Primary acute congested angle-closure glaucoma: Dr.Sajid Al Assady

Introduction:

Primary angle-closure glaucoma (PACG) is an ophthalmic emergency in which aqueous out flow is obstructed solely as a result of closure of the anterior chamber angle by peripheral iris. It occurs in anatomically predisposed eye and is frequently bilateral. PACG affects approximately 1 in 1000 individual over the age of 40 and its prevalence increase with age. Women are affected more frequently than men in a ratio of 4:1.

Anatomical predisposing factors:

1. A relatively anterior location of iris-lens diaphragm

2. Shallow anterior chamber

3. Narrow entrance to anterior chamber angle.

The proximity of the cornea to peripheral iris enable angle closure to occur more easily than in normal eye.

Risk factors:

Known risk factors include:

- Hyperopia:
- Gender: female have a 3-4 times more risk than male.
- Age: above age of 40
- Relatives: first degree with history of angle closure.
- Race: higher prevalence in Chinese.

In a predisposed individual an acute angle closure glaucoma attack may be precipitated by pharmacological pupil dilatation, or physiological dilatation as in stress or in dark such as in movie theatre.

Symptoms:

Are usually sudden in onset and patient present with:

1. Pain in and around the eye.

2. Redness of the eye.

3. Nausea and vomiting.

4. Blurred vision.

5. A prodromal of rainbow colored halo around light may be reported.

Sign:

Examination of the eye may reveal several of the following signs:

1. High elevated IOP (usually over 50 mmHg).

2. Conjunctival injection “ciliary flush” caused by injection of the limbal and conjunctival blood vessels.

3. Shallow AC.

4. Oval mid-dilated sluggishly reacted or non-reacted pupil.

5. Cells and flare in AC (AC reaction).

6. Gonioscopy reveals closed angle (also should examine contralateral eye).

Treatment:

Initial treatment:

- ***admission***

Is aimed primarily to lowering IOP through systemic medications. That is because when IOP exceed 50 mmHg the iris sphincter usually ischemic and paralyzed, so that the intensive meiotic therapy is seldom effective in pulling the peripheral iris away from the angle.

- **Systemic medications**: with intravenous Acetazolamide (Diamox) 500mg and then oral slow release 250 mg twice daily. Hyperosmotic agent may be necessary if the IOP is extremely high or if acetazolamide is ineffective. In some patients analgesics and antiemetic may required.

- **Topical therapy**: with b-blockers twice daily may be helpful in reducing IOP and topical steroids 4 times daily is useful in decongesting the eye. Once the IOP have been reduced (usually after 30-40 minutes of initial systemic therapy), 2%

Pilocarpine is instilled twice (15 minutes apart) and then 4 times daily.

It's also very important to treat the fellow eye prophylactically with 1% Pilocarpine drops 4 times daily until prophylactic laser **iridotomy** has been performed.

Subsequent treatment:

Subsequent treatment is aimed at re-establishing the communication between the posterior and the anterior chamber by making an opening in the peripheral iris. This can perform by laser called peripheral iridotomy, however the peripheral iridotomy can be successful only if less than 50% of the angle is permanently closed by peripheral anterior synaechiae (synaechial closure), and if more than 50% of the angle is permanently closed a filtration procedure **(Trabeculectomy**) is usually required.

The fellow eye should be treated with prophylactic laser iridotomy.

Primary congenital glaucoma:

Introduction:

Although the primary congenital glaucoma (PCG) is the most common form of congenital glaucoma its only affects 1 in 10000 births, with 65% of patients being boys.

Inheritance: Autosomal recessive with incomplete penetrance.

Clinical features:

The clinical features depend on the age of onset and the level of IOP, both eyes are affected in 75% of the cases, although the severity of involvement frequently asymmetrical. According to the age of onset PCG can be divided in to three types:

A .True congenital glaucoma: accounting about 40% of congenital glaucoma and characterized by IOP elevation during intrauterine life, so that the child is born with ocular enlargement (Buphthalmos).

B. Infantile glaucoma: accounting about 55% in which the disease become manifest after birth but before the second birth day.

C. Juvenile glaucoma: is characterized by elevation of IOP after the age of 2 years but before the age of 16 years, in these cases the clinical manifestation may simulate the POAG.

Examination of patient with true congenital and infantile glaucoma shows the following:

1. Corneal haze: resulting from epithelial edema

2. Buphthalmos: ocular enlargement occurs if the IOP become elevated before the age of 3 years.

3. Breaks in decement membrane.

4. Glaucomatous cupping of optic disc in infant may occur early, although it may regress once the IOP is normalized.

Initial evaluation:

The initial evaluation should be performed under GA using Ketamine anesthesia because other agents may cause false lowering of IOP.

Examination involves ophthalmoscope, measurement of IOP, Gonioscopy and measurement of horizontal and vertical corneal diameters.

Treatment:

Surgery: Goniotomy, Trabeculectomy or combination.

Prognosis: good in about 60% of cases.

Secondary Glaucoma:

There are many secondary causes of glaucoma. These are may of open angle or closed angle variety.

A. Secondary open angle glaucoma:

An elevated IOP is caused by some other ocular or systemic disorders with clogs of the trabecular meshwork.

Examples:

1. Steroid induced glaucoma: secondary open angle glaucoma that is due to topical or systemic corticosteroid use.

2. **Traumatic glaucoma:** hyphaema (blood in anterior chamber) can induce elevation in IOP due to blood blocking trabecular meshwork, also the angle recession follow trauma.

B. Secondary angle closure glaucoma:

Mechanical blockage of aqueous access to the trabecular meshwork can cause by associated ocular disorders.

Examples:

1. Neovascular glaucoma (NVG):

Consist of abnormal blood vessels developing on the surface of the iris (rubeosis) as well as over the trabecular meshwork itself.

Initially the angle is open but, as the fibrovascular tissue consolidates; permanent adhesion form and angle closure glaucoma develops.

NVG is commonly due to underlying retinal ischemia with proliferative diabetic retinopathy and central retinal vein occlusion the most common cause.

2. Uveitis:

Uveitis can cause secondary angle closure if the inflamed iris become entirely stuck to the lens, if extensive, these posterior synaechiae can prevent aqueous from draining through the pupil- a secondary papillary block.

LENS-RELATED GLAUCOMA

Phacolytic glaucoma

Pathogenesis

Phacolytic glaucoma (lens protein glaucoma) is open-angle glaucoma occurring in association with a hypermature cataract. Trabecular obstruction is caused by high molecular weight lens proteins which have leaked through the intact capsule into the aqueous humour.

Diagnosis

1. **Presentation** is with pain; vision is already poor due to cataract.
2. **Slit-lamp biomicroscopy** shows corneal oedema, a hypermature cataract and a deep anterior chamber. The aqueous may manifest floating white particles.
3. **Gonioscopy** shows an open-angle.

Treatment

Once the IOP is controlled medically, the proteinaceous material is flushed out and the cataract removed. Care should be taken not to rupture the zonules when performing anterior capsulotomy.

Phacomorphic glaucoma

Pathogenesis

Phacomorphic glaucoma is an acute secondary angle-closure glaucoma precipitated by an intumescent cataractous lens. Equatorial age-related growth of the lens slackens the suspensory ligament and allows the lens to move anteriorly. Associated anteroposterior growth leads to increased iridolenticular contact and potentiates pupillary block and iris bombé.

Diagnosis

1. **Presentation** is similar to acute PACG with a shallow anterior chamber and dilated pupil; cataract is usually evident.
2. **Examination** of the fellow eye may demonstrate a deep anterior chamber and an open angle, thus making PACG unlikely, although Phacomorphic glaucoma is more likely in eyes with a shorter axial length and shallower anterior chamber.

Treatment

Treatment is initially similar to acute PACG, but miotics are omitted as they tend to increase iris-lens apposition and shift the lens anteriorly. Systemic hyperosmotic agents may be required more commonly than in PACG. Laser iridotomy may be worthwhile but is often not possible (due to corneal oedema or lens-cornea proximity) or ineffective. Definitive treatment consists of early cataract extraction, ideally when the IOP is normal and the eye quiet.