Re: Bhatti et al.: Microcystic Macular Edema in Optic Nerve Glioma

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To the Editor,

Bhatti et al. present images from a patient with inner nuclear layer cysts visible on optical coherence tomography (OCT) in the setting of an optic nerve glioma.[1] They attribute these to the effect of vitreous traction on Müller cell footplates (forming the inner limiting membrane) following degeneration of the ganglion cells and retinal nerve fibre layer (RNFL). This is one hypothesis, but it should be noted that another mechanism postulated in the literature is that these cysts represent retrograde trans-synaptic degeneration (presumably of bipolar cells) following loss of ganglion cells.[2,3] It is possible that both mechanisms are contributory.[2]

We have reported previously that patients with visual loss associated with silicone oil (following retinal detachment surgery) exhibit loss of RNFL and ganglion cells, and also demonstrate the same microcystic change after some years.[4] The vitreous traction hypothesis is less likely in these patients as they lack a vitreous, although it remains possible that a tractional element exists. The retrograde trans-synaptic degeneration hypothesis is plausible, and has been demonstrated at other levels in the visual pathway, such as RNFL thinning secondary to occipital cortex lesions.[5] We agree with Bhatti et al. that the term "microcystic macular edema" is a misnomer and we prefer the term "retrograde maculopathy",[3] which is neutral as to the precise mechanism, but emphasises the primary role of ganglion cell loss.

References

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