

Glaucoma Therapy — The Need for a New Direction

Reducing a raised intraocular pressure (IOP) and further lowering a statistically normal IOP has been the mainstay of glaucoma therapy for a long time. In spite of the realisation that IOP is only one of the risk factors and having some idea of the other risk factors, therapy for glaucoma has not evolved beyond lowering the IOP. Our preoccupation with the IOP factor is highlighted by a meta-analysis of 102 randomised clinical trials among which visual function was an outcome measure in only 3 studies.¹

Primary open angle glaucoma can be considered a characteristic progressive optic neuropathy that may or may not be associated with a statistically raised IOP. It is widely recognised that IOP is only one of the many risk factors involved in optic nerve damage in glaucoma. While at increasing levels of IOP the risk for disc damage increases, some studies have not found a correlation between the level of IOP and the severity of glaucomatous damage.^{2,3} It is reported that even after surgery to lower the IOP in one third of the eyes, optic neuropathy can progress.⁴ In addition, the sensitivity of the optic nerve head to IOP-induced damage varies widely from none to maximal.⁵ Currently, there is no definitive means of either assessing the sensitivity of the optic nerve head to IOP-induced damage or of differentiating the subgroup of glaucoma patients where raised IOP is not a contributing factor to disc damage. Hence, all glaucoma patients are subjected to the available means of reducing IOP. In those patients whose optic nerves are not sensitive to IOP-induced damage, some of the therapeutic interventions such as mitomycin C-augmented filtering surgery to lower IOP could be worse than the consequences of the natural course of the disease. Our goal of preserving the vision of glaucoma patients without causing any iatrogenic trauma would be better achieved if we could differentiate the subgroup of glaucoma patients whose optic nerve heads are insensitive to pressure-induced damage. In the absence of this knowledge, along with normalising a raised IOP, glaucoma therapy should aim to improve the blood supply to the nerve head and to rescue the ganglion cells that are programmed to die by stimuli from the adjacent dying cells, a process called 'apoptosis'. These modes of therapy are currently under investigation and the successful clinical use of these agents would be a major step towards our goal of preserving the vision of glaucoma patients. In this issue of Asian Journal of OPHTHALMOLOGY Julian Rait reviews our current knowledge in this regard.

The objective of controlling glaucoma-related blindness is achieved not only by delivering state-of-the-art treatment to the patient in the clinic but also by early detection prior to visual loss. To be able to achieve the latter in an effective manner, an understanding of the prevalence, types and any peculiarities of glaucoma presentation in the given community are essential. This information also helps to plan either a screening programme or to educate the public to seek advice in time. It is known that the prevalence of angle closure glaucoma in Asia is much greater than that in the Caucasian population.⁶ Furthermore, chronic asymptomatic angle closure glaucoma is more common than the acute form in some communities. This knowledge is crucial to the planning of any community-based programmes to control glaucoma-related blindness.

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