Diagnosis and management of common clinical presentations of primary glaucomas

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ABSTRACT

The article discusses the common causes of glaucoma, types of glaucoma, diagnosis and treatment. The article also discusses various equipment and their usage in the diagnosis of glaucoma.

KEYWORDS: Glaucoma, Intraocular pressure, Retinal nerve, gonioscopy

1. INTRODUCTION

Glaucoma is the third common cause of blindness in our country. It is estimated that, about 1.5 million Indians have glaucoma and contributes to 14% of blindness in India. It remains asymptomatic until advanced stage is reached and 90% of the people who are affected are unaware of having the disease.

History taking in glaucoma: As glaucoma is an asymptomatic disease, most patients are detected on a routine eye examination. The initial history involves the routine protocol of asking of chief complaints in the form of redness, pain watering, decreased vision, discharge as would be asked to any patient walking into your OPD. Any specific etiology contributing to the glaucoma e.g. steroid use, trauma, surgery etc.

The following points in the history taking are most pertinent:

Blurred vision with haloes: When IOP is suddenly elevated from any cause, fluid may move into cornea exceeding the capacity of the corneal endothelial pumps to remove it. Resulting corneal epithelial edema can produce symptoms of blurred vision and perception of colored haloes around lights. Colored haloes with blurred vision in the morning upon arising is a common symptom in patients with chandler’s syndrome, an ICE syndrome variant.

Blurred vision without haloes: Glaucoma may be associated with refractive errors. In some forms of secondary angle closure glaucoma such as malignant glaucoma or uveal effusion syndromes in which there is a forward shift of the crystalline lens, symptoms of myopia can occur. Rarely if the retina and choroid are elevated due to suprachoroidal fluid such as may occur after filtration surgery or in certain uveal effusion syndromes, a hyperopic shift can be observed.

Eye pain: In cases of markedly elevated IOP due to acute glaucoma attacks, etc., the patient will frequently experience sudden and severe eye pain. Eye pain may also be caused by factors such as irritation to the ciliary body resulting from corneal epithelial damage or uveitis. Patients with sustained elevation of IOP to levels of 50mm/hg or greater often complain of pain even in absence of corneal edema. Ocular pain may be accompanied by nausea and vomiting.

Subjective visual field defects: Since the chronic open angle glaucoma usually produce peripheral visual field loss, most patients are unaware of their visual field defects and do not demonstrate symptoms until the disease is far advanced.

Subjective loss of vision: With high sustained IOP in angle closure glaucoma but also in certain forms of open angle glaucoma, optic atrophy can develop with accompanying loss of visual acuity and pallor of the optic nerve without the contour changes in the disc rim that characterize the “cupping”. Commonly seen in normal tension glaucoma or open angle glaucoma associated with myopia, this is a dangerous warning sign that at a minimum, indicates the need to advance therapy to further reduce IOP.

Visual signs that can alert to the possibility of glaucoma:

Early onset presbyopia
Frequent change of glasses
Unable to read 6/6
Difficulty in night driving
Colour vision impairment: blue-yellow
Early morning or afternoon blurred vision depending on IOP Peak

Blackouts: With high levels of IOP in the 50mm/hg range or greater whether due to acute or chronic causes patients may experience blackouts. Presumably because the elevated IOP is interfering with vascular perfusion of the optic nerve or retina.

Systemic diseases:
Systemic hypertension and POAG
Thyroid disease may be associated with POAG
Diabetes has sometimes been associated with POAG
Systemic diseases may require medications such as corticosteroids.

It is also very important to probe the patient about cardiovascular disease and especially any respiratory disease that might influence one’s choice of antiglaucoma therapy.

The presence of respiratory allergies or childhood asthma.

Topical beta blockers an also have cardiac side effects. Systemic carbonic anhydrase inhibitor are relatively contraindicated in patients with respiratory or renal disease.

**Activities:** Certain activities can cause an elevation of IOP. Sleeping prone with firm eye pressure against a pillow. Sirshasana during yoga exercise or excessive breath holding during blowing of trumpet or weight lifting, also wearing tight neckties.

Large volume of water consumed rapidly do cause a transient increase in IOP in many glaucomatous eyes and this is the basis of water drinking test.

**Toxicity:** Argemone oil has been an occasional toxic contaminant of edible cooking oils. This has been suspected of causing epidemic dropsy glaucoma. This glaucoma is a reversible glaucoma this glaucoma is a reversible non inflammatory open angle. The IOP is raised and in most severe cases there is glaucomatous damage to the optic nerves.

**Systemic medications:** Atropine and many drugs having anti cholinergic atropine-like properties are dangerous in eyes that are predisposed to angle closure glaucoma.

Psychopharmacologic agents anti histaminics, anti spasmodics, anti-depressants.

Sustained periods of corticosteroid use.

**Past history of trauma:** Angle recession from trauma may be subtle, but typically leads to glaucoma years to decades later.

**Family history:** First-degree relatives of patients with POAG have a risk about 10 times greater than for people with no family history of glaucoma.

Patient with primary glaucoma should be advised to alert relatives to the need for adequate glaucoma screening and follow-up.

Asking about a family history of glaucoma as a part of an ophthalmic history, should be routine in all new patients.

First-degree relatives of patients with POAG should be advised to be screened by an ophthalmologist or optometrist.

**Approach to diagnosis of glaucoma:** Glaucoma can seldom be missed if the following parameters are carefully looked into,

a) IOP (Intra ocular pressure)

**Disc evaluation:** Optic disc evaluation includes

1) Size of disc
2) Size of cup
3) CD ratio between the two eyes
4) Neuroretinal rim
5) Nerve fibre layer
6) Peripapillary atrophy
7) Vascular changes

**Size of the disc:** It is very important while evaluating the disc. A high CD ration can be normal if the optic disc is large and low CD ratio in a small disc can be glaucomatous. In a small disc the nerve fibres being closely arranged gives the disc a pink colour and pallor can be missed. In such cases, the field may not be ordered and there is a chance to miss glaucoma.

**Size of the cup:** especially asymmetry cup between the two eyes is a common finding encountered in daily practice. Its presence should not be over looked and should be considered glaucomatous, unless proved otherwise. However it is also very important to rule out asymmetry of the optic size due to anisometropia. Between two eyes before labeling cup asymmetry of the two eyes

The margin of the cup should be determined by the bend of the small vessel across the disc rim and not by the central pallor because the anatomical contour cupping is larger than colour cupping.

**NRR (Neuro Retinal Rim):** According to ISNT rule, the width of inferior rim is more than that of superior rim and the nasal rim is more than the temporal rim, while evaluating NRR, one should always have ISNT rule in mind and check if the rim is thin or absent at any location of the disc. Thinning, pallor, and notching of the NRR are the indications of the glaucomatous damage.

**Retinal nerve fibre layer evaluation:** In healthy eyes,NFL appears opaque and glistening with radially oriented
striations, the small retinal blood vessels have a blurred appearance as they lie buried in NFL. The fundus of the affected area appears dark and deep red in contrast to the silver or opaque hue of an intact NFL. Wedge shaped defects extending up to disc margin are classical of glaucoma and easier to identify than the diffuse type of atrophy. These defects can be more easily identified by the green (red-free) light of the ophthalmoscope.

Vascular changes: The changes can be noted regarding the retinal vessels on the optic nerve head are as follows
1) Nasal shifting of vessels
2) Baring of circumlinear vessels
3) Bayoneting of vessels can be appreciated if NRR is thin or absent.
4) Splinter hemorrhages on the optic disc is an important clue to suspect glaucoma.

Peripapillary atrophy: present of peripapillary atrophy either focal or circumferential in a small non-myopic discs should be viewed with caution. A highly significant correlation has been reported between the location of the peripapillary atrophy especially medial to OD and visual field changes.

Gonioscopy: Gonioscopy is the technique to visualize the structures in the angle responsible for aqueous out-flow rate of gonioscope in the management of glaucoma is of two fold.
1) To know if angle is open or occludable (and any other angle abnormalities)
2) To know if the angle is free of pigment. PG analogues are contraindicated in pigmentary glaucoma

The structures visualized in sequence from anterior to posterior are

- Schwalbe’s line
- Anterior trabecular mesh work –lightly pigmented
- Posterior trabecular mesh work –densely pigmented
- Sclera spur
- Ciliary body band
- Iris root

Purpose: Assessment of the anatomy of anterior chamber angle by gonioscopy is an essential part in the evaluation of virtually all glaucoma. The major therapeutically in glaucoma is distinguishing open-angle from angle closure glaucoma.

Principle: In healthy eyes, the angle cannot be visualized directly because of the optical principle known as the critical angle (approximately 46 degree for cornea-air interface). Normally the light coming from the anterior chamber angle is totally internally reflected making the viewing of the angle impossible without the aid of a gonioscopic lens.

Common clinical presentations of primary glaucomas and their management:
1) Acute congestive attack of narrow angle glaucoma.
2) Patient presenting with increased IOP with clinically apparently normal disc
3) Patient presenting with normal IOP with structural OD changes
4) Patient presenting with increased IOP and structural OD changes.

Figure 1: Size of the disk

Figure 2: Glaucomatous damage optic disc cupping

Figure 3: Glaucomatous cupping
Management of acute congestive attack of narrow angle glaucoma: The most important differential diagnosis for this condition is hypertensive uveitis. Sometimes it is very difficult to differentiate these two conditions. Then the unaffected eye will help to come to a reasonable conclusion (by doing gonioscopy, in the unaffected eye, the angle should be narrow and occludable) when it is very difficult to differentiate, avoid pilocarpine and treat IOP reduction with other drugs and simultaneously treat uveitis.

Treatment to lower IOP:
- IV mannitol
- Pilocarpine 2%
- Betablockers
- Dorzolamide (Carbonic anhydrase inhibitors)
- Oral Diamox (250mg b.i.d)
- Administer 1 drop of pilocarpine in the normal eye and YAG PI
- After attack subsides YAG PI

Schiotz indentation tonometer
1) Schiotz tonometer works on the basic concept of indentation tonometry
2) The body of the tonometer has a footplate, which rests on the cornea.
3) A plunger moves freely within a shaft in the footplate and the degree to which it indents the cornea gives an estimate of the IOP.

Technique:
1) With the patient is supine position and fixing on a target just overhead the examiner separates the eye lids and gently rests the tonometer footplate on the anesthetized cornea in a position that allows free vertical movement of the plunger.
2) When the tonometer is properly positioned, the examiner observes a fine movement of the cardiac pulsations
3) The scale reading should be taken as the average between the extremes of these excursions. It is customary to start with the fixed 5.5-g weight.
4) If the scale reading is 4 or less, additional weight should be added to the plunger. A conversion table is then used to derive the IOP in mm/hg from the scale reading and plunger weight.

**Sources of error with schiotz tonometry:** Ocular rigidity-glaucoma or vitreo-retinal surgery or open globe injuries due to low scleral rigidity in these conditions. Corneal influences-steeper or thicker cornea leads to a falsely high IOP reading.

**Care of schiotz tonometer**
1) Zero error- should be checked before the day starts.
2) Cleaning in between cases-ether or sodium hypochlorite can be used to disinfect the tonometer in between cases.
3) Cleaning of barrel-should be done daily to avoid plunger sticking to the barrel.
4) Storage should be done in dry, dust free environment with separable parts separated.

**Noncontact Tonometer (NCT)**
1) The NCT was introduced by Grolman and has the unique advantage over other tonometers of not touching the eye, other than with a puff of air.
2) A pneumatic system generates a puff of air which is directed against the cornea and a detector device estimates the IOP based on reflection from flattened cornea.
3) Reliability is reduced in the higher pressure ranges and is limited by an abnormal cornea or poor fixation.corneal thickness has a greater influence on NCT readings than on goldmann tonometry.

**Goldmann Applanation Tonometer (GAT)**
1) GAT work based on the Imbert Fick principle, which states that the pressure(P) inside a sphere is equal to the force(F) necessary to flatten (P=F/A).
2) GAT is designed such that these two opposing forces approximately cancel each other when the applanated area is of 3.06mm diameter.

**How to use GAT**
1) Avoid tonometry in infected or injured eyes
2) Clean prism before first use & when indicated.
3) Disinfect prism before first use and when indicated
4) Wait adequately for the cleaned surface to dry

**Procedure:**
1) Insert the prism into the tonometer holder, ensuring the 0 to 180 marking line up with the white line on the bracket.
2) GAT is usually mount the instrument ion the slit lamp.
3) Increase the light source to maximum intensity with the cobalt blue filter on and the slit opened fully. It should illuminate the prism from the side at about 60 degree.
4) Explain the procedure to the patient. Instil a drop of anesthetic into the eyes.
5) Positioning and cooperation of the patient are vital. Ensure the patient is comfortable with the chin on the chin rest and forehead firmly against the forehead bar. Ask the patient to look straight ahead with eyes wide open.
6) Advance the slit lamp towards the eyes with the joystick. When the tip of prism is within a centimeter or so of the cornea.uses the joystick to gently bring the tip into contact with cornea under direct vision.
7) Look through the slit lamp eyepiece usually the left, is lined up with the prism. You should see two semicircles of fluorescein shifted away from each other along the horizontal axis.
8) Use the slit lamp joystick to position the semicircle at the centre of the prism. Now adjust the dial to alter the force on the prism and thus alter the size and overlap of the semicircles.
9) The end point is regular pulsation of two semic circular rings of equal size the inner edges of which just interlock.

**Common sources of error and ways to avoid them:** Patient should not hold breath during tonometry or have tight collar around neck. Terminal astigmatism of greater than 3D, the applanated area will
be elliptical, not circular. This error can be avoided by applanation at 43 degree to the axis of minus cylinder.

**Calibration error of the Goldmann tonometer:** Insert the prism in the holder and place the tonometer on the slit lamp. At setting 0, if the dial position is moved to -0.05 the feeler arm should fall towards the examiner, if the drum is moved to position+0.05 the arm should fall towards the patient. To check settings 2 and 6, the calibration error check weight bar provided by the manufacturer is used. The calibration error check weight bar has 5 markings on it. The central marking corresponds to level 0. Two on either side of it represent level 2 and the two outermost markings represent level 6. These marking correspond to 0, 20, and 60 mm/hg of IOP respectively.

**Procedure disinfect GAT prism:** Separate the tonometer probe from the prism holder/feeler arm. Rinse the prism in running cold water for 30 to 60 seconds and wipe clean. Place in a disinfectant solution of either 10% sodium hypochlorite or 3% hydrogen peroxide for 10mits. Remove the probe from the solution and place in a clean container. Rinse with cold running water for no more than an hour and no less than 10mits. Dry the probe. Use a soft, clean tissue and wipe only once in a single direction starting at the probe tip. One may simply wipe the tip of the prism with 70% isopropyl alcohol or 2% bacillocid swab. Allow sufficient time for the tip to dry.

**Perkins tonometer:** The Perkins tonometer works on the same principle as the goldmann but is portable and does not require a slit lamp, it is useful for measuring IOP in children sleeping under anaesthesia.

**Tono-pen:** The tono-pen is an applanation tonometer based on the earlier Mackay-Marg design. Its tip incorporates a stainless steel transducer which uses microstrain gauge technology to convert pressure into an electrical signal. The signal analyzed and displayed as the final IOP on a quartz crystal screen as an average of 4 readings. Tono-pen is useful for children, supine patient and when there is a gross corneal pathology.

**Dynamic contour tonometer (DCT):** The pascal DCT is a contact tonometer similar in appearance to GAT and is attached to a slit-lamp by a metal footplate, it has an integrated solid-state piezoelectric pressure sensor which permits a direct IOP measurement free of observer errors inherent in GAT. The contour of the contact surface matches the cornea during tonometry and thereby accommodates physical variations in cornea. The examiner of good contact with the anaesthetized cornea during tonometry. An average contact time of 5 seconds is usually required. During this time up to 100 measurements are taken per second and the average minimum reading is displayed on the digital LCD screen, with a Q value indicating reliability. A measure of ocular pulse amplitude (OPA) is also displayed with each IOP reading, enabling quantification of IOP fluctuations with cardiac pulse. OPA provides an estimation of ocular blood flow to the optic nerve head. DCT measurements are not significantly affected by changes in CCT, Keratometry or corneal biomechanics.

**Target IOP:** There is no standard value of IOP that can be considered safe in all patients and should aim for 20% reduction from initial pressure at which damage occurred or lower IOP to a level below 18mm of hg at all visits in cases of advanced glaucoma. The target IOP can be at a higher level in early glaucoma, short life expectancy and damage occurring at higher IOP the target IOP should be aimed at lower value in cases of glaucoma, long life expectancy and if damage occurs at lower IOP levels. Target IOP always requires modifications depending on the stability of optic nerve function.
Diagnosis procedure for Glaucoma:

Increased intraocular pressure with apparently normal optic disc

- **Gonioscop**
  - Narrow angle
    - Narrow angle glaucoma
    - Field defect
      - YAG PI medical management of IOP control
        - (+) Follow up
        - (-) Surgery
  - YAG PI

- Open angle
  - Field defect
    - (+) Ocular hypertension
      - Associated with family history of glaucoma, myopia, pseudo exfoliation, thin corneal central thickness
      - Medical management & follow up
        - Advised swap & FDT
      - Follow up
    - (-) POAG
      - Follow up not associated with risk factors
Flow chart 2. Normal intraocular pressure with structural OD changes

Flow chart 3. Increased intraocular pressure & structural OD changes
**Flow chart.4. Visual Fields**

**Pearls in medical management:**
Monotherapy 1st drug options

**Beta-blockers:** Gold standard medication if not contraindicated. Systemic examination (especially CVS and respiratory systems) is mandatory. It is better to avoid it NTG.

**Alpha-agonists:** These are generally as good as beta-lockers. Better to avoid in drivers as it induces mild sedation.

**Dorzolamide:** This is a better option especially in NTG as it got vaso-protective action.

**Prostaglandins & Prostamides:** Should be avoid in glaucoma associated with uveitis and pigmentary glaucomas.

**Pilocarpine:** Use is a must in angle closure glaucoma.

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<th>Table.1.List of IOP lowering drugs and their side effects</th>
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<tr>
<td><strong>Category</strong></td>
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<tr>
<td>--------------</td>
</tr>
<tr>
<td>Cholinergic agents</td>
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<tr>
<td>Cholinesterase inhibitors</td>
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<td>Adrenergic agonists</td>
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<tr>
<th><strong>β-Adrenergic antagonists</strong></th>
<th><strong>Timolol</strong></th>
<th><strong>Betaxolol</strong></th>
<th><strong>Levobunolol</strong></th>
<th><strong>Metoprolol</strong></th>
<th><strong>Carteolol</strong></th>
<th><strong>Carbonic anhydrase inhibitors</strong></th>
<th><strong>Acetazolamide</strong></th>
<th><strong>Methazolamide</strong></th>
<th><strong>Dorzolamide</strong></th>
<th><strong>Prostaglandin analogs</strong></th>
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<td><strong>epinephrine and its bio availability is high</strong></td>
<td><strong>Topical β adrenergic antagonist. Timolol inhibits both β₁ and β₂ adrenergic activity</strong></td>
<td><strong>Cardioselective β₁-adrenergic antagonist proved to be used in ophthalmology. Its selective β₁-adrenergic antagonist</strong></td>
<td><strong>It reduces intraocular pressure by reducing aqueous humor formation as well as by enhancing the outflow facility. Its longer duration of action makes its usage as once a day formulation</strong></td>
<td><strong>Metoprolol is a cost effective selective β₁-adrenergic antagonist</strong></td>
<td><strong>Non selective β blocker with intrinsic sympathomimetic activity. Its metabolite 8-hydroxycarteolol has better availability and increased duration of action</strong></td>
<td><strong>Acetazolamide reversibly blocks the enzyme carbonic anhydrase in the ciliary body and thereby reducing the aqueous humor production</strong></td>
<td><strong>Methazolamide is more potent than acetazolamide and it is used in the treatment of chronic open angle glaucoma with uncontrolled IOP</strong></td>
<td><strong>It is a sulfonamide lacks bacteriostatic action, with fewer side effects. Mechanism of action is similar to that of acetazolamide</strong></td>
<td><strong>Latanoprost is highly lipophilic 17-phenyl substituted PGF₂α isopropyl ester prodrug. It undergoes enzymatic hydrolysis in cornea and gets activated to the acid of lanoprost</strong></td>
<td><strong>Irritation, diplopia, ptosis</strong></td>
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2. CONCLUSION

With the advance of the technology and understanding, diagnosis and treatment of glaucoma is possible right now. We can exactly diagnose the type of glaucoma and we can treat glaucoma effectively.

REFERENCES
