Angle Closure Glaucoma

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The current understanding of glaucoma has undergone a significant change over the last few years. Epidemiological data, newer diagnostic procedures, collaborative planned trials, basic research, better documentation and analysis of clinical data, long term follow up of patients and a better understanding of ocular behavior and newer drugs have all contributed to the current understanding of the glaucomas.

A shift in understanding has logically lead to a change in the approach and strategy in glaucoma management. Raised intraocular pressure (IOP) has been a carlinal sign of glaucoma. The relevance of a raised intraocular pressure has to be understood in its proper perspective. In essence a raised IOP does not always need treatment. The ability to distinguish and decide when to treat and how much to treat is today a more scientific step than in yesteryears.

IOP no longer defines glaucoma. Both pressure dependent and pressure independent factors are responsible for the pathogenesis of the glaucomatous damage. Even though factors other than IOP are involved, IOP is the most important risk factor because it is the only risk factor which we can pharmacomodulate todate. The primary aim in glaucoma management is to preserve visual function. Lowering of IOP is only a secondary goal.

Primary glaucomas: Glaucoma is the second leading cause of blindness worldwide accounting for 67 million sufferers. Primary Open Angle Glaucoma (POAG) is estimated to affect 33 million people worldwide, majority of whom (about 26 million) reside in developing countries. 90-100% of those affected in developing countries are unaware that they have the disease. Visual impairment is also more severe. The estimated risk of blindness (over 12-20 years) from POAG ranges from 14.5% to 27% (unilateral) and from 7-9% (bilateral). With an expected increase in the population and longevity, POAG is likely to become a major cause of ocular morbidity in the developing world.

Prevalence in India

Population based studies: 12 million people in India are affected by glaucoma accounting for 12.8% of the blindness in the country. Early population based studies reported a prevalence of glaucoma between 2% and 13%.

Three population based surveys, with modern techniques have been recently conducted.

Table 1. Comparison of results of Vellore Eye Survey (VES) and Andhra Pradesh Eye Disease Survey (APEDS).

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<th>VES</th>
<th>APEDS</th>
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<tr>
<td>Age</td>
<td>30-60 years</td>
<td>≥ 30 years</td>
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<tr>
<td>POAG (95% CI)</td>
<td>0.41% (0.008-0.81)</td>
<td>1.62% (0.77-2.48)</td>
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<tr>
<td>OHT (95% CI)</td>
<td>3.08% (1.98-4.19)</td>
<td>0.32% (0.10-0.78)</td>
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<tr>
<td>PACG (95% CI)</td>
<td>4.32% (3.01-5.63)</td>
<td>0.71% (0.34-1.31)</td>
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<tr>
<td>Occludable Angles (95% CI)</td>
<td>10.3% (8.9-11.7%)</td>
<td>1.41% (0.74-2.09)</td>
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The Vellore Eye Survey (VES) reported a prevalence of POAG as 0.41%, OHT 3.08% and 4.32% for PACG. Occludable angles accounted for 10.3% in the population.

The Andhra Pradesh Eye Disease Survey (APEDS) reported a prevalence of 1.62% for POAG, 0.32% for
OHT. Primary ACG was 0.71% and occludable angles accounted for 1.41% of the study population. The difference in the prevalence of POAG, PACG and occludable angles in the above studies can be explained by the age groups sampled, definitions of POAG, PACG, occludable angles and also the methodology used. The Aravind Comprehensive Eye Survey (ACES) reported a prevalence of 1.7% for POAG, (95% CI 1.3 - 2.1) and 0.5% PACG (95% CI 0.3 - 0.7). The reported prevalence of glaucoma in the ACES study is higher than that reported by the VES and lower than the APEDS although the CI’s overlap. The VES and APEDS did not perform threshold peimetry on all participants. Another reason could be the difference in the age of the study participants, 40-90 years in the ACES study (VES did not include people more than 60 years of age). However prevalence of POAG even in the ACES study was 0.7% (95% CI 0.5 – 1.0) amongst 40 – 60 years, similar to the VES. The VES criteria of occludable angles (inability to visualize 180 degree of the functional trabecular meshwork) and the use of indentation gonioscopy lead to higher prevalence of PACG and occludable angles. The APEDS used the epidemiological criteria for occludable angles.

Hospital based data from India reported POAG as common as PACG, with 45 to 55% of primary glucomas being PACG. Aphakic glaucoma (37.7%) is the commonest type of secondary glaucoma reported in a hospital setting. Others include lens induced (12.5%), corneal pathology (12.2%), neovascular (9.6%) traumatic (8.4%) and chronic uveitis (8.2%). Steroid induced glaucoma and trauma are common causes for secondary glaucoma amongst young people. A prevalence (95% CI 5.3 – 6.6) of Pseudo exfoliation was 6.0%. The prevalence increased with age and was more in males. The prevalence of glaucoma among subjects with pseudo exfoliation was 7.5%. Pseudo exfoliation was present in 26.7% of these with POAG. A hospital based study in 1968 reported a prevalence of 34% pseudo exfoliation amongst glaucoma patients.

**Primary Angle Closure Glaucoma**

Primary Angle Closure Glaucoma (PACG) has not received the same level of attention as POAG. Amongst other reasons, is the preponderance of POAG in Caucasian eyes and also because gonioscopy has not become a routine in the workup of all glaucoma patients. With damage to the optic nerve becoming the diagnostic hallmark of POAG, the definition for primary angle closure glaucoma has also undergone a change.

**Primary Angle Closure (PAC):** there is a significant obstruction of the functional trabecular meshwork by the peripheral iris, in the absence of a secondary pathology. In **Primary Angle Closure Glaucoma (PACG):** this trabecular obstruction is present with glaucomatous damage to the optic nerve head. In this concept, people suffering from an acute rise in intraocular pressure are not considered to have glaucoma unless there is damage to the optic nerve head. This concept is able to explain why 60-75% of people with an acute symptomatic episode of angle closure, recover without optic disc or visual field damage.

The traditional classification of primary angle closure is based on symptomatology (acute, sub-acute and chronic) has its limitations. Estimating the prevalence of PACG and POAG in South Africa it was shown that people with chronic angle closure (white eyes and clear corneas) had intraocular pressure ’s as high as 72 mmHg. They were unable to demonstrate an association between symptoms and development of visual deficit.[16] Even in East Asia, asymptomatic angle closure is more common. Symptomology of angle closure does not specify the involved mechanism. Hence management strategies cannot be based on symptomology alone. Angle closure is a mechanical process and is best classified by physical signs.

**Primary angle closure glaucoma suspect (occludable angle):** Where on gonioscopy there is appositional contact between the peripheral iris and the posterior trabecular meshwork. For epidemiological studies an angle is considered occludable where more than 270 degrees of the trabecular meshwork cannot be seen.

**Primary angle closure:** An eye with an occludable angle on gonioscopy with peripheral anterior synchiae, will have elevated intraocular pressure, or excessive pigment deposition on the trabecular meshwork. The optic disc and fields are normal. Iris
whorling, stromal atrophy are evidence of an old acute attack of angle closure and represent an ischaemic process. Ocular tissues such as the iris and the ciliary body are sensitive to the ischaemic process. Damage to the optic nerve occurs at high IOP. **Signs of anterior segment ischaemia are suggestive but not pathognomonic of damage to the optic nerve.**

**Primary angle closure glaucoma** is characterised by glaucomatous optic atrophy, corresponding visual field defects with occludable angles on gonioscopy or signs of PAC.

**Epidemiology of angle closure**

**Prevalence:** One of the major factors determining susceptibility to primary angle closure is the ethnic background. Primary angle closure is more common amongst Asians. In people more than 40 years of age the prevalence of primary angle closure (number of cases present at one point in time) ranges from 0.09% in Europeans, 1.4% in East Asian and 2.6% in Alaskan Inuit. Data from India, the VES and APEDS shows a prevalence of 4.32% and 0.71% for PACG. Hospital based data suggests an equal number of people with POAG and PACG.

**Sex and Age:** PAC and PACG tend to be higher in women than men. Incidence (number of cases /100,000 persons / year for population aged 30 years and above) of PAC ranges from 4.7 % in Finland to 15.5% in Singapore. Incidence like prevalence increases with advancing age.

**Anatomical factors predisposing to PAC** include shallow anterior chamber, short axial length of the eye, increased lens thickness, forward position of the lens, and how tightly the iris hugs the lens.

<table>
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<th>Table 2. Anatomical predisposition to angle closure</th>
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<tr>
<td>Narrow anterior chamber angle</td>
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<td>Shallow anterior chamber depth</td>
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<tr>
<td>Short axial length of globe</td>
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<tr>
<td>Small corneal diameter</td>
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<td>Increased thickness of lens</td>
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**Screening for Angle closure:** 'Anatomical characteristics (Table 2), make screening for PAC a viable proposition. The aim of screening is to detect disease at an early, pre-symptomatic phase in order to provide suitable treatment which slows or arrests progression.

Screening tests should be quick and reliable. In a routine clinical practice, screening for PAC would involve assessing all patients with age more than 30 years to determining the potential for angle closure in order to identify those who need a gonioscopic examination. Of the various tests available, two commonly used ones are 1) Flashlight test and 2) vonHerrick's test.

**Flashlight test:** A pen torch is held at the lateral canthus to shine a narrow beam of light across the anterior chamber. A shadow is cast on the nasal aspect of the iris with a shallow anterior chamber by the anteriorly situated iris and lens. Using a half iris shadow based data from India has shown a sensitivity of 45% and specificity of 83% and using a third of the iris shadow 86% sensitivity and 71% specificity. In another clinical practice amongst Caucasian eyes sensitivity and specificity of 89% and 88% have been reported. Incorrect identification was more for eyes with plateau iris configuration.

**Von Herrick’s test** is carried out at the slit lamp. A thin bright beam falls perpendicularly on the most peripheral point of the temporal clear cornea. The optical cross section is viewed at a high magnification (16x or 25x) from the nasal side. A sensitivity and specificity of 62% and 89% for detection of angles judged to be occludable on gonioscopic examination have been reported.

In a clinic based setup where the definitive test (gonioscopy) can be done using both the flashlight test to detect eyes with 1/3 iris in shadow and the limbal based test to detect a limbal chamber depth less than or equal to one quarter of the peripheral corneal thickness, few occludable angles would be overlooked.

**Provocative test**

Depending on the criteria used, occludable angles account for 1.4 –10.3% of the studied population. A small proportion of these individuals are at risk of developing PAC at some point of time. Since a laser iridotomy is a fairly safe and simple procedure that can eliminate this risk, early identification of such patients and treating them prophylactically can help eliminate this risk. Eyes at risk can be subjected to provocative testing – dark room test, prone test, dark room prone test or the mydriatic test.

**Clinical relevance of provocative tests:** Without treatment 50% of fellow eyes of patients with acute
angle closure glaucoma developed acute angle closure glaucoma within 5 years. In a prospective multicentric study 129 eyes considered at risk for developing ACG were evaluated by a number of provocative tests. They were then followed up for up to 6 years without any intervention. 25 eyes actually had positive provocative tests, but only 6 developed angle closure. However of the 35 eyes which developed angle closure, only 6 had a positive provocative test.

The overall sensitivity and specificity of provocative tests for identifying eyes at risk for angle closure is low. Of the 4870 patients, subjected to dilatation none developed acute angle closure glaucoma, even though 38 patients were found to have slit to closed angles. Provocative testing can provide supportive evidence

1. Intermittent headache / eye ache with history of colored haloes, normal intraocular pressure, but ? occludable angles on gonioscopy.
2. Positive family history of glaucoma, normal IOP but suspect occludablity on gonioscopy.
3. Suspect occludablity on gonioscopy on a routine eye examination for a patient on treatment with medication which can precipitate a pupillary block.

**Gonioscopy**

Gonioscopy is the examination of the anterior chamber angle of the eye with the aid of special contact lenses and biomicroscopy. It is an essential step in the evaluation of all glaucoma patients and glaucoma suspects. The primary aim is to determine if the patient has an open angle or angle closure. Additionally one would like to assess if the open angles have a tendency to close, or if the angles are narrow with no potential to occlude.

The angle of the anterior chamber is created by two lines. One tangential to the trabecular meshwork and the other along the iris plane. The aqueous outflow system comprises of -

- **Schwalbes line** representing the peripheral edge of Descemets membrane.
- **Trabecular meshwork** the site of conventional aqueous outflow. It has two parts a lightly pigmented anterior part and a darker zone, posteriorly over the Schlemm's canal. The posterior trabecular meshwork is where more aqueous flows and hence greater iris pigment is present.

**Scleral spur** is a white band just posterior to the pigmented trabecular meshwork. It is formed by a projection from the inner scleral canal and represents the posterior boundary of inner scleral canal on which Schlemm's canal rests.

**Ciliary body band** represents the anterior aspect of the ciliary muscle, into which the root of the iris inserts and appears as a dark brown band posterior to the scleral spur.

Because of the air-cornea interface, internal reflection prevents a direct inspection of the angle. However gonioscopic lenses negate the total internal reflection and exceed the critical angle by altering the cornea air-fluid interface. An excellent review to methodology and interpretation is available. Direct gonioscopy is not widely performed in routine clinical practice because the equipment is not readily available for the general clinician and the procedure is less convenient than indirect gonioscopy.

**Direct Gonioscopies.** Koeppes’ lens, Swan-Jacob, Hoskin Barkan

**Indirect Gonioscopies :** Goldmann lenses, Thorpe and Ritch lens

**Indentation :** Zeiss, Posner, Susmann

While performing gonioscopy, the room should be dark and width of the slit beam should not cross the pupil so that light induced miosis does not result in misinterpretation of a narrow angle as not occludable.

Physiological factors which can change angle configuration include parasympathomimetic (causing forward movement of lens iris diaphragm) and sympathetic stimulation (dilation which can produce additional bunching of the iris into the angle). Because of the potential variability in the appearance of the angle, more than one gonioscopic examination is often necessary to determine the risk of developing angle closure.

Q. Is this angle occludable ?

When performing gonioscopy in a dark room with width of the light aperture not crossing the pupil, the aim is to assess if the drainage angle being examined has a potential to close or if there is any evidence that closure may have occurred in the past.

In order to allow comparison of studies (epidemiological research) occludable angles have been defined as one
in which the posterior, pigmented trabecular meshwork is not visible for 270 degrees or more, without indentation or manipulation of the gonioscope. Using the same definition APEDS reported 1.41% of occludable angles.

The big question is: Is this definition applicable to clinical practice. Wider angles may become occludable, and narrow angles may never close. Since gonioscopy is a dynamic process, evidence supporting suspect closure is possible.

- PAS in the superior angle are the most important and are pathognomic of angle closure in the absence of inflammation.
- Patchy pigmentation on the trabecular meshwork the superior angle is also suggestive of angle closure.
- Alternating opening and closure of the beam, a narrow angle may be seen to close and open, demonstrating the potential for closure of the angle ("on-off" sign).

The VES defined occludable angles when 180 degrees of the posterior trabecular meshwork was not seen. (10.3%).

**Ultrasound biomicroscopy**: Ultrasound biomicroscopy of the anterior segment allows accurate visualization of the iris, iris root, corneoscleral junction, the ciliary body and lens. It is of help to elucidate the mechanism of angle closure. However it is expensive with limited availability.

**Mechanism**: The final common pathway in the development of PAC is the formation of irreversible synechial adhesions between the peripheral iris and uveal surface of the trabecular meshwork. This is preceded by the development of appositional contact between the peripheral iris and trabecular meshwork. It is important to identify the involved mechanism as this works as guide to plan the management. It is important to remember that more than one mechanism may be at work.

**a) Pupillary Block**: Obstruction to the flow of aqueous usually arises between the posterior surface of the iris, in the region of the pupillary sphincter and the anterior surface of the lens. With ongoing aqueous production, the posterior chamber bulges forward with increasing pressure and the peripheral iris comes in contact with the trabecular meshwork. Gonioscopy reveals a steeply convex iris which is suggestive of a pressure differential between the posterior and anterior chamber.

**b) Plateaus iris mechanism**: An anatomical abnormality, the peripheral iris crowds the recess of the angle. When the pupil dilates, the iris is thrown into circumferential folds which come in contact with the trabecular meshwork. On Gonioscopy the iris inverts anteriorly in the scleral spur or leaves only a narrow ciliary body band. The iris is almost flat from the periphery to the extreme periphery where it creates a narrow angle recess.

The pure form of the plateaus iris syndrome is extremely rare and is proven by the occurrence of acute angle closure following dilatation despite a patent iridotomy with a deep central anterior chamber. Since more than one mechanism of angle closure may be present, an iridotomy should be done first. Plateaus iris, may then be treated with argon laser iridoplasty and or miotic therapy.

**c) Lens induced angle closure**: A large and/or anteriorly placed crystalline lens can also predispose to angle closure and can worsen the pupillary block.

**d) Creeping angle closure** starts within the depth of a narrow angle and then spread anteriorly to cover the posterior trabecular meshwork and then involve the anterior trabecular meshwork. This zipper effect leading to closure of the angle made Lowe describe it as creeping angle closure.

Chronic angle closure can result from synechial closure of the chamber angle from previous episodes of acute or subacute angle closure. In creeping angle closure the iris base creeps on to the trabecular meshwork leading to peripheral anterior synechiae. When more than $\frac{1}{2}$ of the angle is closed the intraocular pressure rises. Creeping angle closure may arise from an undiagnosed intermittent angle closure. Even chronic miotic therapy may cause worsening of the pupillary block. Creeping angle closure is more common amongst Asians.

**e) Cilio lenticular block**: In some cases misdirection of the posterior aqueous can cause primary angle closure. Typically the ciliary processes comes in contact with the lens equator, and/or a firm zonule/posterior capsule may cause flow of aqueous into the vitreous. The lens iris diaphragm is pushed anteriorly, occluding
the angle. Typically such eyes have narrow anterior chambers and after an iridotomy the use of cycloplics reduces the IOP and miotics paradoxically raise the IOP. Ultrasound biomicroscopy in such situations is very helpful.

f) Combined mechanism glaucoma : Here both pupillary block angle closure glaucoma and open angle glaucoma coexist. Typically a laser iridotomy alone fails to control the glaucoma. This is a form of chronic angle closure glaucoma where the trabecular meshwork may be damaged by intermittent or chronic trauma from the obstruction of the peripheral iris. In such cases the iridotomy helps to prevent further closure of the angle. The damage to the trabecular meshwork further impedes the outflow of aqueous. Here in addition to the laser iridotomy the open angle glaucoma component needs further medical therapy. Systemic drugs can induce angle closure in predisposed patients and include phenothiazines and their derivatives, antidepressants, antihistaminics, anti Parkinson drugs, tranquilizers and parasympatholytic and sympathomimetic agents.

Clinical presentation of angle closure :

1) Acute Angle Closure : The likelihood of a pupillary block producing angle closure depends on a shallow anterior chamber, short axial length of the eye, increased lens thickness and forward position of the lens and tightness of the contact between the iris and the lens. Critical anterior chamber depth most likely to lead to PACG is between 1.5 - 2.00mm. With the peripheral iris blocking the access of aqueous to flow out of the trabecular meshwork, the IOP increase. Physiologic mydriasis (dark room, movie theater), pharmacologic mydriasis (mydriatics and cycloplegics) and anxiety (pain, fear, trauma) and emotional disturbances can precipitate acute angle closure in predisposed eyes.

Gonioscopic proof of a closed angle in the involved eye is the most important sign of angle closure. If visualization of the angle is prevented by the corneal edema, then the uninvolved (fellow ) eye should always be examined for a narrow angle. Other possibilities to keep in mind are a non pupillary block glaucoma, secondary pupillary block and an acute glaucoma in eyes with open angles.

2) Intermittent (sub-acute) Angle Closure: Here attacks occur under conditions similar to acute angle closure, but resolve spontaneously. The intraocular pressure increases causing mild symptoms. However the aqueous then breaks through the pupillary block flow through the pupil into the anterior chamber again. The peripheral iris falls back and aqueous again flows through the trabecular meshwork. The likelihood of reoccurrence is high till a laser iridotomy is performed. Without this alternate passage, intermittent closure continues. Signs and symptoms are mild, and usually resolve spontaneously. The intraocular pressure is often normal between attacks. A shallow anterior chamber with / without PAS on gonioscopy is a hallmark sign. A history of intermittent rise in intraocular pressure is often present.

3) Chronic Angle Closure : Typically patients with chronic angle closure glaucoma have no symptoms and are often mistaken for open angle glaucoma. Gonioscopy is the only way to identify the closure and differentiate them. Initially the closure is appositional, but with time peripheral anterior synechiae develop leading to closure of the angle. This is accompanied by a raised IOP. Medical therapy initially shows a favourable response. However the IOP often fluctuates on treatment. Synechial closure progresses even as more medications are added. The success of treatment here depends on gonioscopy for early diagnosis and laser iridotomy.

Management

PAC presents with a raised IOP which is often symptomatic with or without disc damage. Management resolves around immediate control of symptoms and raised intraocular pressure, modifying configuration of the angle and preventing further closure, detection and prevention of further damage to the optic disc and visual field, as well as treating the fellow eye.
Medical treatment

Table 4. Medical management of acute angle closure

1. Acetazolamide (250-500mg) oral stat, then 125 to 250 mg t.i.d./ q.i.d, until symptoms subside
2. Topical Pilocarpine 2% stat, then q.i.d.
3. Analgesics and antiemetics as required.
4. Topical beta blockers b.d.
5. Topical steroids Loteprednol acetate q.i.d

Contra-indications and hypersensitivity to drugs should be excluded prior to starting treatment

- High doses of Pilocarpine should be used with caution as there is a risk of systemic pilocarpine toxicity.
- Paradoxical shallowing of anterior chamber can further aggravate the pupillary block
- Topical steroids help reduce the inflammatory reaction
- Often patients with PAC have other medical problems. Electrolyte disturbances, particularly hypokalemia can occur. Vomiting along with use of oral acetazolamide may cause or exacerbate this disturbance. Systemic hypotensive effect of beta blockers can be aggravated by the electrolyte disturbance further increasing the risk of circulatory disturbances. Intravenous hyperosmotics can also aggravate circulatory disturbances. Analgesics and antiemetics should be used as required.
- If topical and intravenous therapy is unable to reduce the intraocular pressure within 3-4 hours, additional measures such as corneal indentation, manual compression or a laser iridotomy may be indicated.

Modification of angle configuration:

A laser iridotomy is the definitive treatment for a pupillary block ACG. Since it is often difficult to rule out a pupillary block component in any case of PAC, a laser iridotomy is indicated in every case unless there is a contra indication. It is best performed when the eye is quiet, cornea is clear and there is no intraocular inflammation or uveal congestion. This rarely occurs when a patient presents with an acute ACG.

- If the cornea is clear and inflammation is less a laser iridotomy can be performed.
- In case residual inflammation is present, after successfully breaking an acute attack, the iris tissue is boggy, in such cases the laser iridotomy can be deferred for a few days, maintaining the patient on glaucoma treatment and topical steroids.

* In case the cornea does not clear to allow an iridotomy despite adequate measures, a partial pupiloplasty (to peak the pupil) with low power applications of Argon laser to temporarily break the pupillary block can be considered. Alternatively the argon laser can be used to contract the peripheral iris and pull it away from the trabecular meshwork (peripheral laser iridoplasty)

As with medical treatment, neither of these alternative modalities provide permanent relief from a pupillary block and must be followed by a definitive laser iridotomy.

A laser iridotomy for angle closure glaucoma is not always successful. Treatment of acute angle closure glaucoma with an iridotomy alone or in combination with miotics controlled the intraocular pressure in 77% cases if there was no initial visual field loss. This dropped to 29% in the presence of initial visual field loss. Following laser treatment, if the glaucoma is due to a pupillary block or appositional closure the angle should open wider following treatment. However the central anterior chamber depth may not change. Failure of the angle to open following the laser iridotomy results from extreme PAS, or if the angle closure was not from pupillary block. Additional iridotomies will not remedy this.

As laser iridotomy is sufficiently safe, a trial with laser iridotomy followed by medical therapy is generally quite appropriate before proceeding to more invasive trabeculectomy.

In some eyes the angle will open sufficiently following the laser iridotomy, but the intraocular pressure remains elevated. This is usually from trauma to the trabecular meshwork or where there is an inherently reduced aqueous outflow. Such cases would require additional medical therapy, before proceeding to a filtering procedure.

Prophylactic laser iridotomy

A laser iridotomy is always indicated in the fellow eye of a patient who has suffered an acute angle closure in the first eye. Other indications include, an angle so narrow that a provocative test is dangerous and unnecessary (an angle which is narrowed to a slit or closed, or requires indentation gonioscopy to view the scleral spur. Other indications for a laser iridotomy are
presence of PAS in an eye with a narrow angle, family history of angle closure glaucoma and need for frequent pupillary dilatation in patients with narrow angles (diabetes)

Like open angle glaucoma ACG is a bilateral disease. The fellow eye almost always has an occludable angle and should be treated with a laser iridotomy, once the first eye is stable. However an iridotomy in the fellow eye may not prevent the need for treatment. 50% of patients with PACG treated with bilateral peripheral iridectomies required additional treatment of some type in the involved eye. 25% needed treatment in the uninvolved eye post laser.

**Surgical Iridectomy**

Given the current evidence, the sole indication for a surgical iridectomy is probably the lack of access to a laser. The risk of complications from intraocular surgery such as endophthalmitis and iris prolapse do not seem justified when a closed surgical technique is available. The technique may be of use in eyes in which a symptomatic rise in IOP which cannot be controlled by medical or laser therapy, especially those in whom corneal edema is a persistent problem.

Glaucomatous optic neuropathy in primary angle closure : Management and visual prognosis

When symptoms have settled and short term IOP control has been achieved, a full glaucoma work-up should be carried out. A detailed gonioscopic examination of both eyes, visual field assessment and recording of the optic disc status are essential to plan appropriate long-term management. If glaucomatous optic neuropathy is detected, the therapeutic options are as follows:-

**Laser Peripheral Iridotomy**

Laser peripheral iridotomy is the treatment of choice for people with glaucomatous optic neuropathy in PAC. In one study 140 eyes of 104 people with PAC in Japan, treated by argon laser iridotomy revealed that prior to treatment 73/109 (67%) eyes had a cup: disc ratio of 0.7, the cup: disc ratio enlarged in 31 (28%) and was unchanged in 64 (59%), mean follow-up 1.7 and 2.7 years (in two groups) , visual fields defects were minimal or absent in 96/118 (81%), moderate in 19/118 (16%) and advanced in 3/118 (3%). The defects progressed in only 3 patients (all with initially mild changes). **IOP < 21 mmHg (with or without medication) after PI was achieved in 94% (44).**

IOP control was more likely to be successful if there were 0 degree PAS. There was no significant change in the amount of PAS during the follow-up period. Loss of visual acuity by more than 3 lines occurred in 19%, due to progression of lens opacities.

Another retrospective analysis of 57 Singaporeans with symptomatic PAC found that more than 24 hours delay in presentation, or the need for a laser iridoplasty to achieve short-term pressure control, was associated with worse pressure control after laser iridotomy (mean follow – up period 20 months). Another retrospective study in South Africa of 52 asymptomatic patients (78 eyes) followed for a mean period of 22 months reported that IOP was controlled (< 21 mmHg) without medication in 9%, and with medication in 51% of eyes, trabeculectomy was required in 29% of eyes, risk factors for needing trabeculectomy were: IOP on presentation > 35 mm Hg, 3 quadrants of synechial angle closure, and cup: disc ratio of >0.6. 36% of eyes with these risk factors needing trabeculectomy were controlled by PI with or without medication.

The likelihood that a non-invasive procedure will control IOP and arrest progression of optic neuropathy justifies the use of laser PI as first line treatment in all but the most severe cases.

**Medical Therapy**

If satisfactory pressure control cannot be achieved with a laser iridotomy alone, topical medical therapy can be used in a manner similar to that for POAG. A target pressure should be set according to the degree of nerve damage and field loss. If the iris contour has been satisfactorily changed by iridotomy (implying that pupil block was the predominant mechanism), then a first line drug as felt appropriate may be used. If the iris profile has not changed after the laser iridotomy (suggesting peripheral iris crowding is the predominant mechanism), Pilocarpine 1-2% is a more appropriate choice. An α₂-agonist is an appropriate second-line therapy.

**Trabeculectomy**

Trabeculectomy is indicated in cases of PAC with glaucoma that cannot be controlled by laser iridotomy
and medication. There is often concern that aqueous (ciliolenticular block) misdirection may complicate trabeculectomy in cases with PAC, although published data and anecdotal experience do not support this. Despite the finding that eyes with PAC do not seem to suffer especially high rates of malignant glaucoma, cases of this problematic complication do occur. The condition may be recognized by progressive asymmetrical axial shallowing of the anterior chamber. The disorder stems from misdirection of aqueous flow by closure of the ciliolenticular space. Dilating the ciliary ring is probably the best preventive measure and the agents of choice being either cyclopentolate or homatropine.

Primary trabeculectomy is an option for cases of PAC in which immediate pressure control cannot be achieved. Patients with very advanced PAS, optic nerve damage, and visual field loss, are often considered for primary trabeculectomy. A trial of laser iridotomy in all cases, although if synechial angle closure for more than 180 is identified after laser treatment, the patient should be considered at high risk of needing a trabeculectomy to achieve control.

**Lens extraction** Since the position of the lens determines the iris profile, and therefore the angle configuration, lens extraction is a logical choice for surgical management of raised IOP in cases of PAC with visual impairment due to cataract. Extracapsular cataract extraction was used in the management of PAC in 21 eyes of 20 patients (2 with raised IOP alone, 5 symptomatic, and 14 asymptomatic). In 14 cases lens extraction was performed in place of filtering surgery, where peripheral iridectomy or previous filtering surgery had failed. Mean IOP reduced from 31 to 16 mm Hg after surgery, 16/21 eyes did not require further medication (follow up:6-42 months), IOP was reduced even if there were extensive previous PAS, in 6 patients with previous failed filtering surgery, lens extraction gave a median IOP reduction of 17.5 mmHg (range 5-30)

**Management of the asymptomatic narrow angle**

In a multi-centre study of 129 asymptomatic patients with anterior chamber depth < 2 mm, or drainage angles that were potentially occludable, only 6% developed signs or symptoms consistent with PAC over a mean period of 2.7 years (maximum follow up 6 years) it would therefore appear that an individual risk of developing visually threatening sequelae is low on a year-to-year basis. However, **it is now an accepted practice to perform a laser iridotomy on patients with early gonioscopic evidence of angle closure, reflecting the perceived (although unproven) high benefit/risk ratio for this procedure.** This view is probably justified when one considers the potential for late-presentation or misdiagnosis under non-ophthalmic care, and low incidence of sight-threatening complications of laser iridotomy.

The management of an eye contralateral to one that had an episode of symptomatic PAC is open to less conjecture. Follow up of 200 such “fellow” eyes found 113 were managed by observation or with topical Pilocarpine. Of this number 58 developed symptomatic PAC (half within a five year period), 26 of the 58 were using topical Pilocarpine. In a further 250 patients with PAC, 72 did not have prophylactic peripheral iridectomy. Forty three developed PAC (33 symptomatic, 10 asymptomatic or unknown), 33 of these were affected within 6 years. This is overwhelming evidence in favour of prophylactic peripheral iridotomy by laser, or surgical iridectomy if no laser is available.

**The Indian Perspective:**

In the glaucoma clinic of an eye hospital, 45.9% of all primary adult glaucomas were of angle closure glaucoma. Of these 24.8% had acute angle closure glaucoma, 31.2% had subacute and 44% had chronic glaucoma. More than 80% of the chronic eyes had no significant symptoms. Nd Yag laser iridotomy alone or with topical anti glaucoma medication controlled the IOP in 48.3% of acute angle closure glaucomas, 78.8% of subacute and 30% of chronic eyes. Similar data from another tertiary setting reported 15.88% acute, 19.26% subacute and 64.86 % chronic angle closure amongst the 888 patients with Primary angle closure glaucoma. Over a period of five years, 22% of occludable angles progressed to primary angle closure glaucoma and 28.5% of the primary angle closure developed optic disc and visual field changes.

Light and electron microscopic studies have revealed accumulation of pigment in the widened trabecular
spaces and Schlemm’s canal (acute PACG). The endothelial cells were attenuated and devoid of subcellular components. Chronic angle closure was associated with loss of the trabecular architecture with narrower trabecular spaces and fusion of the trabecular beams. Loss of endothelial cells and reactive repair was visible in areas away from peripheral anterior synechiae. Following an acute attack of PACG, long term followup is needed despite a laser iridotomy as the IOP may rise later due to progressive compromise of the outflow facility.

**Conclusion**

Primary Open Angle Glaucoma is characterized by a typically progressive glaucomatous optic neuropathy with correlating visual field loss. IOP is one of the risk factors responsible for this damage to the optic nerve. However even though factors other than IOP are involved in the pathogenesis of glaucoma, IOP is the only factor that can be modulated to date. The decision to treat is individualized depending on the whether the level of IOP will lead to progressive nerve damage. Available treatment algorithms rely on medical management to achieve the target IOP, failing which filtering surgery can be resorted to. In the Indian context, early filtering surgery to achieve the desired target pressure is a viable alternative. Laser trabeculoplasty is an intermediate step. The role of neuroprotection is not yet established clinically.

Primary Angle Closure Glaucoma: There is a significant change in the perception of PACG. The definition of PACG has undergone a change. Angle closure is now described as an anatomical disorder where symptomatology does not specify the involved mechanism. Screening for angle closure glaucoma appears tempting, but is still not a viable option. Improved detection with simple tests (flashlight test and von Herrick’s test) and confirmation on gonioscopy plays a key role in diagnosis. Provocative testing is likely to provide a supportive role in asymptomatic occludable angles. Asymptomatic, chronic angle closure glaucoma mimicking POAG is common. Gonioscopy is the confirmatory test. After the definitive treatment, laser iridotomy, angle closure is treated medically or surgically in the same manner as open angle glaucoma. Treatment of the fellow eye with a laser iridotomy is mandatory.

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Anatomical and Visual Outcomes of Surgery for Idiopathic Macular Holes

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Abstract

Purpose: To determine the rate of anatomical closure and visual outcome following vitreous surgery for idiopathic macular hole (IMH) using Optical Coherence Tomography (OCT).

Methods: Interventional case series. 23 eyes with idiopathic macular hole having a preoperative OCT macular hole diameter ranging from 280 to 1051 microns underwent vitrectomy with internal limiting membrane peeling and perfluoropropane gas injection.


Results: Type I closure was seen in 14 eyes and type II closure was seen in 7 eyes. Anatomical closure was not achieved in 2 eyes with macular hole diameter of greater than 400 microns.

Conclusion: OCT measurements are useful to predict the anatomical and visual outcomes following surgery for idiopathic macular hole.

Introduction

The first clinical description of a macular hole was published by Henry Noyes in 1871. Since then our understanding of development and pathogenesis of macular holes has improved. However, it took more than 100 years, until Kelly and Wendell reported the first successful closure of a series of macular holes by pars plana vitrectomy and induction of posterior vitreous detachment in 1991. Several authors have reported significantly higher rates of anatomical closure and visual rehabilitation in many of these cases. The postoperative success rate varies between 86% and 95% with improvement in visual acuity in a large percentage of cases.

Recent attempts to use imaging techniques such as confocal scanning laser tomograph and scanning laser ophthalmoscope to predict success suggest a correlation between the macular hole size and visual recovery. This study used optical coherence tomography (OCT) to measure the preoperative macular hole size and correlated this with the postoperative rate of anatomical closure. OCT is a recently introduced diagnostic tool for high resolution, cross sectional imaging of the posterior and anterior segment of the eye with an axial resolution of 10µm and a transverse resolution of 30µm. OCT has been of immense use in understanding the pathogenesis of and staging of macular holes prognostication of surgical outcome and grading the surgical outcomes as well.

Materials and methods

A retrospective review between January 2005 to April 2006 of all eyes with an idiopathic macular hole that were examined preoperatively and postoperatively by OCT at our hospital was performed. Only eyes diagnosed as having idiopathic macular holes were