Glaucoma – Differential diagnosis and management

DESCRIPTION, SYMPTOMS, CLASSIFICATION AND SIGNS
See Glaucoma classification.

PREVALENCE AND SIGNIFICANCE
Glaucoma is one of the leading causes of visual disability in Western countries, along with macular degeneration, diabetic retinopathy and cataract. One study of elderly Australians estimated the prevalence of glaucoma at 3.5 per cent.

DIFFERENTIAL DIAGNOSIS AND OTHER OPTIC NEUROPATHIES
● Other optic neuropathies – Arteritic ischaemic optic neuropathy (AION) (giant cell arteritis), Non-arteritic ischaemic optic neuropathy (NAION)
● Compression of visual pathways eg pituitary tumour
● Congenital anomalies – Optic nerve pit, Optic nerve drusen, Coloboma, Morning glory syndrome, Tilted disc, Physiological cupping
● Acquired anomalies – Toxic optic neuropathy, Traumatic optic neuropathy.

SEE ALSO

MANAGEMENT
The presence of glaucomatous signs is an indication for treatment, whereas the presence of risk factors is an indication for advice and review. Glaucoma is most often treated by a therapeutic reduction in intraocular pressure (IOP).

Urgent
● Angle-closure glaucoma with excessive IOP is a medical emergency, requiring treatment within hours. If IOP is over 40mmHg, then IOP reduction is urgent and all topical glaucoma medications should be used, subject to the medication contraindications and precautions. Check IOP every 10-20 minutes and treat both eyes. In acute angle-closure, with IOP over 45 to 50mmHg, oral or intravenous (IV) agents may be helpful in treatment, such as carbonic anhydrase inhibitors eg acetazolamide 500mg and IV hyperosmotics such as mannitol. Systemic contraindications and precautions must be observed
● Open-angle glaucoma with excessive rise in IOP is a medical emergency, requiring treatment in 24-30mmHg, although the more recent guidelines favour the lower end of the range, particularly when other risk factors are present, including positive family history, increased optic disc cupping, systemic risk factors, or a thin cornea (Ocular hypertension treatment study).

Topical medication
The early manifest glaucoma trial (EMGT) had an untreated control arm and proved conclusively that medical treatment delays glaucomatous progression. First-line medical treatment in recent years has become the prostaglandin-analogues eg Bimatoprost, Latanoprost or Travoprost qd nocte or Unoprostone bid. Prior to this the beta-blockers, eg Timolol maleate, Betaxolol, Carteolol, Levobunolol, Mitipranolol qd mane or bid were the mainstay of treatment, although caution was required in patients with systemic conditions, including cardiac and pulmonary disease. Other topical drug categories available include adrenergic agonists, carbonic anhydrase inhibitors and cholinergic (miotic) medications. See the appendix for more details. Secondary glaucomas are usually treated according to the underlying cause.

Laser surgery
Argon laser trabeculoplasty (ALT) is considered equally effective as medical therapy for POAG (Collaborative initial glaucoma treatment study and glaucoma laser trial). Its efficacy depends on the degree of pigment in the angle. Furthermore, ALT has a higher initial success rate than medical therapy in pseudoexfoliation glaucoma. However, the improvement may not be permanent and continued observation will be necessary. Selective laser trabeculoplasty (SLT) is a newer technique than ALT, but with less data available. A YAG laser peripheral iridotomy is a definitive treatment for angle-closure glaucoma.

Incisional surgery
Trabeculectomy filtration surgery is beneficial in lowering IOP when medical treatment fails (Advanced glaucoma intervention study, AGIS).

Refractive correction or LVAs
Consider low-vision referral for advanced glaucoma.

Additional procedures
Fundus photos or scanning laser ophthalmoscopy can help identify optic nerve head changes. Ocular coherence tomography and nerve fibre layer analysers enable assessment of the nerve fibre layer.

Reviews
Glucoma and glaucoma suspect patients are usually reviewed at six- or 12-month intervals, the aim being to identify any change in the IOP, visual fields, nerve fibre layer or optic nerve.

The full series of these articles will be available in the book Posterior Eye Disease and Glaucoma A-Z by Bruce AS, O’Day J, McKay D and Swann P. £39.99 For further information click on the Bookstore at opticianonline.net

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