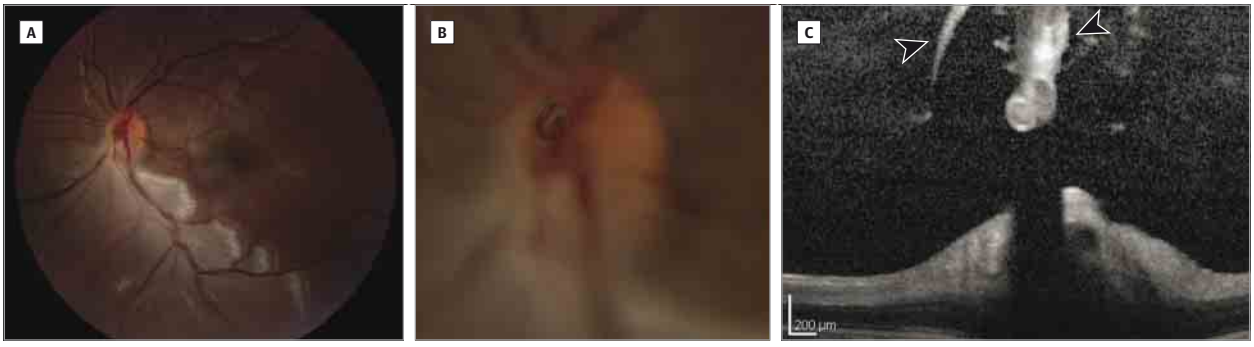
Figure. Prepapillary Vascular Loop and Associated Branched Retinal Arterial Occlusion



A and B, Color fundus photographs demonstrating a coiled loop with superficial retinal whitening along the inferotemporal arcade. C, Spectral-domain optical coherence tomographic image revealing adhering vitreous gel (arrowheads) at the apex of the loop.

We agree with Dr Walland and had similarly proposed a contributing etiologic role of the intact or acutely detaching vitreous gel in inducing a tractional kinking or twisting of the loop around its central axis, ultimately obstructing distal blood flow and triggering the arterial occlusive event. We similarly are intrigued that these 2 cases have occurred in younger patients, who are more likely to have a formed intact vitreous gel, although Dr Walland's suggestion that a component of reporter bias may be present given the anomaly of a BRAO occurring in a young patient cannot be overlooked.

Since our article was published, we have evaluated yet another young patient with a traumatic BRAO and associated prepapillary vascular loop (**Figure**). A previously healthy girl in her late teens presented with acute onset of a superotemporal scotoma in the left eye starting shortly after being punched on the left side of her head in a physical altercation. On presentation, Snellen visual acuity was 20/25 OU. Confrontational visual field testing confirmed a superotemporal deficit in the left eye. While anterior segment examination findings were unremarkable bilaterally, ophthalmoscopic examination of the symptomatic left eye revealed a partial posterior vitreous detachment, superficial retinal whitening along the inferior arcade consistent with an inferotemporal BRAO (Figure, A), and a coiled prepapillary vascular loop emanating from the optic disc (Figure, B). Ophthalmoscopy of the right eye demonstrated a perfused prepapillary loop but was otherwise normal. Interestingly, ancillary spectral-domain optical coherence tomography registered through the loop of the left eye (Figure, C) showed what appeared to be vitreous adherent to the vasculature, further suggestive of a vitreous-derived tractional force in inducing the loop torsion and subsequent BRAO.

As Dr Walland suggested and our 2 cases demonstrate, ongoing advances in retinal multimodal imaging are helping to better define the relationship of prepapillary vascular loops to the overlying vitreous compartment as well elucidate any potential contribution to pathological disease states that can infrequently result as a complication.

Ehsan Rahimy, MD
Nadim Rayess, MD
Richard S. Kaiser, MD

Author Affiliations: Retina Service of Wills Eye Hospital, Mid Atlantic Retina, Philadelphia, Pennsylvania.

Corresponding Author: Richard S. Kaiser, MD, Wills Eye Hospital, 840 Walnut St, Philadelphia, PA 19107 (richardskaisermd@gmail.com).

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Comparative Effectiveness of Bevacizumab and Ranibizumab in the Comparison of Age-Related Macular Degeneration Treatments Trials

To the Editor In 2012, Martin et al[1] published the results at 2 years of the Comparison of Age-Related Macular Degeneration Treatments Trials and found a nonsignificant difference between bevacizumab and ranibizumab according to the continuous end point of the change in letters of visual acuity (mean difference for bevacizumab vs ranibizumab, −1.4 letters; 95% CI, −3.7 to +0.8 letters; $P = .21$). More recently, Ying et al[2] carried out a multivariate analysis of the same study data in which the bevacizumab group was shown to have higher odds of losing 15 or more letters than the ranibizumab group (odds ratio, 1.83; 95% CI, 1.07 to 3.14; $P = .03$).

If the crude rates of failures reported by Ying and colleagues are examined by univariate analysis (37 of 501 patients for bevacizumab vs 24 of 528 patients for ranibizumab), the odds ratio is 1.67 (95% CI, 0.99 to 2.84; $P = .05$), which is very close in terms of magnitude and statistical significance to that obtained by multivariate statistics. Hence, while these univariate results and the results from the multivariate analysis are similar, they suggest the possibility of a different conclusion from the primary results of the Comparison of Age-Related Macular Degeneration Treatments Trials.[1]

In interpreting their results, Ying and colleagues postulated that, as compared with the previous results by Martin and colleagues, the more pronounced difference between bevacizumab and ranibizumab was related to the multivariate de-

sign of their analysis and to the consequent adjustment for covariates. However, the observation that univariate and multivariate results were similar suggests another explanation: the inferiority of bevacizumab vs ranibizumab could be a spurious result related to the adoption of a dichotomous end point in substitution for the continuous one.

In general, when the same data set is analyzed by using a continuous or a dichotomous variable, the most information is contained in the analysis of the continuous variable. Expressing the results according to the dichotomous variable (defined according to a cut point) can determine a more effective communication of the results, but some information is unavoidably lost. This loss of information can have unpredictable effects, particularly if there is a considerable amount of data near the cut point. For this reason, the inferiority of bevacizumab vs ranibizumab found by Ying and colleagues could be an artifact related to converting the continuous end point into a dichotomous one. To exclude this artifact, a reanalysis could be made by the authors in which the end point is kept as a continuous variable. If this reanalysis confirms the inferiority of bevacizumab, the clinical relevance of this important finding would be strongly enhanced.

Andrea Messori, PharmD

Author Affiliation: Health Technology Assessment Unit, Area Vasta Toscana Centro, Regional Health Service, Firenze, Italy.

Corresponding Author: Andrea Messori, PharmD, Health Technology Assessment Unit, Area Vasta Toscana Centro, Regional Health Service, Via di San Salvi 12, 50100 Firenze, Italy (andrea.messori.it@gmail.com).

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