■ *Guest editorial*

GLAUCOMA MANAGEMENT: A PUZZLE

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It is, perhaps, still a common and widespread understanding that primary open angle glaucoma is associated with a significantly raised intra-ocular pressure (IOP). However, encountering a patient with normal IOP and even below the mean value in association with deteriorating glaucomatous optic neuropathy and progressive visual field loss is not unusual in ophthalmic practice[1]. Similarly, we have seen individuals whose visual field remains intact despite the high IOP of around 30 mmHg. The glaucoma, therefore, is something more than just a raised IOP.

Because of its typical appearance, the glaucomatous optic atrophy can be distinguished from the other causes of optic neuropathy by stereoscopic view of the fundus of the eye. It is characterized mainly by progressively deepening cup of the optic disc with thinning of the neuro-retinal rim. The appearance of the disc is almost the same in glaucoma with or without a raised IOP. It is, therefore, important to look for other risk factors responsible for uncontrolled glaucomatous optic atrophy with normal IOP.

The prevalence of systemic hypertension today is increasing because of change in life style and many other factors which is beyond the scope of this article to describe in detail. The objective of this "write-up" is to highlight the relationship between progressive glaucoma and systemic hypertension.[2,9]

The systemic hypertension most of the time is diagnosed in the hospital. This could ignore the 'white-coat effect'. The 24-hour ambulatory blood pressure monitoring has shown a larger drop in mean systolic and diastolic blood pressure at night (nocturnal hypotension) among the subjects with normal tension glaucoma.[3,4,8] The Baltimore Eye Study has confirmed the significant correlation between the systemic blood pressure and glaucoma.[10] The cardio-vascular physicians have realized that adverse cardiac events increase, if systemic blood pressure drops below a certain level. It is, therefore, a plausible explanation that a similar process could occur in the optic nerve head resulting in anterior ischemic optic neuropathy due to fluctuating perfusion pressure in the capillaries supplying the optic nerve. Further, this concept has been proven by various scientific studies.[6, 10]

Until recently before the prostaglandin analogues were available, the beta-blocker eye drops were the preferred drug in glaucoma management. The beta-blocker, even in the form of an eye drop, can reduce the systemic blood pressure, which could further accelerate the glaucomatous progression.[5, 7, 8]
Therefore, management of glaucoma remains a challenge requiring co-ordination between the treating physicians of various sub-specialties particularly the internists and the ophthalmologists.

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