Target Intraocular pressure

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For a long time in ophthalmic history, intraocular pressure (IOP) and glaucoma were synonyms. In the1996 edition of the American Academy of Ophthalmology, preferred practice pattern for primary open angle glaucoma (POAG), elevation of IOP is neither a component of the definition of POAG nor a clinical characteristic of it. IOP has been re-classified as a risk factor for the clinical process, rather than a clinical feature of POAG.

For simple office practice and for a general opthalmologist measuring IOP together with optic disc fundoscopy remain the handy tools for diagnosing and following up cases of glaucoma. Measuring and evaluation of IOP should be considered as an input and processed as a quality concept to achieve a target value.

Input

Processing _____ Target

Output

The clinical disease course of some glaucoma variants can support the non directional relationship of glaucoma and IOP as low as normal tension glaucomas, while glaucomatous optic neuropathy is a direct evidence of mechanical insult of the optic nerve head by elevated IOP.

Rationale of elevated IOP as a risk factor in glaucoma

The exact etiology is uncertain but we do have some substantial evidence that support this direct relationship.

- Aqueous outflow is lower than aqueous inflow.
- There is a definite site of resistance of aqueous flow.
- The site of resistance is of an indefinite location.
- There is definite genetic involvement (TIGR gene and/or its product).

The evidence of outflow resistance is a characteristic of glaucoma associated with some ocular pathologies as an exfoliative and pigmentation affections.

So for each patient with glaucoma there should be a target IOP that directly involves the management of the case.

All target IOP values should go low, lower than the initial baseline IOP.
The question remains. How low should we go?

There are no good data for target IOP, and this is well evidenced in normal tension glaucoma and during the course of all glaucomas the higher IOP the worse is the clinical course and prognosis.

So our aim in cases with high IOP alone is to lower IOP 20% than base line (usually <24 mmHg).

We should lower more in patients with high tension and field defects and GON lower IOP >30% than base line (usually <15mmHg).

These numbers are applicable to most glaucoma cases but should not be taken as dogmatic values; they should be low if the fellow eye is damaged by GON.

Target IOP is a dynamic value and can change from one event of the disease to another; they can change by time and even change diurnally. So we should have a range for target IOP not a fixed value.

Glaucoma practicing doctors should always be pessimistic because glaucoma is a chronic disease with no definite cure; it is not life threatening (even with malignant glaucoma!) so never give flowery prognosis and never give a fixed IOP level to be safe for the patient.

Monotherapy usually achieves 20% reduction of IOP if not change to another monotherapy treatment.
Adjunctive therapy should add more than 15% reduction in IOP. We should put in consideration the phenomenon of long term drift for chronic use of any drug that is the effect decreased by time.
Drug interaction can add negative impact on the results obtained.

Target IOP is inversely proportional to years, not months, in well controlled patients, when target IOP is achieved the story begins:

- ❖ Tracking the structural and functional status of ON:
- OCT basis and yearly.
- Sterioscopic OHN pictures basic and yearly.

- ❖ Visual function:
- Reliable achromatic field plotting basis quarter yearly then half yearly.
- SWAP field (very early).

References and further readings

- American Academy of Ophthalmology primary open angle glaucoma, preferred practice pattern, San Francisco, 1996.

- Canadian Ophthalmological Society Annual Meeting, Hull, Quebec, June 13 – 16, 2002.

- Leske, MC:
  Early manifest glaucoma trial. Design and baseline data.
  Ophthalmology, 106:2144-53, 1999.

- Quigley, H; Addicks, E and Green W:
  Optic nerve damage in glaucoma, quantative correlation of nerve fiber loss and visual dfields defects in glaucoma cases.
  Arch. Ophthalmol., 100: 135, 1992.

- Quigley, H and Vitale, S:
  Model of glaucoma prevalence and incidence in the United States,
  Invest. Ophthalmol. Vis. Sci., 38: 83-91, 1997.

- Odelberg, T:
  Visual field prognosis., in advanced glaucoma, Acta Ophthalmol.,
  65, Suppl., 27-29, 1997.