy and safety of immediate anterior chamber paracentesis in the treatment of acute angle-closure glaucoma: a pilot study. Ophthalmology 2002; 109: 64-70.
10. Hayashi K, Hayashi H, Nakao F, Hayashi F. Changes in anterior chamber angle width and depth after intraocular lens implantation in eyes with glaucoma. Ophthalmology 2000; 107: 698-703.
11. Roberts TV, Francis IC, Lertusumitkul S, Kappagoda MB, Coroneo MT. Primary phacoemulsification for uncontrolled angle-closure glaucoma. J Cataract Refract Surg 2000; 26: 1012-1016.

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