Clinical Practice Module

Quality Assurance in

Management of Primary Angle Closure Glaucoma

Dr. S.R. Krishnadas

Prepared by

Aravind Eye Hospitals
Madurai, Tirunelveli and Coimbatore

For

National Programme for Control of Blindness
Directorate General of Health Services
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THE EYE IN MYTHOLOGY

The Greek goddess Pallas Athena, the daughter of Zeus, the supreme deity of ancient Greece, She was born in full battle attire. She cured the blinded eyes of Lycurgos, whereupon a temple was erected to her in Sparta as Athena Ophthalamitas, the healer of eye diseases.

She was also known as Athena Hygeia, Purifier of the air. She was the patroness of Athens. When the cult spread to Rome, she became Minerva Medica, the goddess of the healing arts.

The element Palladium - Pd - known since 803, was names after the planet Pallas, named for Pallas Athena.

Primary Angle Closure Glaucoma

Definition

Primary angle closure glaucoma is characterised by appositional or synechial angle closure of the drainage angle of the eye due to relative pupillary block[1] in the absence of other causes of angle closure. The patients may or may not present with elevated IOP or optic nerve damage. Although patients may present with symptoms of primary angle closure glaucoma in one eye, the anatomical and pathophysiological factors predisposing to the development of glaucoma is bilateral.

Relative pupillary block is due to increased resistance to flow of aqueous from the posterior to the anterior chamber resulting from enhanced iridolenticular contact in predisposed eyes. As a consequence there is a greater pressure differential in the posterior chamber with forward bowing of peripheral iris (iris bombe) with closure of the drainage angle (appositional angle closure). Prolonged contact of the peripheral iris with trabecular meshwork leads to formation of peripheral anterior synechiae and functional damage to trabecular meshwork.

Acute Angle closure glaucoma

Acute primary angle closure glaucoma occurs due to rapid elevation of IOP as a result of sudden blockage of the trabecular meshwork by the iris. The condition is manifested by pain, blurred vision, colored halo, nausea and vomiting. IOP elevation is high and causes corneal epithelial edema which is responsible for blurring of vision and halos. The typical signs of acute angle closure glaucoma include:

- High IOP.
- Mid dilated, sluggish and vertical oval or irregular pupil.
- Corneal epithileal edema.
- Shallow anterior chamber.
- Mild aqueous flare and cells.
- Optic nerve head edema.
- Diminished visual acuity.

Definitive diagnosis depends on demonstration of closed angles on gonioscopic evaluation. Corneal edema during an acute attack may preclude gonioscopic examination, in which case it may be repeated once corneal edema clears after medical treatment. Topical anhydrous glycerine may also be administered to clear the edema and enable gonioscopic visualisation of the angle. Estimation of peripheral anterior chamber depth by Van Herick's[9] technique and demonstration of narrow angles by gonioscopy of the fellow eye are other methods to confirm angle closure glaucoma.

Sequelae of previous attacks of acute angle closure include posterior synechiae, peripheral anterior synechiae, impaired outflow facility, anterior subcapsular lens opacities (glaucomflecken of Vogt), sector or generalised atrophy of iris, pallor and cupping of optic disc and visual field loss. Differential diagnosis of acute angle closure include certain open angle glaucomas with high IOP such as post traumatic angle recession, exfoliation syndrome, glaucomatocyclitic crisis and early neovascular glaucoma. These entities are diagnosed by careful clinical and gonioscopic evaluation.

Subacute Angle Closure Glaucoma

Subacute, or intermittent or prodromal angle closure glaucoma is characterised by episodes of blurred vision, haloes, and mild pain caused by elevated IOP. These episodes resolve spontaneously or by miosis induced by sleep. The IOP is normal between episodes and correct diagnosis is arrived only by a high index of suspicion and demonstration of narrow and occludable angles on gonioscopy with or without evidence of peripheral anterior synechiae. Subacute angle closure glaucoma may be confused with headache or migraine. This condition may progress to chronic angle closure glaucoma or interrupted by acute angle closure glaucoma that does not resolve spontaneously. Laser iridotomy is the treatment of choice in subacute angle closure glaucoma.

Chronic Angle Closure Glaucoma

Chronic angle closure develops either after acute angle closure in which peripheral anterior synechiae (PAS) persist or when the anterior chamber angle closes gradually and IOP rises as the filtration angle is increasingly compromised. Indentation gonioscopy reveals presence of permanent PAS. In the absence of any symptoms, the clinical course resembles that of primary open angle glaucoma with only a modest elevation of IOP, progressive excavation of the optic nerve head and characteristic glaucomatous visual field loss. Gonioscopic evaluation of the angle is critical in the diagnosis of chronic angle closure glaucoma. Laser iridotomy is necessary to relieve pupillary block and prevent further peripheral anterior synechiae and angle closure.

The Narrow Anterior Chamber Angle (Angle Closure Glaucoma Suspect)

A small percentage of patients with narrow angles develop angle closure glaucoma. Many investigators have attempted to predict which asymptomatic individuals with narrow angles will develop angle closure by performing provocative tests. These tests are planned to result in a limited form of angle closure, which can be detected by gonioscopic evaluation and IOP measurement. The methods commonly used are summarised in the section on Diagnosis. An IOP increase of 8 mmHg or more with gonioscopic evidence of angle closure is considered positive. An asymmetric pressure rise between the two eyes with a corresponding degree of angle closure is also considered positive of angle closure glaucoma. The positive predictive value of these provocative tests have not been validated in any prospective study.

The decision to treat an asymptomatic patient with critically narrow or occludable angles rests on the clinical judgement and accurate assessment of the anterior chamber angle by the attending ophthalmologist. An iridotomy is not necessary as a prophylactic measure in all individuals with narrow angles. All individuals demonstrated to have narrow angles on gonioscopic evaluation are to be advised about the symptoms of angle closure, of the need for immediate ophthalmic referral, attention to symptoms that might occur and of the need for long term periodic follow up.

Intervention in the form of prophylactic iridotomy can be however, considered for individuals deemed to be at high risk of angle closure in the foreseeable future, such as those with extremely narrow angles as judged by the attending ophthalmologist, those who require recurrent dilation for assessment of the posterior segment (diabetics), those who require topical or systemic medications that pose a significant risk of precipitating angle closure glaucoma. Individuals with narrow angles should be warned about medications that are likely to precipitate angle closure glaucoma by pupillary dilation.

Prophylactic iridotomy may also be indicated in the following situations besides the fellow eyes in patients with acute angle closure glaucoma:

Patients with very narrow angles who report symptoms of angle closure (subacute angle closure).

Patients with narrow angles and a well documented family history of angle closure glaucoma and/or with physical findings suggestive of previous episodes of angle closure.

Patients with anatomically narrow angles and appositional closure of at least some portion of the angle are also likely candidates for prophylactic iridotomy.

The availability of laser iridotomy and its relative safety as compared to surgical iridectomy has resulted in enhanced benefit-to-risk ratio and permits consideration of intervention in individuals without documented evidence of angle closure, but who have a reasonable likelihood of benefit from prophylactic therapy, such as those with narrow angles and elevated IOP or with documented progressive narrowing of the angle.

Epidemiology of Primary Angle Closure Glaucoma

Population based studies in the West have led to a better understanding of the prevalence of POAG, but primary angle closure glaucoma is a relatively rare disease in Europe and America (0.09% in Wales[3], 0.17% in Bedford[4], and 0.17% in Dalby[5], Sweden). Substantial evidence of population based studies confirm high prevalence of primary angle closure glaucoma in Eskimos, which is estimated to be about 20-40 times that in the Caucasians. In South Asia, the inference from the case series and population based surveys is that the proportion of open and closed angle glaucoma is roughly equivalent among individuals with primary glaucoma. Pararajasekaram reported that in a consecutive 325 individuals with primary glaucoma in Sri Lanka, 51.4% had PACG with a majority (66%) classified as chronic. In a ten year case series of 4365 glaucoma patients at AIIMS, New Delhi, 55% patients with primary glaucoma were diagnosed as PACG. Hospital surveys in Chandigarh and Aravind Eye hospital, Madurai, India also identified 47% primary glaucomas to be from angle closure[6,7]. A population based study in Chandigarh, India revealed PACG to account for 40% of all primary glaucomas. The blindness rate due to angle closure glaucoma reported in a New Delhi study were 13% monocular and 2.4% bilateral blindness (by WHO criteria).

Recently, well designed population based studies in India have estimated the prevalence of primary angle closure glaucoma in the community. The Andhra Pradesh Eye study[8] had determined the prevalence of manifest PACG (IOP 22 mmHg, optic disc damage and visual field loss with narrow angles on gonioscopy) to be 1.08% in individuals aged over 40. Occludable angles (defined as pigmented trabecular meshwork not visible by gonioscopy in three quarters or more of the angle) was prevalent in 2.21% in people aged over 40[8]. 83% of those with manifest PACG had chronic angle closure and 41% of individuals were blind in one or both the eyes. Only a third of individuals with manifest PACG had been previously diagnosed, while 8% alone with manifest PACG had iridotomy performed previously. Half of those with previously diagnosed PACG were blind in one or both eyes either due to delay in the initial diagnosis or inadequate treatment. The prevalence of occludable angles without manifest PACG has been observed to be 2.21% in individuals over 40 years.The Aravind Comprehensive Eye Survey had estimated the prevalence of PACG to be 0.7% *(Ramakrishnan et al, presented at the annual meet of the ARVO, May 2000 at Fort Lauderdale, Florida)*. The Vellore Eye study had estimated PACG to be prevalent in about 4.4% of the population.

With a conservative estimate of manifest PACG to be around 0.7-1.08% in adults aged over 40 years, about 3 million adult Indians have manifest angle closure glaucoma and as many as 1.2 million (41%) of these individuals are blind due to glaucoma in one or both their eyes. Another 6 million Indians have occludable angles and are at risk of angle closure. Considering the fact that about 9 million in India have angle closure glaucoma or at risk of developing angle closure glaucoma, efficient screening measures are mandatory to diagnose PACG early, particularly since intervention in the form of laser iridotomy can reduce vision loss.

Issues in Screening for Angle Closure Glaucoma

Various methods employed to screen the individuals in a population for angle closure glaucoma include the oblique flashlight test, Limbal anterior chamber depth or Van Herick technique, Provocative tests, IOP estimation, Gonioscopy combined with optic disc evaluation and visual field analysis.

The *oblique flashlight test* uses a penlight held parallel to iris plane on the temporal aspect of the eye and the shadow cast on nasal iris is evaluated. Shun-Yi[24] County study has estimated the sensitivity of the flashlight test to be 81% and specificity as 96%. The chief advantages of this technique are its low cost and ease of use by non ophthalmologists in population screening. Several investigators[25], however opine that this is a crude mode of screening for angle closure glaucoma and has not been evaluated in any large scale population screening.

Limbal Anterior chamber depth or *Van Herick's technique* estimates the depth of limbal anterior chamber in the slit lamp as a fraction of the corneal thickness. Individuals with grade 1 or 2, where the angle width is < ¼ corneal thickness, are at risk of angle closure. This technique has been used in several population based screening in Caucasians, Chinese, Japanese and Eskimos. Vargas reported a sensitivity of 85% and sensitivity of 84% of limbal anterior chamber depth technique in screening individuals for PACG.

The value of provocative tests have not been fully elucidated in population based prospective studies and current opinion does not recommend provocative tests to identify individuals at risk of angle closure glaucoma. Gonioscopy is the most definitive diagnostic tool to diagnose angle closure, but would be impracticable in the setting of population based screening in the community. Until better methods are available, the oblique flashlight test and Van Herick techniques will remain the most useful means to screen population at risk for angle closure.

Natural History

Useful vision was invariably lost in eyes with angle closure glaucoma prior to introduction of iridectomy as treatment by Von Graefe [2] in 1856. 58-74% of untreated fellow eyes develop angle closure glaucoma in five years, the natural history was for the patient to develop bilateral, painful blind eyes from glaucomatous optic atrophy and elevated IOP.

Diagnosis

History- the comprehensive baseline glaucoma evaluation include a history with particular attention to eliciting symptoms that are suggestive of episodes of angle closure glaucoma (blurred vision, colored halos around lights, eye pain, browache, headache, ocular congestion, etc). A family history of acute angle closure glaucoma, or history of blindness from glaucoma is also suggestive and should be specifically sought.

Ocular Examination

The diagnosis of PACG requires the presence of narrow angles and evidence of pupillary block in the absence of secondary mechanisms of angle closure. A positive evidence of pupillary block leading to angle closure include the following:

- Appositional closure of the drainage angle of the anterior chamber.
- Evidence or episodes of past angle closure- peripheral anterior synechiae, glaucomflecken, iris atrophy, pigments anterior to schwalbe's line, etc.

Refraction and Gonioscopy are two aspects of ocular evaluation that should receive particular attention.

Hyperopic eyes are at increased risk of angle closure. Slit lamp evaluation should document changes in the cornea, peripheral and central anterior chamber depth and signs of previous angle closure such as segmental iris atrophy, glaucomflecken, peripheral anterior synechiae, posterior synechiae and pupillary dysfunction are identified and carefully documented.

Gonioscopy is required for all patients in whom glaucoma is suspected and for evaluation of angle closure. Gonioscopic evaluation of the anterior chamber angle is critical in the diagnosis and follow up management of eyes with angle closure glaucoma. Numerous grading systems have been evolved in an attempt to correlate the appearance and width of the anterior chamber angle with potential for angle closure. Shaffer[10] used the angular width of the angle recess as the criterion for grading the angle in an attempt to correlate it with the potential for angle closure. Spaeth[11] had suggested that in addition to angular width of the angle recess, the configuration of the peripheral iris and the apparent insertion of the iris root are critical in evaluation of the angle in PACG. Compression (Indentation gonioscopy) is performed to differentiate appositional iris closure from synechial angle closure.

As in all patients with glaucoma, IOP is measured, visual fields evaluated and optic discs examined and documented. Some components of the eye evaluation may have to be deferred in some eyes until after acute angle closure glaucoma has resolved or until iridotomies have been performed for pupillary block.

Provocative Tests in Assessing Angle Closure Glaucoma

Most individuals with narrow angles do not develop angle closure glaucoma. The prevalence of anatomically narrow angles in the US is 2-6%, while less than 0.2% develop glaucoma. Prevalence studies in India have shown that prevalence of angle closure glaucoma in the population to be between 0.7-1.1%, a fraction of those with occludable angles. If one could predict which eyes are potentially at high risk of angle closure, then much morbidity and visual impairment could be prevented by prophylactic iridotomy. Some clinicians suggest use of provocative tests to predict which eyes are at high risk of developing angle closure. The rationale of performing these tests is that producing a mild episode of angle closure under physician's observation is unlikely to result in permanent damage since the condition is promptly reversed by treatment and also indicate which eye, with occludable angle will require prophylactic laser iridotomy. The various provocative tests include the Mydriatic test, Dark room test, Prone test, Dark Room-Prone test, and Phenylephrine-Pilocarpine test.

Most ophthalmologists question the clinical value of provocative tests in assessment of eyes with potential for angle closure glaucoma. In a study[12] of a longitudinal follow up of 129 eyes suspected of angle closure glaucoma, who were subjected to gonioscopy, refraction, anterior chamber pachymetry, ultrasound biomicroscopy and an angle-closure provocative test, it was concluded by the authors that none of the diagnostic tests including the provocative tests, showed a high sensitivity or positive predictive accuracy in detecting eyes that later developed angle closure glaucoma. It has been suggested that a high index of suspicion from an accurate history and clinical evaluation alone is the best guide to determine, which individual with narrow angles would require prophylactic iridotomy[13].

Treatment

The definitive primary modality of treatment of all forms of primary angle closure glaucoma is essentially surgical- either laser iridotomy or incisional iridectomy. Laser treatment is the preferred method to perform iridectomy owing to its increased benefit-risk ratio. It is however, desirable to first reduce the IOP by medical measures before definitive treatment is instituted.

The treatment of ***acute angle closure glaucoma*** has the following specific goals:

- To reverse or terminate the acute angle closure by medical treatment.
- By rapid reversal of acute attack, the damage to the optic nerve head and the trabecular meshwork is minimised and formation of peripheral anterior synechiae is retarded.
- To perform the definitive treatment of iridotomy to eliminate pupillary block.
- Assess the anterior chamber angle, optic disc and visual field damage and institute adjunctive medical treatment.
- Prophylactic management of the fellow eye[22] prone for angle closure by performing an iridotomy.

The critical approach, in patients with acute angle closure glaucoma, is to cause moderate pupillary miosis to tighten and thin the peripheral iris by cholinergic agents like pilocarpine. This pulls the peripheral iris from the angle and allows egress of aqueous. 1-2% pilocarpine is administered two to three times over a 30 minute period and thereafter once in six hours. The temptation to instill pilocarpine every few minutes is to be avoided, since such excessive use of miotics could result in cholinergic crisis, nausea, vomiting, diarrhoea, sweating, bradycardia and hypotension, especially in the elderly.

In some instances, where the IOP is extremely high, the pupillary sphincter muscle is paralysed due to pressure induced ischaemia and may be unresponsive to pilocarpine[14,15]. The IOP needs to be reduced by other medical measures (aqueous suppressants)[16] before the sphincter becomes responsive to the effect of miotics. A combination of topical beta antagonist, alpha receptor agonist, carbonic anhydrase inhibitor and hyperosmotic agents are employed to reduce aqueous formation and intraocular pressure. In the absence of nausea and vomiting, oral acetazolamide is administered in the dosage of 500 mg. The hyperosmotic agents are often the most effective agents in the management of acute angle closure glaucoma. Oral glycerine is administered as a 50% solution in the dose of 1.5-4.0 ml/kg body weight. This drug is avoided in diabetics, owing to its high calorific value. If the patient is nauseous or vomiting, 20% mannitol is administered intravenously in a dose of 2-7 ml/kg body weight.

An acute angle closure glaucoma is considered to be reversed only when IOP is low, pupil is miotic (unless iris sphincter muscle is not too atrophic), and angle is open on gonioscopy. Mere reduction of IOP alone is not sufficient evidence of reversal of acute angle closure, since ciliary shock following an acute episode of angle closure can cause profound hyposecretion and hypotony. Unless the filtration angle opens, IOP is likely to rise to high levels once the ciliary body resumes aqueous secretion.

Iridectomy (incisional) or laser iridotomy is the definitive treatment for acute angle closure glaucoma with pupillary block once the acute episode of IOP elevation has been reversed. In the absence of iridectomy, repeated episodes of angle closure cause progressive synechial angle closure and refractory glaucoma. Iridectomy establishes a free communication between the anterior and posterior chambers bypassing the increased resistance to the aqueous flow at the pupil and there is insufficient pressure differential to push the peripheral iris against the angle of the anterior chamber. This causes widening of the peripheral anterior chamber depth and reversal of the appositional angle closure. If extensive peripheral anterior synechiae have developed from prolonged angle closure, iridectomy alone is insufficient to reduce IOP, and adjunctive medical therapy or filtering surgery is indicated.

In the past two decades, argon and Nd:YAG lasers have largely replaced surgical iridectomy owing to their favorable risk to benefit ratio. When an iridotomy cannot be achieved due to corneal edema, laser pupilloplasty or peripheral iridoplasty[17] may break the acute attack of angle closure. Surgical iridectomy is now largely indicated only in the following circumstances:

- Lasers fail to achieve a patent iridotomy.
- Recurrent closure of laser iridotomies.
- Non-availability or shut down of laser apparatus.
- Unresolving corneal edema and extremely shallow chambers.
- Patient unable to sit in slit lamp or co operate for the procedure.

An attack of acute angle closure glaucoma produces iris atrophy and sufficiently elevate the pupil and pupillary block may thus be spontaneously reversed. An iridectomy is not necessary in these eyes, although medical treatment to reduce IOP may be required. In eyes with visually significant cataract, acute angle closure glaucoma if reversed medically, can be followed up with cataract extraction with iridectomy. The crystalline lens plays a critical role in pathogenesis of relative pupillary block in angle closure glaucoma and hence its removal eliminates future pupillary block and angle closure.

Prolonged episodes of acute angle closure glaucoma result in extensive PAS and eventually require glaucoma surgery to reduce IOP. Considerable controversy exists as to whether non resolving acute angle closure glaucoma should be primarily treated with filtering operation, or be subject to the sequence of iridectomy, stepped up medical treatment and finally, glaucoma drainage operation. Some investigators have suggested to proceed with glaucoma filtering surgery as judged by the following criteria:

- The duration of acute attack- for attacks lasting longer than 36 to 72 hours, primary filtering surgery has been proposed.
- Eyes with aqueous facility of outflow < 0.10 ml/min/mmHg after acute angle closure glaucoma respond better to filtering surgery.
- The extent of peripheral anterior synechiae as judged by compression gonioscopy-if synechiae covered more than 50-75% of the anterior chamber angle, filtering operation may be indicated.
- The presence of optic nerve cupping and visual field loss may also favor primary filtering operations.

Several studies, however, demonstrated the practical difficulties in predicting the eye that would require filtering surgery after iridectomy for angle closure glaucoma. Many eyes had well controlled IOP despite extensive damage to the drainage angle from acute angle closure glaucoma. Iridectomy combined with medical treatment had resulted in equally good IOP control as compared to filtering surgery with fewer surgical complications.

With the advent of lasers in management of glaucoma, iridotomy with lasers gives an excellently favorable benefit to risk ratio when compared to surgical iridectomy. ***The current recommendation is for all eyes to receive a laser iridectomy, and management of residual glaucoma by sequential medical treatment and glaucoma filtering surgery.***

There are few reports that peripheral anterior synechiae can be lysed surgically (goniosynechiolysis) or reversed by argon laser iridoplasty, but these claims require confirmation by long term prospective studies.

Prophylactic Treatment of Fellow Eyes

40-80% of the contralateral eyes develop angle closure[20,21] glaucoma over a 5- to 10-year period. In the current opinion, it is safer to perform prophylactic laser iridotomies in these eyes than to observe them for development of angle closure glaucoma. Chronic miotic therapy either for prophylaxis or treatment of established angle closure is not a substitute[20,21] for iridotomy. 39% of fellow eyes treated in this manner develop acute angle closure in five years and several eyes will have progressive synechial angle closure despite use of miotics. Prolonged use of miotics increase pupillary block and cause synechiae formation. Acute angle closure has been reported to be rare[23] after prophylactic iridotomy.

Subacute Primary Angle Closure Glaucoma

The subacute primary angle closure glaucoma with pupillary block has also been referred to as prodromal, intermittent or subclinical angle closure. In this milder form of angle closure, symptoms are minimal or absent. Patients report ocular discomfort, pain, blurred vision, congestion and haloes which were relieved by sleep or exposure to bright light. The symptoms of prodromal angle closure glaucoma may be confused for transient ischaemic attacks or other neurological causes with intermittent visual loss. Between such prodromal episodes, the IOP and outflow facility are normal and there is no evidence of peripheral anterior synechiae. Subacute angle closure glaucoma may progress to acute angle closure or chronic angle closure glaucoma. The definitive treatment for subacute primary angle closure glaucoma is laser iridotomy.

Chronic Primary Angle Closure Glaucoma

Chronic primary angle closure glaucoma with pupillary block is also referred to as creeping angle closure. The condition resembles POAG in its clinical presentation with cupping and atrophy of optic discs and asymptomatic nature. Gonioscopy is the key to diagnosis and reveals narrow angles with appositional closure of peripheral iris to the trabecular meshwork in most of the circumference of the angle. IOP rises substantially only when considerable portion of the angle is closed and the height of IOP elevation is often positively correlated with the extent of the angle closure. Chronic angle closure glaucoma may also follow acute or subacute angle closure glaucoma following extensive peripheral anterior synechiae and decreased facility of aqueous outflow. The treatment for primary chronic angle closure glaucoma is laser iridotomy[18] with management of residual glaucoma by sequential combination of medications and glaucoma filtering surgery.

Follow up after Iridectomy for Angle Closure

Some eyes bear sequelae of previous attacks of angle closure, including peripheral anterior synechiae, posterior synechiae, glaucomflecken, iris atrophy, visual field loss, optic nerve pallor and cupping, cataract, decreased facility of aqueous outflow and IOP elevation. Elevated IOP in the early post iridectomy phase may be due to incomplete or closed iridectomy, plateau iris, inflammation, extensive PAS or corticosteroid administration. IOP elevation, months or years later have been reported in 24-72% eyes, following iridectomy for angle closure glaucoma. Follow up studies indicate that many eyes treated with an iridotomy alone will eventually require medication to control chronic pressure elevation and some will need filtering surgery[19]. Patients must be instructed to pursue life long follow up to eliminate residual glaucoma. IOP elevation is seen most commonly in eyes with extensive PAS. Even in eyes without PAS, appositional angle closure before iridectomy causes trabecular dysfunction from damage to outflow drainage channels. IOP elevation after iridectomy for acute angle closure glaucoma is managed by sequential medical treatment. If a combination medical therapy or maximal tolerated medical therapy fails to reduce IOP adequately, glaucoma filtering surgery is advised.

Provider and Setting

Diagnostic procedures like refraction, tonometry and perimetry may be delegated to appropriately trained and supervised para medical personnel. The interpretation of the diagnostic procedures and management of the disease is the obligation of highly trained and skilled ophthalmologists. The performance and interpretation of gonioscopy is crucial in the diagnosis and management of primary angle closure glaucoma and is also the prerogative of trained ophthalmologists which cannot be delegated to the para medical personnel. Diagnostic and therapeutic procedures including laser iridotomy are performed as out patient procedures. Hospitalisation is required for surgical intervention and also for laser procedures in individuals with advanced glaucomatous visual field loss at risk of post laser IOP spike and in patients in whom complications have occurred after laser or surgical procedures and in patients with specific medical or social needs.

Health Education, Counselling and Referral

Glaucoma is a chronic and inexorable disease with irreversible loss of visual function and requires unceasing commitment from patients to arrest further visual loss. Compliance to suggested therapy and indefinite follow up on a regular and periodic basis is crucial to success of therapy. It is not unusual for patients with advanced visual field loss and glaucomatous optic nerve damage to become depressed and despondent. The ophthalmologist need to remain sensitive to these needs and provide support and encouragement. Understanding the disease in its proper perspective and implications of suboptimal or inadequate treatment leading to inevitable blindness should be conveyed to the patient by appropriate counselling. It has been proved that health education and counselling enhances patient compliance to treatment and significantly contribute to the success of therapy. Patients with advanced visual loss and blindness should be referred to and encouraged to use appropriate low vision devices and services. Referral to a subspecialist trained in glaucoma is suggested when any aspect of diagnosis or management of glaucoma is in question or if the disease is refractory to suggested measures of treatment and management.

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Appendix I

Nd: YAG Laser Iridotomy

Although Meyer-Schwickerath first reported the use of xenon arc photocoagulator to create an iridotomy, it was not until argon laser became popular in the 1970s that laser iridotomy became clinically a practicable procedure[1]. Lasers had replaced incisional iridectomy by the end of the decade. The use of Neodynium: YAG[2] laser for performance of laser iridotomy had largely simplified this important procedure in the following decade.

Indications

Laser iridotomy is indicated in the presence of pupillary block and angle closure glaucoma. It is also indicated to prevent pupillary block in eyes at the risk of angle closure as determined by gonioscopic evaluation or because of angle closure in the fellow eye.

Pre operative Considerations

Laser iridotomy is difficult in eyes with acute angle closure glaucoma due to corneal edema, shallow anterior chamber and dilated pupil as well as inflammed iris. Medical treatment to reverse acute attack should precede laser treatment. The patient is maintained on medications to clear corneal edema and allow constriction of pupil. Significant anterior chamber inflammation may persist after acute glaucoma has subsided and it is advisable to use topical steroids for 24-48 hours before performing laser iridotomy. Pre treatment with pilocarpine facilitates easy penetration of laser by stretching and thinning the iris. Drugs like Brimonidine may blunt the post laser IOP spike which prevents further compromise in vision in individuals with advanced optic nerve damage.

Topical anaesthesia (4% lignocaine or 0.5% proparacaine) is sufficient in nearly all patients to perform laser iridotomy, although a retrobulbar anaesthetic may be required in individuals who are un- cooperative or in those with nystagmus.

Selecting the Treatment Site

Any quadrant of iris may be selected, though the superior quadrant is preferable, since it places the iridotomy below the upper lid. The 12 o'clock position is usually avoided since gas bubbles may collect and interfere with completion of the procedure. The slit beam is always positioned in a manner that laser beam is aimed away from the macula. The iridotomy is placed between middle and peripheral third of iris. If this cannot be done due to peripheral corneal haze or arcus senilis, or extremely shallow peripheral anterior chamber, a more central location is chosen, taking enough precaution to avoid the sphincter muscle. An area of thin iris or large crypt is chosen for easier penetration.

Techniques and Instrumentation

The pulsed Nd: YAG laser is the most commonly used unit for creating laser iridotomies. YAG laser generally requires fewer pulses and lesser energy than argon laser to create a patent iridotomy and the effectiveness of this laser does not depend on the iris color.

A contact lens is useful in performing laser iridotomy since it keeps the lids separated, minimises corneal epithelial burns and provides control of excessive eye movements. The convex surfaced lenses (such as the Abraham contact lens) increase the power density[3] on the iris. This contact lens further decreases the power density at the level of cornea and also beyond the target tissue (iris), so that the laser beam does not cause damage to cornea or retina.

Laser iridotomy technique with Nd: YAG laser

The extremely high energy levels and short exposure times of YAG. Lasers disrupt tissues electromechanically irrespective of pigmentation. For lasers with multiple bursts, 2-4 bursts using 2 to 6 mJ per bursts is effective in most irides. Careful focusing is important. The shockwave travels anteriorly towards the surgeon and it is suggested that the laser beam is focussed within iris stroma. This is accomplished by focusing the laser beam on the surface of iris and off setting the YAG beam so that it converges about 0.1 mm in the iris stroma. The wavelength of the Nd: YAG is beyond the visible spectrum, a helium-neon is used for focusing on the iris. Enlarging a YAG iridotomy is hazardous because of possible injury to the lens and in case the opening is inadequate, an alternative site is selected and iris perforated at an alternative site. Cases have been reported with small, but patent iridotomies, and it has been suggested that an iridotomy should at least be 150-200 microns[4] for subsequent prevention of pupillary block.

Complications

Transient IOP rise

This is the most common and potentially serious of complications with YAG[5] iridotomy. This pressure response is related to a release of prostaglandin into aqueous and a break down in blood-aqueous barrier and accumulation of plasma and fibrin in the anterior chamber angle. Risk of IOP rise may be related to the total energy delivered and pre laser reduction in facility of aqueous outflow. Topical apraclonidine 0.5% or Brimonidine 0.2% (Alphagan) is employed to blunt post laser IOP rise.

Anterior uveitis

Transient iritis occurs in all eyes due to disruption of blood aqueous barrier. Topical corticosteroids for 3-5 days is usually sufficient to control the inflammation. Marked granulomatous inflammation with hypopyon has been reported in rare cases.

Corneal damage

It include focal epithelial and endothelial burns when a large amount of laser energy is used, although they heal without sequelae. Corneal decompensation[7] have been reported when angle closure is associated with pressure elevation and inflammation, cornea guttatta, diabetes and high levels of energy delivered.

Iridotomy closure

Iridotomy may close during the first few weeks, following accumulation of pigment debris and granules. It is therefore, advisable to continue pilocarpine for the first 4-6 weeks since acute angle closure glaucomas may be precipitated with very small or imperforate iridotomies. Late closures[6] have been reported to be rare with YAG iridotomies. Patency of iridotomy openings is best ensured by visualising the lens capsule through the opening.

Hyphaema

A small amount of bleeding and microhyphaemas[2] are a common feature of YAG laser iridotomies and are not of any serious consequence, as they are usually self limiting. Bleeding during the procedure is usually found due to firm pressure on the eye with the contact lens.

Cataract

Focal anterior lens opacities are common if lens is injured during Nd: YAG iridotomy but these are non progressive. Lens changes are reported to be much less common in eyes undergoing YAG laser iridotomy[8] as compared to argon laser.

Retinal burns

These are minimised by aiming the laser beam towards the peripheral retina. Serious and permanent loss of vision has been reported due to inadvertant foveal photocoagulation[9] during laser iridotomy. Such risk of retinal injury is reduced by use of contact lenses (such as Abraham lens) while performing laser iridotomy. A case of bilateral serous choroidal and non rhegmatogenous retinal detachment has been reported following YAG laser iridotomy.

Malignant glaucoma[10]

It is not unknown after YAG laser iridotomy for chronic or acute angle closure glaucoma.

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Appendix 2

Primary Acute Angle Closure Glaucoma Management

Immediately

- Oral or intravenous osmotic agent
- 2% Pilocarpine 1 drop every 10 min for 3 doses; subsequently once 4-6 hourly
- β-blockers
- α_2 agonist (Brimonidine 0.2%)
- Topical steroids to reduce inflammation,