Choroidal Response to Anti-VEGF Therapy in Subgroups Classified by Preexisting Ophthalmic or Systemic Condition

We thank Zhou et al. for their interest in our article. We appreciate this opportunity to discuss tailored therapy based on preexisting ophthalmic condition for myopic CNV.

Zhou et al. described several preexisting ophthalmic conditions that may lead to choroidal thinning and suggested the stratification of patients with myopic choroidal neovascularization (CNV) according to these conditions. In particular, they highlighted the association between choroidal thinning and retinopathy of prematurity (ROP).

Accordingly, we reviewed preexisting ophthalmic or systemic conditions, including ROP among our 54 patients. There was no history of ROP in any patient. A total of 17 patients with hypertension, three with diabetes, and four with hyperlipidemia were included in our study; however, no patients with hypertensive retinopathy or diabetic retinopathy were included because these conditions comprised exclusion criteria. Patients with age-related macular degeneration also were excluded. Therefore, of the five conditions associated with choroidal thinning proposed by Zhou et al., (ROP, myopia, diabetic retinopathy, geographic atrophy, and nonarteritic anterior ischemic optic neuropathy), only myopia was present in these cases. Therefore, in our patient cohort, we could not stratify patients according to these preexisting ophthalmic disorders.

Regarding preexisting systemic disorders, we agree that the patients with diabetes mellitus, hypertension, and hypercholesterolemia may have exhibited choroidal microvascular changes, although they did not indicate retinopathy. Patients could be divided into two groups based on the presence or absence of hypertension (N = 17 and 37, respectively) whereas the number of patients with diabetes (N = 3) and hyperlipidemia (N = 4) was too small for statistically significant stratification.

We believe that preexisting systemic or ophthalmic disorders may affect choroidal thickness after anti-VEGF therapy in two ways. First, the disorders can affect baseline choroidal thickness (before anti-VEGF therapy). Second, they can affect changes in choroidal thickness (choroidal response) after anti-VEGF therapy. In our patients, we did not detect any significant difference in choroidal thickness at baseline (P = 0.835) or 1 month after anti-VEGF therapy (P = 0.707) between eyes with and without hypertension. Furthermore, there was no significant difference in choroidal thickness change after anti-VEGF therapy between the two groups (P = 0.453). Therefore, based on the data, we are unable to conclude on the potential association between preexisting ophthalmic or systemic disorders and choroidal thickness in eyes with myopic CNV treated with anti-VEGF therapy.

Although patients with myopic CNV could be stratified according to preexisting ophthalmic disorders associated with choroidal thinning and eyes with such disorders may show different choroidal responses to anti-VEGF therapy, our data cannot guide treatment decisions according to preexisting systemic or ophthalmic disorders. We believe that in a typical clinical setting, the association between systemic conditions and choroidal thickness is negligible during the treatment of myopic CNV as the effect may be minimal or absent. To develop tailored treatment for myopic CNV based on preexisting ophthalmic conditions, we suggest that further studies should be performed to compare choroidal responses to anti-VEGF therapy in resolved or recurrent cases between eyes with and without such preexisting ophthalmic disorders. Based on the results, conditions affecting choroidal thickness should be considered carefully in the interpretation of choroidal responses to therapy.

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Acknowledgments

The authors alone are responsible for the content and writing of the paper.

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Citation: Invest Ophthalmol Vis Sci. 2015;56:8364.
doi:10.1167/iovs.15-18634