

Management Tips for Glaucoma

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This article highlighted practical tips for glaucoma management. These tips include dealing with the glaucoma suspect, history taking, general medical history, gonioscopy, visual fields, optic disc cupping, secondary glaucomas, normal tension glaucoma and of course raised intraocular pressure. Emphasis being on practical tips rather than being comprehensive on glaucoma.

“Most ophthalmologists on planet earth are confused regarding diagnosis and management of glaucoma” This is a sentence which caught our attention when reading an article on glaucoma in “Highlights of Ophthalmology” some years ago. We were quite amused by this remark but over the years while working in glaucoma units abroad as well as within Pakistan we realized that there is plenty of truth in it. It has been our experience that most of the “confusion” is found in ophthalmologists of junior grade especially those in training but by no means are the seniors exempted.

We tried to analyze reasons for “confusion” and found the following to be some of the reasons for “confusion” and found the following to be some of the reasons responsible for this.

- 1 Most ophthalmologists regard an intraocular pressure of more than 21 mmHg synonymous with glaucoma and would start treatment.
- 2 Inability to appreciate other lesions or diseases which may on occasions mimic glaucoma.
- 3 Inability to appreciate pitfalls in intraocular pressure measurement.
- 4 Negligence of various diagnostic protocols for different types of glaucomas.

So we decided to write this article based on our personnel experience, teachings and experience of our mentors while working in various glaucoma units. Aim being to give practical tips rather than being comprehensive on glaucoma.

THE GLAUCOMA SUSPECT

Whenever a patient is suspected to have glaucoma do not start treatment if doubt exists regarding diagnosis¹. When dealing with suspected glaucoma it is better to investigate the patient further until things become clear rather than putting him on unnecessary lifelong treatment, since it is a common observation that once treatment is started in a particular patient, it is continued forever and subsequent ophthalmologists who manage the patient will probably continue the same management.

HISTORY TAKING

History taking is the most neglected aspect of glaucoma and ophthalmology in general since the physical findings are so apparent. However sometimes a carefully taken history will make the difference between successfully managing the disease and failure.

1. Most cases of chronic open angle are asymptomatic while acute glaucoma by contrast will present with dramatic symptoms of pain, redness and blurred vision.
2. Subacute attacks of angle closure glaucoma may present with migraine like symptoms with intermittent attacks of pain and visual disturbance.
3. A disease identical to chronic open angle glaucoma can be produced by previous trauma or steroid eye drops use so a history of eye injury, often many years previously or of eye drops use, should be sought.
4. Patients with significant myopia and glaucoma should prompt an examination for pigment dispersion as a cause of glaucoma.
5. Anisometropia and amblyopia are often associated with asymmetrical optic disc appearance, if the congenital disc asymmetry is not recognized a false suspicion of glaucoma may be raised
6. History of refractive corneal surgery has implications since eyes with corneal thinning may have erroneously low IOP readings with Goldman applanation tonometry.

GENERAL MEDICAL HISTORY

1. Bilateral adrenal hyperplasia is the only known medical condition to cause chronic glaucoma and is a very rare condition, whereas exogenous steroids are a much more frequent and unfortunately overlooked cause.
2. If patient is a hypertensive, topical Beta antagonists should not be used in those taking systemic beta antagonists².
3. If patients has a history of asthma or chronic obstructive airway disease, Beta blockers should be avoided.

DEALING WITH RAISED INTRAOCULAR PRESSURE

Intraocular pressure should never be considered in isolation.

If IOP is found to be "raised" in a particular patient, the individual either has glaucoma or patient is ocular hypertensive.

However pitfalls, in intraocular pressure measurement should be excluded in suspected glaucoma patients' .e.g. Check central corneal thickness³.

1. Before labeling a patient as ocular hypertensive, secondary glaucoma must always, be excluded. This is because disc cupping and visual field loss are not absolutely essential to diagnose secondary glaucoma.
2. Ocular hypertensive should have yearly fields done to check for development of glaucoma.
3. IOP is genetically determined. Therefore a difference of more than 5mmHg between the two eyes should be viewed with suspicion even if IOP is within 'normal' range.

GONIOSCOPY

One of the most common causes of an incorrect diagnosis is the omission of gonioscopy. The reasons offered is that if slit lamp examination does not suggest a narrow angle, ocular inflammation, new vessel formation or signs of previous trauma, the patient must be having open angle glaucoma. Chronic angle closure glaucoma and many other forms of glaucoma can therefore be overlooked.

VISUAL FIELDS

1. Before interpreting fields one must always refer to the reliability indices.
2. Fields with significant errors in reliability indices should be repeated if possible.
3. The most important scotomas in glaucoma appear in the Para central areas or the Bjerrum's area. Therefore a visual field with only "edge effects" type of Scotomas should be considered artifactual and fields should be repeated.
4. Visual field which is normal (in the presence of reliable reliability indices) should be taken as correct and need not be repeated.
5. A scotoma which obeys the vertical meridian almost always points to a neurological pathology rather than glaucoma.
6. Always remember that no field defect is pathognomonic of glaucoma. The field defects which are typical of glaucoma can also be produced by lesions such as anterior ischemic optic neuropathy, branch retinal vein occlusion etc
7. Purely central scotomas with preservation of peripheral fields are suggestive of neurological pathology rather than glaucoma e.g. optic neuritis.
8. Rapidly progressive field loss and markedly asymmetric field loss also raise the possibility of neurological disease rather than glaucoma.

9. As part of learning curve or fatigue patient may have non specific field defects.

If such is the case repeat fields and artifactual scotomas will disappear.

OPTIC DISC CUPPING

1. Cup disc ratio (C/D) is genetically determined; therefore an asymmetry of greater than 0.2 between the two eyes should be regarded with suspicion⁴.
2. Optic disc cupping is not unique to glaucoma only and can also occur in conditions such as compressive optic neuropathies, anterior ischemic optic neuropathies etc⁵.
3. Cupping can also be seen in elderly individuals with atherosclerosis even if there is no definitive field loss⁶.
4. Optic disc colobomas are very frequently confused with pathological cupping especially because they also produce significant field defects.
5. Pallor involving cup as well as the neuroretinal rim suggests a neurological cause of cupping rather than glaucoma⁷. In glaucoma, the remaining neuroretinal rim retains its normal pink colour.

SECONDARY GLAUCOMAS

1. Generally speaking there are two glaucomas where the diagnosis is not essentially based on visual fields
 - a. Angle closure glaucoma.
 - b. Secondary glaucomas
2. For the diagnosis of secondary glaucomas intraocular pressure almost always has to be raised.
3. Raised IOP in presence of other ocular evidence of secondary glaucoma is enough to make a diagnosis even in the presence of healthy discs and fields. Of course fields will be indicated for follow up of disease progression or to judge efficacy of treatment.
4. The above points will be highlighted by the following example.

A 45 years old lady came to our out patient clinic. She was being observed for glaucoma as we noticed from her file notes. Her vision was 6/6 with -2.50 DS each eye. Her IOP was 22mmHg and 24mmHg in the right and left eye respectively as we observed from her notes. When we took the IOP, almost similar readings appeared. She was presently not on any medication. Her optic discs

were healthy looking and her visual fields were completely within normal limits. However, the decision of starting the antiglaucoma treatment remained controversial. When we examined the patient on slit lamp we noticed that the patient had classic Krukenberg spindles on the posterior aspects of both corneas and anterior chambers were deep in both eyes. Gonioscopy revealed open angles with marked pigmentation in the trabecular meshwork. Water drinking test was performed and within an hour the IOP shot from 22 mmHg and 23 mmHg in the right and left eye respectively to 37 mmHg and 39 mmHg in the right and left eye respectively. This rise in IOP was dramatic and could not be ignored. It showed compromised facility of outflow. A diagnosis of Pigment Dispersion Glaucoma was made and we put her on treatment in spite of the fact that fields were normal. However we did advise her to get fields done on yearly basis in order to judge effectiveness of the treatment.

5. Whenever you diagnose "uniocular" POAG, beware, you probably are dealing with a secondary glaucoma in which signs are subtle and have been missed. i.e. exclude secondary glaucoma before diagnosing uniocular POAG.
6. Patient who has traumatic angle recession should be followed up for the rest of their lives even if IOPs are normal since pressure may rise later on.
7. Any eye with intraocular inflammation should be presumed to be having raised IOP until proved otherwise.

NORMAL TENSION GLAUCOMA (NTG)

1. The biggest mimicker of NTG is Primary open angle glaucoma itself.
2. If you diagnose NTG on the very first visit of a patient the chances of the diagnosis being correct are minimum.
3. If NTG is considered a diagnosis of exclusion, chances of making mistakes in diagnosis are minimized.
4. Common conditions which are likely to cause errors in diagnosis are:
 - a. Pitfalls in IOP measurement.
 - b. Optic disc anomalies e.g. colobomas
 - c. Neurological lesions producing optic disc cupping.
 - d. Misinterpretation or wrongly performed visual fields.
5. If patient has typical glaucomatous cupping with field defects and "normal" IOP, perform water

drinking test to see how much IOP rises. In true NTG rise in IOP should not be significant.

6. In case water drinking test is negative it is prudent to perform neuroimaging of brain and visual pathways before finally labeling patient as having NTG.

APHAKIC GLAUCOMA AND GLAUCOMA IN APHAKIA

Aphakic glaucoma is a definite disease entity while glaucoma in an aphakic eye may have many reasons and mechanisms.

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