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Branch Retinal Vein Occlusion and Peripapillary Choroidal Thinning

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The Editor

I was interested to read the article by Kang and colleagues, entitled “Significant reduction of peripapillary choroidal thickness in patients with unilateral branch retinal vein occlusion” published in the *Retina*.¹ This is based on a retrospective study of 29 patients with unilateral branch retinal vein occlusion (BRVO). The authors conclude: “Peripapillary choroidal thickness decreased significantly over 12 months in BRVO-affected eyes and nonaffected eyes in patients with unilateral BRVO.”

I have researched comprehensively the retinal and choroidal vascular beds in health and disease for more than 50 years. Kang and colleagues’ study deals with BRVO. I prospectively investigated 214 consecutive BRVO patients seen within 3 months of onset.² Based on that experience, I have several problems with the study by Kang and colleagues¹, including the following.

1. The authors state: “Many investigators have shown the close association between retinal vein occlusion and glaucoma”; this article deals with BRVO. They cited my study³ in support of this statement. However, the cited study dealt with the association of central (CRVO) and hemisentral (HCRVO) retinal vein occlusions with glaucoma and ocular hypertension (OHT), not with BRVO; I clearly stated “no similar association between branch retinal vein occlusion and glaucoma/OHT has been shown by our study”. I have discussed that clinically and pathogenetically HCRVO is a variant of CRVO and NOT a major BRVO.⁴
2. The authors state: “The vascular theory is one of the hypotheses that explains the pathophysiologic correlation between glaucoma and RVO”. In support of this statement, they cited two of my studies^{5,6}, neither of which contains any such information related to RVO.
3. The authors have cited many references in support of their statements. Some, like mine are not relevant. Moreover, unfortunately, not everything that gets

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published is scientifically valid. To cite misleading articles simply perpetuates misinformation.

4. According to the authors: “percentage of patients with branch retinal vein occlusion (BRVO) who later develop glaucoma is also higher than in normal population”. My experience of more than 250 BRVO patients, with long follow-up, has shown no such phenomenon.
5. They state: “a simple cause-and-effect relationship between RVO and glaucoma cannot be applied.” Here it seems they are talking of all types of retinal vein occlusion. This is a misleading statement. In our article³ we discussed the “Role of glaucoma/OHT in pathogenesis of CRVO/HCRVO”.
6. The authors state “There are several hypotheses that explain the association between BRVO and glaucoma..... RVO and glaucoma share a common underlying vascular abnormality in their pathogenesis”. First, as discussed above, their claim of an “association between BRVO and glaucoma” is not valid. Second, peripapillary choroidal thinning in their study was seen not only in the eye with BRVO but in both eyes. Third, based on my studies dealing with glaucoma and BRVO, I find no logical relationship between the development of BRVO and of peripapillary choroidal thinning, because etiologically and pathogenetically the two disorders are of totally different natures. My studies have shown that the “systemic risk factors”, postulated by the authors, do not explain this. This indicates that some important piece of information is missing in this small retrospective study, or the simultaneous presence of these two completely unrelated disorders must be accounted for in some other way.
7. Their study showed no significant change in the subfoveal choroidal thickness. Therefore, their proposed “systemic risk factors” theory cannot explain localized peripapillary choroidal thinning only.
8. They state: “we speculate that the changes in peripapillary choroidal thickness are a characteristic finding in BRVO, supporting the vascular theory”. There are two issues here. (i) Based on my comprehensive study of more than 250 BRVO eyes, compared to their 29, their statement that “changes in peripapillary choroidal thickness are a characteristic finding in BRVO” is not true. Retinal and choroidal circulations are totally independent, so that retinal pathology could not possibly affect choroidal circulation or vice versa. (ii) What do they mean by “vascular theory”? Why does their vascular theory apply to the peripapillary choroid only and not to the subfoveal choroid which was normal in their study? There was no glaucoma in these eyes. These facts show that their speculation is not valid.
9. They state: “careful monitoring of peripapillary choroidal thickness may be useful to detect early glaucomatous changes in these patients”, i.e. in BRVO. This statement is based on a retrospective study of only 29 eyes. But, as discussed above, my large BRVO study² showed that the prevalence of glaucoma in BRVO is no higher than that seen in the general population. The authors have

ignored one fundamental factor in the pathogenesis of BRVO – the site of occlusion in BRVO is at the arteriovenous crossing at the retina and not in the optic nerve (seen in CRVO and HCRVO); so glaucoma or OHT do not influence the retina directly, although they do influence the optic nerve.

10. The authors state that in BRVO “the changes in peripapillary choroidal thickness have not been heretofore investigated despite its association with glaucoma.” First, importantly, there is no reason to investigate glaucoma in BRVO because, as discussed above, there is no association between BRVO and glaucoma. Second, there is no logical reason for any association between BRVO and peripapillary choroidal changes.
11. Their study showed “The mean RNFL (retinal nerve fiber layer) thickness decreased significantly over 12 months in BRVO-affected eyes ($P=0.009$) but not in nonaffected eyes ($P=0.206$).” That is obviously because of ischemic damage to the retinal ganglion cells and retinal nerve fiber layer in the BRVO eye.

In conclusion, all these problems cast a shadow over the study.

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