Glaucoma
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Glaucoma is the second leading cause of blindness in the United States and the leading cause of blindness in African Americans. Early detection and treatment can prevent blindness.

Glaucoma as we understand it today represents a group of disease entities which have as a final common pathway damage to the optic nerve and as a consequence, visual field loss. These changes can result in blindness. Traditionally, the glaucomas were classified simply as primary, secondary, combined or congenital. However, it is now clear that glaucoma is a much more complicated disease.

In recent years, the study of molecular genetics has opened new areas of understanding glaucoma. Juvenile open angle glaucoma, primary infantile glaucoma and other developmental glaucomas have been determined to have a molecular basis. With increasing knowledge in these areas, the classification of the glaucomas will be partially based on the findings of the defective gene or gene products. Primary open angle glaucoma, the most common form of glaucoma, may eventually be subdivided genotypically into many different diseases.1 In these cases, gene therapy may offer a new treatment modality.

Glaucoma is the second leading cause of blindness in the United States and the leading cause of blindness in African Americans. The most common form of glaucoma, primary open angle glaucoma, is a major health problem with 2 million Americans having this disease. Half of these Americans may not be aware that they have this disease.2 More than 7 million office visits occur per year with the primary diagnosis to monitor patients with glaucoma and patients at risk for developing glaucoma.3 About 80,000 Americans are legally blind from glaucoma and many more have severe visual impairment.

Primary open angle glaucoma accounts for 70% of the glaucoma cases in the United States.4 Important risk factors include the level of intraocular pressure, race and age. The prevalence of primary open angle glaucoma increases with increasing intraocular pressure. Primate studies and observations in angle closure and secondary glaucomas provide good evidence that elevated intraocular pressure can lead to optic nerve damage. However, significant individual variation exists in correlating the optic neuropathy and intraocular pressure. Studies show only one-tenth or less of those with elevated intraocular pressure have glaucomatous field loss.5 About one-sixth of patients with glaucomatous optic nerve damage with field loss have less than 21 mmHg intraocular pressure even after repeated measurements.

Several factors may cause the progressive optic neuropathy of primary open angle glaucoma besides the intraocular pressure but the intraocular pressure is treatable and therefore treatments have included medications, laser surgery or other surgical treatments. This is a reasonable approach to therapy for this condition because progressive field defects occur in patients with higher intraocular pressures.6

African Americans have a prevalence of 4 to 5 times greater than other races, making race an important risk factor in primary open angle glaucoma. Compared to Caucasian Americans, blindness due to glaucoma is 4 to 8 times greater in African Americans.7 Japanese appear to have a higher prevalence of low tension glaucoma.

Age is another important risk factor for primary open angle glaucoma. The Baltimore Eye Survey demonstrated this. African Americans 80 years or older have a prevalence exceeding 11%.

Family history of glaucoma is a risk factor. The risk of developing glaucoma is at least 3 times greater in patients who have a sibling with primary open angle glaucoma.

Other risk factors which have not shown consistency include cardiovascular disease, hypertension, diabetes and myopia.7

Besides measuring intraocular pressures, screening of patients for primary open angle glaucoma must include an assessment of the optic nerve status. This can be done by direct observation of the optic nerve or nerve fiber layer or by an analysis of the visual fields. Visual field defects may not be noted until one-third or more of the optic nerve fibers have been destroyed. Glaucoma can be suspected if the intraocular pressure exceeds 21 mmHg or the vertical cup/disc ratio of the optic nerve head is greater than 0.5. Intraocular pressure measurement alone cannot be relied on to make the diagnosis of primary open angle glaucoma. Therefore, probably the more efficient way to detect and diagnose primary open angle glaucoma is a routine periodic comprehensive eye examination.3

The examination for glaucoma must include the family history, ocular history and systemic disease history. A history of prior eye injury or diseases may give clues to the etiology of the glaucoma. A strong family history might suggest a genetic link while the presence of some systemic diseases like pseudoexfoliation syndrome might correlate with the presence of glaucoma.

The ocular examination must include the recording of visual acuity. The pupillary responses must be checked for any afferent pupillary defect. A Goldmann type applanation tonometer is preferable to measure the intraocular pressure. The time of the pressure measurement should be recorded as diurnal variations may influence the pressure measurements. Sometimes diurnal measurements may be necessary in those cases where the severity of the optic nerve damage does not correlate with the normal or less than normal intraocular pressure.

The slit lamp examination of the anterior ocular segments, gonioscopy to determine open or narrow angle, optic disc and nerve fiber layer evaluation and fundus examination are part of the glaucoma evaluation. (Fig. 1) The optic nerve appearance can be described but photographs of the optic nerve head and if available, stereophotography, are preferred methods of documenting the optic nerve head.
congenital and infantile glaucoma. In this sym-
ptoms can be treated. Ocular developmental ab-
normalities can result in glaucoma. A new vessel formation may result from diabetic retinopathy or occlusive disease. The use of steroids can result in occlusions and long-term use of steroids. Retinal ischemia may develop, which may result in the eyes developing acute glaucoma. A prophylactic laser iridotomy is done on the unaffected eye. Glaucomas with related ocular conditions are termed secondary glaucomas. These include trauma, uveitis, diabetic retinopathy, vein occlusions and long-term use of steroids. Retinal ischemia as a result of diabetic retinopathy or occlusive disease may result in new blood vessels growing into and obstructing the trabecular meshwork. This can result in neovascular glaucoma, a difficult glaucoma to treat. Ocular developmental abnormalities can lead to congenital and infantile glaucoma. In this group, symptoms can
include corneal enlargement, epiphora, photophobia, blepharospasm, and corneal edema. The preferred treatment in congenital and infantile glaucoma is surgery. Medical management usually has a limited long-term value.

In summary, glaucoma is a unique ophthalmic disease. Before treatment, the patient may have been totally asymptomatic. Major effects on the quality of life can occur once therapy is initiated. Medical therapy may be life long but untreated, the disease in its natural course may result in blindness. Surgery can be offered to patients if the disease progresses and the patient becomes intolerant to medications. An ongoing assessment of the quality of life is an essential portion of the management of the glaucoma patient.

References