



Mechanism of Glaucoma

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DESCRIPTION

Glaucoma is a collection of eye illnesses which bring about harm to the optic nerve and imaginative and prescient loss. The glaucoma, wherein the drainage perspective for fluid inside the attention stays open, with much less unusual place sorts along with closed-perspective glaucoma and normal-anxiety glaucoma. Open-perspective glaucoma develops slowly over the years and there may be no pain. Peripheral imaginative and prescient might also additionally start to decrease, accompanied by vital imaginative and prescient, ensuing in blindness if now no longer treated. The surprising presentation might also additionally contain intense eye pain, blurred imaginative and prescient, mid-dilated pupil, redness of the attention, and nausea. Vision loss from glaucoma, as soon as it occurs, is permanent. Eyes tormented by glaucoma are known as being glaucomatous.

SIGNS AND SYMPTOMS

Due to the silent nature of glaucoma, patients usually exhibit some symptoms or visual impairment later in the course of the disease, especially in primary open-angle glaucoma. However, angle-closure glaucoma and secondary glaucoma can cause a sharp rise in intraocular pressure, which is usually symptomatic, especially when the intraocular pressure is above 35 mmHg.

The patient's medical history should pay particular attention to the following:

- Past eye history
- Previous ophthalmic suge, including phocoagulation or refractive surgery
- Eye / head trauma
- Previous medical history
- Current medicine
- Risk factors for glaucomatous optic neuropathy

PATHOPHYSIOLOGY

The exact cause of glaucomatous optic neuropathy is unknown, but many risk factors have been identified, including elevated

intraocular pressure, family history, ethnicity, age 40 and older, and myopia. Elevated intraocular pressure is the most important clinically treatable risk factor for glaucoma. There are several theories that intraocular pressure may be one of the factors that cause glaucoma injury in patients. Two of the main theories are: (1) Onset of vascular dysfunction that causes optic nerve ischemia, and (2) Mechanical dysfunction due to compression of the lamina cribrosa. Several years of research have shown that when IOP exceeds 21 mm Hg, the proportion of patients who develop visual field loss, especially at pressures above 2630 mm Hg increases rapidly. Patients with IOP greater than 28 mmHg are about 15 times more likely to develop. The population of patients with elevated intraocular pressure should not be considered uniform, as they are more likely to experience visual field loss than patients with a pressure of 22 mm Hg. In addition, the following factors should be considered in relation to the achieved IOP value before starting treatment for a patient based on a particular IOP measurement.

- Fluctuations in tonometry readings from inspector to inspector (usually seen at about 10% or 12 mm Hg)
 - Effect of corneal thickness on IOP measurement accuracy (see other tests)
 - Diurnal fluctuations in intraocular pressure (often peaks early in the morning but can reach intraocular pressure at any time)
- Disc cutting and loss of up to 40% of nerve fiber layer. The actual loss of vision that has been demonstrated will be. Therefore, visual field tests are not the only tool to determine when a patient begins to experience undeniable glaucoma damage. Also, it should not be used alone as a standard of treatment. Increased IOP is generally associated with reduced opportunities for water spills adopted.
- The occurrence of this increased drug is suggested by several theories, including:
- Blockage of trabecular meshwork due to accumulated material
 - Loss of trabecular endothelial cells
 - Decrease in pore density and size of trabecular in the endothelium of the inner wall of Schlemm's canal
 - Loss of giant vacuoles in the endothelium of the inner wall of Schlemm's canal

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- Loss of normal phagocytosis
- Interference with neurological feedback mechanism

Other processes believed to play a role in exudate resistance include altered corticosteroid metabolism, dysfunction of adrenergic control, abnormal immunological processes, and oxidative damage to networks. Many other undetermined factors are thought to play a role in the etiology of glaucoma. Basic and clinical studies continue to play a role in the search for such factors that contribute to the onset and prognosis of POAG patients.

Diagnosis

Screening for the general population of primary open-angle glaucoma consists of intraocular pressure measurements, especially in high-risk individuals such as African Americans and the elderly, where screening is combined with an assessment of optic nerve status. Assessments of patients with suspected primary open-angle glaucoma include:

- Anterior eye slit lamp examination
- Fundus examination
- Intraocular pressure measurement
- Gonioscopy
- Thickness measurement

Clinical examination

Laboratory tests that can be used to rule out other causes of optic nerve damage in patients with suspected normal-tension glaucoma include:

- CBC count
- ESR
- Syphilis serology (Microhaemagglutination Treponema pallidum [MHATP], not the Sexually Transmitted Diseases Research Institute [VDRL] test)
- Rarely serum protein electrophoresis: for individuals with possible autoimmune etiology of some glaucomatous optic neuropathy

Imaging research

The following imaging tests can be used to evaluate patients with suspected primary open-angle glaucoma.

- Fundus photo
- Depiction of the retinal nerve fiber layer on high-contrast black-and-white film using red-free technology
- Confocal scanning laser scanning ophthalmoscope
- Scanning laser polarization measurement
- Optical coherence tomography
- Neuroimaging as suggested by the patient's visual field loss pattern

Imaging modality in the evaluation and management of patients with primary open-angle glaucoma includes:

- Fluorescein angiography
- Ocular perfusion analysis using a laser Doppler velocimeter
- Color blindness measurement
- Contrast sensitivity test
- Electrophysiological test
- Ultrasound biomicroscopy