"Is this simply a headache?"

DALHOUSIE UNIVERSITY Inspiring Minds

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Dorothy's case

Dorothy, 62, presents to the ED with a 2 hour history of sudden onset headache. The symptoms started as she was reading a book in bed.

She describes her pain as becoming progressively worse. She reveals it has deep and nagging quality which radiates from behind her left eye to her forehead.

She says that she has felt nauseous since the onset of her symptoms and has vomited once.

History

Dorothy's medical history consists of migraines, osteoporosis and hypertension. She is farsighted and wears glasses.

Examination

Her vital signs show mild tachycardia.

On inspection, she is sweating profusely and has increased lacrimation of the left eye. On specific questioning, she reports blurred vision and haloes around lights.

Further examination reveals left conjunctival hyperemia with limbal prominence, a fixed oval-shaped and mid-dilated pupil and left corneal and palpebral edema.

Dorothy's visual acuity is 20/100 on the left and 20/20 on the right.

Ophthalmoscopy suggests diffuse left papilledema.

Tonometry reveals an elevated intraocular pressure (IOP) of 67 mmHg in the left eye.

For Dorothy's diagnosis, look to page 4.

Questions & Answers

1. What is the most likely diagnosis?

Dorothy is likely suffering from acute angle closure glaucoma (AACG). The major characteristic feature of AACG is an occlusion of the angle of the anterior chamber of the eye. Aqueous humour secreted by the ciliary bodies into the posterior chamber normally drains into the anterior chamber and, from there, through the trabecular meshwork and scleral venous sinus (Canal of Schlemm) (Figure 1). If these are blocked by a displaced iris, the aqueous accumulates in the posterior and anterior chambers instead, increasing their pressures (Figure 2). Intraocular pressure (IOP) quickly increases, ultimately damaging structures such as the:

- Lens
- Iris
- Corneal endothelium
- Optic nerve
- Retinal ganglia

2. What causes AACG?

Pupillary block

Contact occurs between the iris and lens such that aqueous cannot drain from the posterior to anterior chambers. This forces the iris to bulge anteriorly, which occludes the anterior chamber angle through which the aqueous must drain.

Narrow anterior chamber angle

Mydriasis in eyes with shallow anterior chambers can cause the peripheral iris to block the trabecular meshwork and also bow inward to cause papillary block.

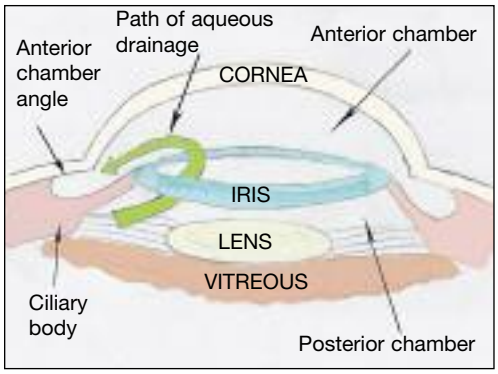

Figure 1. Normal aqueous drainage.

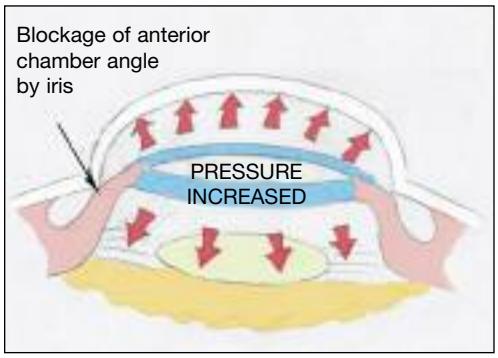

Figure 2. Acute angle closure glaucoma.

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Plateau iris

This is an anatomic abnormality involving an unusually flat iris that forms a right angle at the interface with the ciliary body. Mydriasis causes occlusion of the trabecular meshwork by this angle.

Neovascularization

Conditions such as diabetes mellitus and central retinal vein occlusion can cause neovascularization that occludes the aqueous drainage conduits.

3. *How does AACG present?*

Patients often experience unilateral blurred vision due to optic nerve damage and severe headache on the temporal side of the affected eye that can radiate towards the forehead. A fixed mid-to-dilated oval-shaped pupil that is not reactive to light is secondary to parasympathetic nerve damage. Corneal edema separates light into its component colours, causing the halo-effect seen by individuals with AACG. Parasympathetic stimulation can cause excessive lacrimation, nausea and vomiting. Sympathetic stimulation can cause hyperhidrosis. Symptoms are acute in onset and are commonly precipitated by dimly lit rooms, emotional stress, or various anticholinergic and sympathomimetic medications.

AACG causes a rapid increase in IOP, which can cause permanent visual defects and blindness if not discovered and treated rapidly. It is imperative for emergency physicians to be aware of AACG when patients present with nonspecific symptoms like headache, nausea and vomiting.

4. *Who is at risk of getting AACG?*

People aged 55 to 70 years are at higher risk due to lens thickening causing anterior iris displacement. Women, farsighted people, Inuit and East Asians are at increased risk due to their eyes having narrow anterior chamber angles.

Dorothy's diagnosis

A diagnosis of acute angle closure glaucoma is made. Dorothy is started on timolol, acetazolamide, pilocarpine and oral glycerol.

Ophthalmology is consulted to perform a bilateral surgical peripheral iridotomy. Post-operatively, she is discharged and advised to continue taking the same antiglaucoma medications used in the acute setting until the next day, when she should return for follow-up. Prednisone acetate eyedrops are added to her treatment regimen.

Follow-up

A follow-up tonometry reveals an IOP of 20 mmHg in the left eye. Dorothy is taken off of the acute medications and advised to continue applying the steroid for 1 week.

Diabetics have a higher risk of presenting with secondary AACG due to ocular neovascularization. Keep in mind that AACG typically occurs in patients with no previous history of glaucoma.

5. *What investigations are indicated?*

Visual acuity should always be tested. In AACG, visual acuity is reduced in the affected eye.

Ophthalmoscopy will show papilledema in an acute attack. If glaucoma has occurred previously in that eye, the optic disc may appear excavated.

Tonometry is the most indicative test to detect AACG. Normal IOP is 10 mmHg to 21 mmHg. In AACG, the IOP commonly reaches ≥ 50 mmHg.

6. *What is the treatment?*

Swift treatment on presentation is required to prevent further ocular damage. If intervention is late, AACG can result in permanent vision impairment and blindness.

To decrease aqueous production, a ß-blocker, such as timolol, or an α-agonist, such as brimonidine, should be administered simultaneously with the carbonic anhydrase inhibitor acetazolamide. Caution should be taken when giving ß-blockers to patients with reactive airway disease.

Reduction of vitreous volume can be accomplished by administering hyperosmotic agents such as oral glycerol or IV mannitol.

Mimetic agents, such as pilocarpine, can be given to constrict the iris; thus, widen the anterior angle of the eye, facilitating aqueous drainage.

Antiemetics and sedatives may be administered as needed.

A consult to ophthalmology is always warranted for immediate surgical intervention and follow-up. Dx

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