How to manage a patient with glaucoma in Asia

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Introduction
Glaucoma affects nearly 70 million people worldwide, of which nearly half are in Asia. Although more people are affected by primary open-angle glaucoma (POAG) than by primary angle-closure glaucoma (PACG), the latter is more common in Asians and carries a higher burden of morbidity. An estimated 13.6 million people will suffer from PACG in Asia by 2010, of which nearly 3.5 million will be bilaterally blind. Unlike cataract, visual loss from glaucoma is irreversible. The methods for management of POAG are similar to those described in the other sections of this issue, therefore we will concentrate on PACG in this article. Traditional definitions of PACG have emphasised the symptomatic aspect of the disease. However, only 25 per cent of PACG is symptomatic, therefore more modern definitions rely on objective evidence of damage to the trabecular meshwork and optic nerve. A diagnosis of glaucoma means that there is damage to the optic nerve as shown by changes in the optic disc and a characteristic visual field defect. One important distinction is that acute angle closure, where there is a sudden rise in intraocular pressure (IOP) causing pain and blurred vision, is not considered as glaucoma.

As management pathways for PACG are different to that of POAG, accurate detection as well as treatment are important when dealing with glaucoma in Asia.

Population burden of glaucoma
Primary angle-closure glaucoma is a largely asymptomatic disease. There are a large number of patients in the community who have undiagnosed disease. A glaucoma survey in Mongolia showed that 91 per cent of patients were not aware that they had glaucoma, and in a similar survey in Singapore this figure was 21 per cent. This suggests that economic conditions and health service provision are a consideration in the population burden of the disease.

Age and gender are major risk factors for PACG. Angle closure is rare before the age of 40 and more common in females. The main anatomical risk factors are shorter axial length and a shallow anterior chamber depth (ACD). Asian people who present with an acute episode of angle closure have shorter axial lengths compared to those with chronic angle closure, and both these groups have shorter axial lengths than people without angle closure. Although anterior chamber depth is correlated with angle closure in different populations of Asian descent, there is a suggestion that this association differs between different populations.5 There is little information about the natural history of the disease. Current understanding indicates that people at risk of disease develop an anatomically narrow angle (or primary angle-closure suspect, PACS) with no other abnormality. Signs of prolonged appositional closure can appear afterwards, where there is raised IOP or peripheral anterior synechiae (PAS); this is termed primary angle closure (PAC). The final stage is angle closure combined with a glaucomatous optic neuropathy (GON) where there is structural damage to the neuroretinal rim of the optic disc and a reproducible visual field defect. This is classified as PACG.

Recent data from south India describes the natural history of normal subjects and people with narrow drainage angles who were enrolled from a population survey. Among the people with narrow drainage angles (PACS), 22 per cent (95 per cent CI: 9.8, 34.2) had developed synechial (64 per cent) or appositional angle closure (36 per cent) over a period of five years.2 Twenty-eight people with established angle closure at baseline were also examined, and 8 out of 28 (28 per cent, 95 per cent CI: 12, 45) had progressed to PACG over five years.1 In this group, one of nine who had laser peripheral iridotomy (LPI) at baseline had progressed compared to 7 of 19 who refused LPI. Although more information on the natural history and progression of angle closure is needed, in the context of the existing knowledge there is a strong protective effect from peripheral iridectomy.

Primary angle-closure glaucoma is a good candidate for screening; the population undergoes a simple test to detect those at risk or with early disease, who are then subsequently referred for diagnosis and treatment. However, the paucity of data on the natural history of the disease will hamper the understanding of the potential benefits of prophylactic treatment, and we are awaiting results of a randomised controlled trial assessing this strategy.

Treatment
Medical therapy
There is clear evidence that reduction of IOP with medical therapy is effective in preventing the onset of POAG6 and averting the progression of early POAG.3 However, the cost of maintaining therapeutic goals to prevent one case is too high for resource-poor countries. In addition, these trials were performed on western populations and the generalisability of the results is limited, especially when considering calculations such as numbers needed to treat. In patients with angle closure and a patent peripheral iridectomy, medical treatment with latanoprost is effective at reducing IOP even in the presence of synechiae.10

Peripheral iridotomy
Laser or surgical peripheral iridotomy (PI) is the mainstay of treatment for angle closure. There is strong evidence to show the protective effect of PI in the fellow eye in a patient presenting with an acute attack, and this is standard practice worldwide. In units where laser treatment is not available, a surgical PI is as effective as laser PI. The emphasis is that patients with an acute attack should not leave the treatment unit without a PI in both eyes. The presence of a PI, together with medical and other laser treatments, will often be successful even when early glaucomatous damage has occurred to disc and field. In Asian eyes, PI alone may be insufficient for long-term IOP control following acute angle closure; in one study from Singapore 58 per cent of such eyes required additional methods of management, including trabeculectomy.11

In patients who have not suffered symptoms from angle closure, there is no published evidence on the efficacy of prophylactic PI for PACS. However, a longitudinal study from Mongolia showed that when PIs were performed on eyes with PAC or PACG, these eyes were more likely to require further treatment than eyes with the earlier stage of PACS. As a PI can protect a fellow eye from an acute attack, current practice is based on the assumption that it will protect a predisposed individual to a first attack. Therefore, current expert consensus suggests that all eyes with angle closure require a PI to protect them from an acute attack and to eliminate pupil block. This is especially the case in developing countries, where treatment centres can be very far away for the patient.

Laser iridoplasty
Iridoplasty refers to a method where the position of the peripheral iris is altered by applying contraction burns. This pulls the peripheral iris away from the angle structures and can reverse appositional closure. Current expert opinion suggests that if IOP is not promptly controlled by medical means in an acute attack, the patient should be considered for laser iridoplasty. In cases of asymptomatic angle closure due to plateau iris (in which a PI has been performed), iridoplasty can increase the angle width. Limited
Laser treatment for angle closure

Laser treatment is the cornerstone of management of angle closure, and is generally very effective. However, lasers are powerful surgical tools, which, if used incorrectly, can do considerable harm. The following is intended as a guide for experienced users.

**Laser iridotomy**

**Indications**
- in both eyes of patients who suffer an acute episode of angle closure
- patients with asymptomatic narrow angles, established primary angle closure, and those with primary angle closure and early glaucomatous optic neuropathy.

1. **Explain the reasons for carrying out the procedure, what it will feel like, and what to expect afterwards.**
2. **Instil pilocarpine 2% for blue eyes and 4% for brown-eyed patients, and apraclonidine 0.5% (unless contra-indicated) half an hour before, and again five minutes before the procedure.**
3. **Ensure the laser is set to zero defocus.**
4. **Anaesthetise the cornea, and insert a Wise or Abrahams lens.**
5. **If possible, identify an iris crypt (indicating a thin area of iris) in the peripheral iris at, or very close to, 12 o’clock. Take very careful aim on the most peripheral area of iris that can be seen adjacent to the limbal zone. Accurate focussing increases the effectiveness of the laser energy considerably.**

**Argon**

6. **Argon laser pre-treatment is very useful in achieving a laser iridotomy in thick, deeply pigmented irises.**

- **Set the laser to 50 µm spot, 120 mW, 0.05 s, and apply approximately 30 laser shots to the iris in an overlapping rosette pattern to achieve a “beaten copper” appearance on the surface of the iris. This helps to avoid large, adherent bubbles when performing the second phase of laser.**

- **Set the laser to 50 µm spot, 700 mW, 0.1 s and apply approximately 5 to 10 laser shots to the iris in the same area to form a crater in the stroma. Complete the iridotomy with a few, low-power shots of YAG laser.**

**YAG**

9. **Multiple lower energy shots (0.6 to 1.2 µJ) are most effective, and prevent the dispersion of pigment that may cause pressure spikes. The use of argon pre-treatment in dark brown irises is important.**

**Aftercare**

Intraocular pressure should be measured at least one hour after treatment. If IOP is high, the protocol used at Moorfields Eye Hospital is:

- If IOP above 30 mm Hg: 250 mg acetazolamide orally stat.
- 125 mg acetazolamide tds for two days.

If IOP above 40 mm Hg:

- Acetazolamide and topical treatment as appropriate.

A response to therapy must be demonstrated before discharge. All patients then receive prednisolone 1% (Pred Forte) hourly for 24 hours (taking a break through the night), and then four times a day until seen in clinic one week later. All regular medication for glaucoma is continued in both eyes.

**Clinic**

All patients are seen one week later in clinic and re-gonioscoped. Stop steroids, unless there is evidence of continued inflammation. If the IOP is raised and there is anterior segment inflammation, swap to a topical NSAID.