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CORRESPONDENCE

Light exposure in acute central serous chorioretinopathy

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To the Editor:

With great interest, we read the article by Maltsev et al. [1] exploring the association between axial length (AL) and morphological and clinical characteristics in acute central serous chorioretinopathy (CSC). There was an increasing rate of subretinal fluid recurrence, rate of bilateral involvement, and number of leakage points with shortening AL. The authors proposed that choroidal thickness may play an intermediate role for linking AL with the location of the leakage point probably by taking into account choroidal vessel diameter [2]. However, the mechanisms underlying the link among AL, choroidal thickness and vessel diameter, and CSC recurrence still remain obscure.

The highly vascularized choroid plays a major physiologic role for maintaining a stable temperature in the macula [3]. Almost all the light rays passing through the pupil are absorbed by the melanosomes in the retinal pigment epithelium and the choroid. It leads to high heat production and demands high blood flow to dissipate the light-generated heat and modulate tissue temperature in the macula [3]. The heat-induced choroidal vasodilation, whether passively or reflexively [4], could account for the thick choroid and short AL observed in animals and humans experiencing high light exposure [5, 6].

The rate of thermal energy accumulation per unit time in the macula has been overlooked in investigating causes of retinal disorders. When the accumulation rate is very high, the tissue burns like what happens in solar retinopathy. At very low rates, there is enough time for the tissue to be structurally adapted to the new condition like increases in choroidal thickness in individuals spending considerable time outdoors. At a moderate thermal energy accumulation rate, the heat-induced choroidal vasodilation may be severe enough to increase the permeability of the choriocapillaris and cause serous leakage. The number of leaks would be correlated with the accumulation rate at the moderate levels.

The mechanisms linking AL, choroidal thickness, choroidal vessel diameter, and leakages could potentially involve the light-generated heat in the macula. If it turns out to be true, we need to revise our strategies to prevent and manage CSC considering the rate of changes in environmental light intensity per time.

Compliance with ethical standards

Conflict of interest The author declares no conflict of interest.

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