Relation of glaucoma progression and myopia: the role of axial length

Dear Editor,

The article by Perdicchi et al entitled "Visual field damage and progression in glaucomatous myopic eyes" was interesting (1). In that article, it was stated that visual field defect progression was worsened with the increase of myopia. As we know, myopia is a complex pathology of ocular structures with elongated axial length (AL) as one of its features. In this letter, we discuss a theoretical model that passes the more rapid progression of visual field defect in myopic eyes on the elongated AL rather than the myopia itself.

In contrast to the old assumption that causally links direct effect of increased intraocular pressure (IOP) to glaucoma, it was recently proposed that biomechanical factors such as tensile stress in the optic nerve (ON) head is the primary insult that leads to retinal ganglion cell loss, and increasing IOP is thought to act as only one of the determinant factors of glaucoma (2, 3).

Tensile stress causes deformations of the lamina cribrosa and results in neural tissue strain and consequently neural tissue damage. Tensile stress (σ) of the wall of a sphere is calculated by the formula $\sigma = pr/2t$, where p is pressure, r is the inner radius of the sphere, and t is the wall thickness, with tensile stress and r directly related (3).

Intuitively, it seems that the same target IOP for highly myopic patients should control the glaucoma progression to the same extent as normal eyes but the same IOP will have higher tension on ON head in myopic eyes. For example, if the AL of the eye increases from 20 mm to 26 mm, the optic nerve head will suffer 30% more tensile stress. This stress causes neural tissue strain which may reach the critical point and lead to ganglion cell loss in spite of apparently normalized IOP.

The results of the above-cited article imply that glaucomatous myopic eyes are doomed to suffer a more rapid progression of glaucoma. If it is proved that the glaucoma progression is related to the AL and not to the myopia itself, then the glaucoma progression may simply be halted by decreasing the target IOP with antiglaucoma medications to the level that compensates the detrimental effect of higher AL on ON head tension in myopic eyes. Therefore, the more rapid glaucoma progression would not be an inherent characteristic of myopia, but rather is a consequence of more ON head tension as a result of longer AL.

For corroboration of this assumption, the authors can reanalyze the data in a multivariate fashion by grouping the myopic cases into axial and refractive. Alternatively, they can perform multivariate analysis for different ranges of AL. The presence of more rapid progression of glaucoma only in axial myopic eyes (and not in the refractive myopia group) would justify our hypothesis.

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Author reply

I read the interesting correspondence about the role of axial length in progressing damage in glaucomatous myopic eyes. The hypothesis that a faster glaucoma progression in higher myopic eyes should be a consequence of more optic nerve head tension as a result of longer axial length is interesting but an important role must be related to the altered vascular factors in myopic eyes, probably due to their larger dimensions. Both axial length and vascular factors may play a role in determining the progressing damage. I will try to re-analyze patients considering the axial length but it seems clear that a lower target intraocular pressure must be pursued in high myopic eyes.

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