Why glaucoma prevalent in Asia

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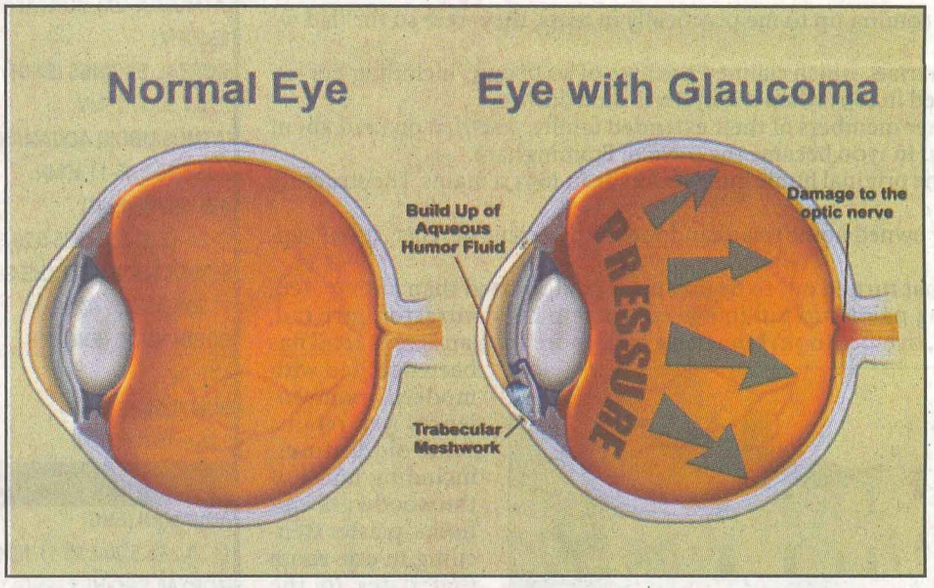
AFTER cataract glaucoma is the main cause of blindness worldwide, with Asians accounting for approximately half of the world's population. Zainal and associates in a National Eye survey Malaysia, among 18, 207 residents examined found that the age adjusted prevalence of blindness and low vision was 0.29 per cent and 2.44 per cent respectively. Reddy and colleagues in a study of 311 rural populations aged 40 years and above in Selangor, reported the prevalence of impaired vision was 0. 29 per cent and 2. 44 per cent respectively. Reddy and colleagues in a study of 311 rural populations aged 40 years and above in Selangor, reported the prevalence of impaired vision was 18.9 per cent and blindness 2.9 per cent. A detailed study of 150 patients revealed the common causes of visual impairment and blindness were refractive errors, cataract, glaucoma, diabetic retinopathy and age related macular degeneration in order of frequency. Researchers in a Singapore Malay eye study, a population based cross-sectional survey that examined 3, 280 participants [aged 40-80 years]. Of the 3, 280 participants 150 (4.6 per cent) had diagnosed glaucoma. The prevalence of glaucoma among the Malay population 40 years of age and above older in Singapore is 3.4 per cent, comparable to ethnic Chinese and other ethnic groups in Asia.

Origins

The word "glaucoma" is from ancient Greek *glaukos* which means blue, green or grey. Glaucoma is a group of eye diseases which result in damage to optic nerve and vision loss. As of 2010, there were 44.7 million people in the world with open angle glaucoma. By 2020, the prevalence is projected to increase to 58.6 million worldwide and 3.4 million in the United States. Both internationally and in the US, glaucoma is the second-leading cause of blindness. Globally cataract are a more common cause. Glaucoma is also the leading cause of blindness in African Americans, who have high rates of primary open angle glaucoma. Bilateral vision loss can negatively affect mobility and interfere with driving. The most common type is open angle glaucoma with less common types including closed angle glaucoma and normal tension glaucoma. Open angle glaucoma develops slowly over time with no pain. Closed – angle glaucoma can present gradually or suddenly. The sudden presentation may involve severe eye pain, blurred vision, mid-dilated pupil redness of the eye and nausea. Vision loss from glaucoma, once it has occurred, is permanent. Contributory factors for glaucoma include increased pressure in the eye, a family history of condition and high blood pressure. For eye pressure a value of greater than 21mmHg or 2.8 kpa is often used with higher pressure leading to greater risk. Some individuals with high eye pressure for years and never develop damage. Conversely, optic nerve damage may occur with normal pressure, known as normal tension glaucoma. Laser treatments may be effective in both open and closed-angle glaucoma. Treatment of closed-angle glaucoma is a medical emergency.

Contributory factors

There are several contributory factors (causes) for glaucoma, ocular hypertension (increased pressure within the eye), is the most important risk factor in most glaucoma, but in some populations, only 50 per cent of people with primary open-angle glaucoma actually have elevated ocular pressure. Open-angle glaucoma accounts for 90 per cent of glaucoma cases in the United States. Closed-angle accounts for less than 10 per cent of glaucoma cases in the United States but many as half of glaucoma cases in the other nations (particularly East Asian countries. There is no clear evidence indicates vitamin deficiencies cause glaucoma in humans. It follows, then that oral vitamin supplementation is not a recommended treatment for glaucoma.) Caffeine increases intraocular pressure in those with glaucoma, but does not appear to affect normal individuals.



Role of ethnicity: Many people of East Asian descent are prone to developing angle-closure glaucoma due to shallower anterior chamber depths, with the majority of the cases of glaucoma due to shallower anterior chamber depths, with the majority of the cases of glaucoma in this population consisting of some form of angle closure Higher rates of glaucoma have also been reported for Inuit (people living in Siberia, Canada, Alaska and Greenland) populations compared to white populations in Canada and Greenland.

Role of genetics: Individuals with family history of glaucoma are at higher risk. The relative risk of having primary open angle glaucoma (POAG) is increased about two-to-four fold for people with a sibling with glaucoma. Glaucoma, particularly POAG, is associated with mutations in several genes, including MYOC, ASB10, WDR56, NTF4, TBK1 and RPGRIP1 although most cases of glaucoma do not involve these genetic mutations. Normal tension, which compromise one third of POAG, is also associated with genetic mutations, including PA1 and OPTN genes. Various rare congential/ genetic eye malformations are associated with glaucoma. Occasionally, failure of the normal third trimester gestational atrophy of the hyaloid canal and tunicavasculosa lentis is associated with other anomalies. Angle closure-induced ocular hypertension and glaucomatous optic neuropathy may also occur with these anomalies and has been modelled in mice.

Disease progression

The underlying cause of open angle glaucoma remains unclear. Several theories exist on its exact etiology. However, the major risk factor for most glaucoma and the focus of treatment is increased intraocular pressure. Intraocular pressure is a function of production of liquid aqueous humour by the ciliary processes of the eye, and its drainage through the trabecular meshwork. Aqueous humour flows from the ciliary processes into the posterior chamber, bounded by the lens and the zonules of Zinn, and anterior of the iris. It then flows through the pupil of the iris into anterior chamber, bounded posteriorly by the iris and anteriorly the cornea. From here the trabecular meshwork drains aqueos humour via the scleral venous sinus (Schlemm's canal) into scleral plexuses and general blood circulation.

In open/ wide angle glaucoma, flow is reduced through the trabecular meshwork, due to the degeneration and obstruction of the trabecular meshwork, that original function is to absorb the aqueous humour. Loss of aqueous humour absorption leads to increased resistance and, thus, chronic, painless build-up of pressure in the eye. In close/ narrow angle iridocorneal angle is completely closed because of forward displacement of the final toll and root of iris against the cornea, resulting in the inability of the aqueous fluid flow from the posterior to the interior chamber and then out of the trabecular network. This accumulation of aqueous humour causes an acute increase in pressure and pain.

The inconsistent relationship of glaucomatous optic neuropathy with increased intraocular pressure has provoked hypotheses and studies on anatomic structure, eye development, nerve compression trauma, optic nerve blood flow, excitatory neurotransmitter, trophic factor, retinal ganglion cell/ axon degeneration glial support cell, immune system, aging mechanism of neuron loss, and severing of the nerve fibres at the scleral edge.

This series on glaucoma continues next week with a look at the signs, symptoms and diagnosis for the eye problem.

About the authors

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