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Editorial

An Overview of Glaucoma

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Editorial

Glaucoma is a disease which is caused owing to- (i) optic neuropathy (ii) specific pattern of visual field defect and (iii) raised Intraocular Pressure (IOP) from 21mmHg. It is third most prevalent cause of visual impairment and blindness. Approximately more than 70 million people worldwide and 7 million estimated blind [1], therefore, it is the leading cause of irreversible blindness in the world. It can remain asymptomatic until it is severe, resulting in a high likelihood that the number of affected individuals is much higher than the number known to have it [2]. It is a multifactorial disease in which both genetic and environmental factors involved. Therefore, it is necessary to deepen inside the influence of polymorphisms of each gene with the disease and analyze the gene-gene and gene-environment interactions [3]. There is a diverse variety of glaucoma, inspite of which, Primary Open Angle Glaucoma (POAG) and Angle Closure Glaucoma (ACG) are most common. Myocolin(MYOC), Optineurin(OPTN),CYP1B1 (GLC3A), WDR36, GLC1D, GLC1F, GLC3B gene mainly involve in the pathogensis of glaucoma [3].

Not much known about the pathogenesis of disease, except POAG and ACG. In POAG, Obstruction occurs at the level of the Trabecular Meshwork (TM), therefore IOP increases (> 21 mmHg). Progressive loss of visual field manifests (due to optic nerve damage), over time from periphery to center and presence of hollow optic disc ('cupping'), due to retinal ganglion cell death [4]. However, in ACG, Apposition (contact) of iris and TM so the angle between iris and cornea (iridiocorneal angle) become narrows or closes, therefore obstructs outflow of the aqueous humor from the eye. If pressure rapidly build up in the eye, causing pain and redness (symptomatic or acute angle closure) [4]. The process of apoptosis and apoptotic factories/factors also triggers pathogenesis of glaucoma [5]. The p53gene, Bax, Bcl-2, Caspase 3, 7, 9, cytochrome-c, Poly ADP Ribose Polymerase 1 (PARP1) etc. involve in pathogenic processes of glaucoma in the retina and optic nerve [5].

Techniques/testing used for detection of glaucoma includes- Tonometry (inner eye pressure testing), ophthalmoscopy (dilated eye examination), perimetry (visual field test), gonioscopy (measure angle in the eye where the iris meets the cornea), pachymetry (Thickness of the cornea), nerve fiber analysis[6,7]. Therapeutics includes drug and surgical treatment (Argon laser trabeculoplasty, trabeculectomy, filtering procedure, cyclocryotherapy, iridotomy). Several drugs like β-blockers (levobunolol, timolol, carteolol, betaxolol), α-2 adrenergic agonists (apraclonidine, brimonidine), carbonic anhydrase inhibitors (acetazolamide, dorzolamide)-used for decrease in aqueous production/IOP [4,8,9]. On the other hand drug those increase aqueous outflow include- nonspecific adrenergic agonists (epinephrine, dipivefrin), parasympathomimetics (pilocarpine, carbachol, echothiophate), prostaglandins (latanoprost) [8,9].

The application of translational research is to integrate advances in clinical trials and research from the bench to the bedside [10]. It is more useful methods that allow a better application of basic research based knowledge to the medical field including the area of ophthalmology.

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