

Acute Angle closure glaucoma (AACG): an important differential diagnosis for acute severe headache

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Key Learning Points

- Always consider Acute Angle Closure Glaucoma (AACG) when acute headache is associated with unilateral visual disturbance
- AACG is an ophthalmologic emergency and early diagnosis and treatment is essential to prevent optic nerve damage and visual loss

Abstract

Acute headache is a common presenting symptom in the acute medical unit. We present a case of Acute Angle Closure Glaucoma (AACG) presenting with acute severe headache. It highlights the importance of remembering this ophthalmologic emergency and reminds us of its clinical presentation. The rapid assessment and diagnosis of AACG allowed prompt treatment and likely prevented the patient from losing her vision.

Keywords

Acute, Angle, Closure, Glaucoma, Headache

Case History

An 83-year old female was referred by her general practitioner (GP) to the acute medical unit (AMU) with a two-day history of severe right-sided headache and vomiting. She had sustained a mechanical fall three days prior to admission, falling onto her right hand side and banging her head without loss of consciousness. The following day, she had developed a severe right-sided headache of acute onset, associated with vomiting. Her right eye was also painful and the vision in her right eye was blurred.

On arrival to the AMU, she was afebrile with a blood pressure 140/80, pulse 100 regular, oxygen saturation 95% in air, respiratory rate 16/min. Cardiovascular, respiratory and gastrointestinal examination were normal. Her right eye appeared red and the right pupil was dilated and unreactive, with a partial ptosis but normal eye movements. Visual acuity in her right eye was significantly reduced with ability to see hand movements only. Fundoscopic examination was limited and the right fundus was not visible. The remainder of the examination was within normal limits.

An urgent CT scan revealed no evidence of an acute intracranial bleed. In view of the visual loss, an urgent ophthalmologic opinion was sought. Slit-lamp examination of the eyes revealed a mid-dilated pupil with extensive corneal oedema (see Figure 1). The intraocular pressure (IOP) in the right eye was measured at 56mmHg (normal range 10-21mmHg),

confirming the diagnosis of Acute Angle Closure Glaucoma (AACG). Intravenous and oral acetazolamide (500mg) were administered to reduce

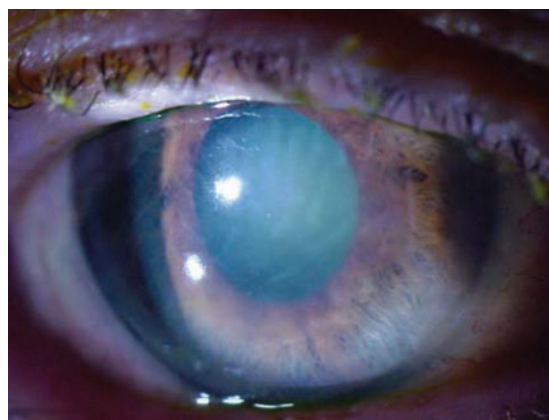


Figure 1. Slit lamp photograph of the right eye showing an injected sclera with mid-dilated, oval pupil and corneal oedema. (Compare to figure 2 to appreciate difference in corneal clarity).

intra-ocular pressure. Topical β -blockers (guttae timolol 0.25%, one drop, BD), prostaglandin analogues (latanoprost 50 micrograms/ml, one drop, nocte) and carbonic anhydrase inhibitors (brinzolamide 10 mg/ml, one drop, TDS) were administered to the right eye and topical miotics (pilocarpine 2%, one drop, QDS) were given to both eyes. The intraocular pressure dropped to 10mmHg by the next morning and her pain completely resolved. She was discharged from hospital the following day. Her vision initially remained poor due to persistent corneal oedema, but this settled after a week, with a subjective and objective corresponding improvement in vision. She went on to have bilateral YAG laser peripheral iridotomies and is making a good recovery.

Discussion

Acute headache is a common presentation to the AMU. In most cases, the primary diagnosis for exclusion is subarachnoid haemorrhage (SAH), requiring investigation with CT brain scan followed

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Figure 2. Slit lamp photograph of the left eye showing constricted pupil and clear pupil and iris detail due to corneal clarity.

by lumbar puncture if this is normal. The diagnosis of SAH is frequently excluded allowing the patient to be discharged; however it should be remembered that other potentially serious causes of acute headache require consideration following normal initial investigation. Furthermore, the common practice of delaying lumbar puncture for 12 hours after onset of headache may lead to delays in diagnosis if the history is not carefully evaluated at presentation and alternative diagnoses are not considered at that time. Early diagnosis is essential as prolonged elevated IOP will lead to non-reversible glaucomatous optic neuropathy and permanent visual loss.

In this case, the combination of headache with visual loss led to the prompt involvement of the on-call ophthalmological team when the CT scan was found to be normal. The finding of a red eye with pupillary dilatation was also suggestive of a primary ophthalmological cause. Other important symptoms exhibited by our patient include nausea and vomiting, although these can also be features of intracranial causes of headache. Headache, visual loss and pupillary dilatation are recognised features of 3rd cranial nerve palsy due to posterior (or less commonly anterior) communicating artery aneurysm, although the normal eye movements in this case helped to exclude this diagnosis. The other differentiating features include a normal consensual light reflex in the fellow eye (provided this is healthy) and a cloudy cornea, identified as poor clarity of the iris compared to the other eye (see Figure 2). The headache in AACG is typically severe and is usually, but not exclusively, felt in the brow above the affected eye. Visual loss is initially due to the corneal oedema. Ptosis is not usually a presenting feature of AACG, however a painful eye will usually have some element of mild lid swelling or drooping and may have accounted for these findings in this case.

References

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Hypermetropes (long-sighted individuals) – they generally have smaller eyes and narrower angles. This can be checked by looking at their glasses. If objects are magnified through the lenses they are hypermetropic.

Older age groups

Anatomy – narrow angles, shallow anterior chambers, shorter axial eye length, lens positioning

Cataracts – reduce available drainage space in the anterior chamber

Family history of angle closure glaucoma

Female Sex - 3:1 female: male preponderance in Caucasians

Chinese & S.E Asian ethnicity

Table 1. Patient groups at risk of developing ACG.

AACG is a term, by strict definition, reserved for an episode of angle closure which has led to optic nerve damage and associated visual field loss. Primary angle closure (PAC), which results in an acute episode of elevated IOP, will not always lead to glaucoma, particularly if managed in timely fashion. Most literature alludes to a single episode of high pressure as AACG, even without optic nerve damage.¹

PAC occurs due to obstruction of the aqueous outflow system of the eye. The trabecular meshwork through which aqueous drains is located in the “angle” formed between cornea and iris and hence the term “angle closure” in anatomically predisposed eyes. Certain patient groups are particularly at risk as outlined in Table 1.

The prevalence of ACG was estimated as 15.7million worldwide in 2010, Europe contributing 1.4million (8.7%) of the total.² Crude annual incidence of acute primary angle closure in the over-45-year-old population in Scotland has been recently estimated at 3.6 per 100 000.³

Trauma, as in our case, is a recognised cause of AACG in susceptible patients.^{4, 5} Other precipitating factors include dimming light, rapid correction of hyperglycaemia, and topical or systemic medications that cause mydriasis.⁶

Definitive treatment for AACG is laser peripheral iridotomy. Medical therapy aims to prepare the eye for this, by reducing the production of aqueous fluid, lowering the IOP and constricting the pupil. As in this case, oral and intravenous carbonic anhydrase inhibitors (e.g. acetazolamide) are administered to reduce IOP rapidly, followed by topical carbonic anhydrase inhibitors, beta blockers, alpha adrenergic agonists and prostaglandin analogues. Topical miotics constrict the pupil and allow increased outflow of the aqueous.

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