Angle-closure glaucoma

What is angle-closure glaucoma (ACG)?

Angle-closure glaucoma (ACG) is a condition of of acutely raised intraocular pressure (IOP) associated with a physically obstructed anterior chamber angle. It may be chronic or, in around 10% of cases, acute. Symptoms of acute angle-closure glaucoma (AACG) include severe ocular pain with decreased acuity - immediate treatment is needed to prevent permanent visual loss [1] [2] .

Angle-closure glaucoma is a major cause of severe sight impairment worldwide with a particularly high incidence in some Inuit and Asian populations [3] . There is a familial tendency and age and hyperopia increase the risk. It is divided into primary and secondary types and the distinction is important as the treatments vary. Both primary and secondary types may cause acute painful attacks or chronic asymptomatic disease. Early diagnosis and management stabilise disease and minimise visual loss.

Understanding the anatomy

In the normal eye, aqueous humour is produced by the ciliary body behind the iris and flows through the pupil to drain into the trabecular meshwork which lies around the circumference of the angle between the iris and the cornea. This junction of the iris and cornea at the periphery of the anterior chamber is the anterior chamber angle.

Occasionally, the iris can become apposed to the trabecular meshwork and so block off the aqueous drainage. This results in a rise in IOP which causes a number of symptoms and signs, depending on the type of angle closure.

Definitions: angle closure with or without glaucoma

Primary angle-closure (PAC), primary angle-closure glaucoma (PACG), acute angle-closure (AAC) and acute angle-closure glaucoma (AACG) are all terms that tend to be used interchangeably for the situation.

The angle may close acutely (AAC/AACG), intermittently or insidiously (PAC/PACG) and the condition may be primary or secondary in each case. Intermittent angle closure occurs if the episode of pupillary block resolves spontaneously in hours, usually after sleeping supine. The term glaucoma should only be applied if there are accompanying disc changes characteristic of glaucoma.

In primary angle-closure, the underlying mechanism is pupillary block, whereas in the secondary form other underlying issues push or pull the iris forward to contact the trabecular meshwork.

Primary angle-closure glaucoma

- Angle closure can arise as a consequence of the anatomy of the eye: some people's angles are naturally very narrow which makes the angle more vulnerable to blocking off. Severe hypermetropes fall into this category. In this case the condition is known as primary AAC.
- Narrow angles are not present in young people. The lens grows as we age and in some people this pushes the iris forwards, narrowing the angle. Risk factors include family history, advanced age and Asian or Inuit ethnicity.
- Other susceptible patients include those with a thin iris, a thick lens and a shorter axial length of the eyeball (front to back). Ocular shape is to some extent hereditary, so there may be a familial tendency.
- Pupillary dilation with topical mydriatics can push the iris into the angle and precipitate AAC in anyone with narrow angles. Some systemic drugs which dilate the eye, such as alpha-adrenergic agonists used in urinary incontinence, can produce the same effect.

Secondary angle-closure glaucoma

- Angle closure can also occur as a result of forces exerted on the iris either anteriorly (eg, secondary to peripheral anterior synechiae pulling the iris up) or posteriorly (eg, the lens bulging forwards as a result of swelling).
- Secondary closure can also arise through blockage, as a result of the trabecular meshwork being blocked by matter such as blood (from a hyphaema), blood vessels (from poorly controlled advanced diabetic eye disease) or proteins (as seen in hypertensive uveitis).

Stages of angle-closure glaucoma[1]

Five stages are classically described in the progression of primary angle-closure glaucoma:

- Latent - there are anatomical predispositions present.
- Subacute - there may be mild symptomatic episodes which suggest incomplete angle closure and which have spontaneously resolved.
- Acute - discussed here, and the most likely to present in primary care.
- Chronic - discussed in the final section of this article.
- Absolute - the end stage of untreated disease (an irreversibly severely sight-impaired eye).

AAC is an emergency - prompt diagnosis and treatment are essential to save sight and prophylactic measures will be needed to prevent an attack in the fellow eye. A brief account of chronic angle-closure glaucoma (CACG) is provided at the end of this article.

Epidemiology of angle-closure glaucoma[3]

- The prevalence of primary angle-closure glaucoma in those aged 40 years or more is 4 in 1,000 with the prevalence rising with increasing age[4] . It most frequently occurs in the 6th to 7th decade of life.
- Accounting for ageing population structures, cases were predicted in 2012 to increase by 19% in the UK within the subsequent decade[4] .
- It is more common among the Southeast Asian population, Chinese individuals and Inuits. It is rare among black people.

- Females are affected more commonly than males (4:1).
- First-degree relatives are at greater risk (eye shape is often inherited).

Presentation of acute-angle glaucoma[5] [6]

History

- **Pain** - this is severe and rapidly progressive. It may be confined to the eye but more usually spreads around the orbit with an associated frontal or generalised headache. Some individuals - particularly black people (in whom this condition is uncommon) - have surprisingly little pain; however, this is not the norm.
- **Blurred vision** (rapidly progressing to visual loss).
- **Coloured haloes** around lights. Transient blurring of vision and haloes around light, suggest mild, subacute attacks.
- **Systemic malaise** - nausea and vomiting are common and may be the main presenting feature in some patients - particularly where obtaining a history is a problem (eg, the demented elderly patient).
- **Attack precipitants** - a common aetiology of acute angle-closure glaucoma (75% of occasions) is pupillary block[7] . The mid-dilated pupil snags on to the lens, so causing a build-up of aqueous beneath it which further pushes the iris forwards, so eventually blocking off the trabecular meshwork. It is therefore not uncommon to hear that the attack came on in situations where there was mid-dilation of the pupil - eg, during a moment of stress or excitement, whilst watching TV in dim lighting conditions or after topical mydriatics or systemic anticholinergics. The same situation can occasionally occur in older people after general anaesthetic - the lens can accumulate fluid and, in the predisposed individual, bulge forwards so also causing a pupillary block.
- **Background** - a previous history of acute angle-closure glaucoma is infrequent. These attacks are usually the first indication of such a problem.

Examination

- The patient is often generally unwell.

- Examination shows a red eye in the form of a ciliary flush: the redness is more marked around the periphery of the cornea. There is a hazy cornea and a non-reactive (or minimally reactive) mid-dilated pupil.
- Palpation of the globe will reveal it to be hard. The IOP will be raised (normal range from 10-21 mm Hg - see the separate Examination of the Eye article).
- Slit-lamp findings include shallow anterior chambers in both eyes, closed iridocorneal angles and corneal epithelial oedema.
- If the acute episode has been precipitated by a secondary cause, there may be evidence of this on examination - eg, peripheral anterior synechiae associated with uveitis. If the acute rise in IOP has come about on top of a secondary open-angle glaucoma, there may be evidence of trabecular meshwork obstruction due to clogging by abnormal deposits.
- The acute attack is usually unilateral; however, the predisposing factors are bilateral and long-term management will be to both eyes.

Diagnosis of acute angle-closure glaucoma

Diagnosis of acute angle-closure glaucoma is based on at least two of these symptoms:

- Ocular pain.
- Nausea/vomiting.
- History of intermittent blurring of vision with haloes **and** at least three of the following signs:
- - IOP greater than 21 mm Hg (clinically this can mean a stony hard pupil).
  - Conjunctival injection.
  - Corneal epithelial oedema.
  - Mid-dilated non-reactive pupil.
  - Shallow chamber in the presence of occlusion.

Differential diagnosis

The symptoms and signs are fairly classic. Acute angle-closure glaucoma is an emergency. It should be at the forefront of the list of differentials in all cases of eye pain affecting vision, as it is sight-threatening and usually reversible if caught in time. Differential diagnoses include:

- Other causes of acutely raised IOP:
    - Traumatic glaucoma.
    - Pigmentary glaucoma.
    - Glaucomatocyclitic crisis (Posner-Schlossman syndrome).
- Other causes of acute, severe ocular pain associated with visual loss:
    - Corneal disorder.
    - Anterior uveitis.
    - Scleritis.
    - Endophthalmitis.
    - Optic neuritis.
- Other causes of red eye:
    - Conjunctival causes (eg, keratoconjunctivitis).
    - Corneal causes (eg, keratitis).
    - Other causes (eg, trauma).
- Other causes of general systemic malaise - eg, viral illnesses, connective tissue disorders, infectious disease.

Angle-closure glaucoma treatment and management

Refer immediately - day or night. Patients need urgent treatment in order to save sight.

Medical [8] [9]

- Initial medical treatment typically involves all topical glaucoma medications that are not contra-indicated in the patient, together with intravenous acetazolamide. Patients lie supine.
- Topical agents include:
    - Beta-blockers - eg, timolol, cautioned in asthma.
    - Steroids - prednisolone 15 every 15 minutes for an hour, then hourly.
    - Pilocarpine 1-2% (in patients with their natural lens).
    - Phenylephrine 2.5% (in patients who do not have their own lens).
- Acetazolamide is given intravenously (500 mg over 10 minutes) and a further 250 mg slow-release tablet after one hour - check for sulfonamide allergy and sickle cell disease/trait. U&E should be monitored.
- If there is no response, systemic hyperosmotics (eg, glycerol PO 1 gm/kg of 50% solution in lemon juice or mannitol 20% solution IV 1-1.5 gm/kg) may be added.
- Offer systemic analgesia ± antiemetics.

This should tide the patient over until they are able to be seen by a duty ophthalmologist who will assess the situation at short intervals until the acute attack is broken. These treatments may be repeated depending on the IOP response and a combination of these medications will be given to the patient on discharge. The patient will remain under close observation (eg, daily clinic reviews or as an inpatient). Subsequent treatment is aimed at the underlying cause.

Surgical[3]

- **Peripheral iridectomy (PI)** - this refers toa hole being made in the iris by removing a full-thickness piece from the periphery. The procedure should be as peripheral as possible and covered by the eyelid to avoid monocular diplopia through this second hole in the pupil. It is usually performed using a laser. It provides a free-flow transit passage for the aqueous. Both eyes are treated, as the fellow eye will be predisposed to an AAC attack too [10] . This procedure can usually be carried out within a week of the acute attack, once corneal oedema has cleared enough to allow a good view of the iris.
- **Surgical iridectomy** - this is carried out where laser treatment is not possible. It is a less favoured option, as it is more invasive and therefore more prone to complications.
- **Lensectomy** - one of the few situations where cataract surgery is performed on an urgent basis is when the cataractous lens has swollen to precipitate an attack of AAC. The lens is extracted at the earliest opportunity. Beyond this particular situation there is some debate as to whether a lensectomy should be routinely performed; it is not the routine practice in this country [11] .

Other treatment considerations

Breaking the acute attack medically, and subsequent PIs, is normally sufficient to manage acute primary angle-closure glaucoma. Where glaucoma is secondary to other factors, these will need addressing in due course.

Complications

These include permanent loss of vision, repetition of the acute attack, attack in the fellow eye and central retinal artery or vein occlusion.

Prognosis

In an uncomplicated case of acute angle-closure glaucoma, the outlook is excellent. Patients can expect full recovery if the diagnosis and treatment are prompt.

Delay can lead to complications. Comorbid pathology will increase the risk of complications (especially if additional treatment is needed) and result in a more guarded prognosis.

Prevention

The first attack usually comes without warning, but prophylactic PIs usually prevent further problems. Patients should be informed about the increased risk to first-degree relatives: if they are found to have shallow anterior chambers, they can be offered prophylactic PIs.

Chronic angle-closure glaucoma

Chronic angle-closure glaucoma (COAG) refers to an insidious, progressive closing off of the trabecular meshwork, resulting in scarring and a gradual rise in IOP. This can arise due to a very gradual thickening of the crystalline lens associated with a predisposing anatomy. COAG is a bilateral but usually asymmetric condition, affecting women more commonly, particularly hypermetropes.

Just as in POAG, patients are usually asymptomatic unless the condition is very advanced, in which case there may be decreased acuity or peripheral visual field loss. Occasionally, in the advanced stages, there may be some redness and ocular discomfort but this is not comparable to the pain of acute angle-closure glaucoma.

Once COAG has been diagnosed, the patient should undergo prompt PIs in both eyes. Some patients need additional medical therapy with the topical glaucoma drugs used for POAG. Compliance can be an issue and patient education and eye clinic follow-up are essential.

Dr Mary Lowth is an author or the original author of this leaflet.

Further reading

- Assessing fitness to drive: guide for medical professionals; Driver and Vehicle Licensing Agency

Authored by:	Peer Reviewed by: Dr Hayley Willacy, FRCGP	
Originally Published: 20/11/2023	Next review date: 11/10/2021	Document ID: doc_6929

View this article online at: patient.info/doctor/angle-closure-glaucoma

Discuss Angle-closure glaucoma and find more trusted resources at Patient.