Early Macular Vessel Density Loss in Acute Ischemic Optic Neuropathy Compared to Papilledema: Implications for Pathogenesis

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Dear Editor:

We read with great interest the article by Fard et al.¹ comparing macular and parafoveal vasculature in 20 eyes with acute nonarteritic anterior ischemic optic neuropathy (NAION), 39 eyes with papilledema at first presentation, and 22 healthy eyes. They found that superficial and deep vasculature density values were significantly lower in NAION eyes compared to control and papilledema eyes. By contrast, no significant differences in ganglion cell complex (GCC) thickness were observed among NAION, papilledema, and control eyes.

On the other hand, the authors mentioned the study by Akbari et al.,² who found that ganglion cell loss does not occur at presentation in NAION eyes, to support that macular vasculature abnormalities occurred before detectable macular GCC atrophy. Focusing on NAION eyes, we would like to clarify these findings based on prior published data,³,⁴ which, however, have not been mentioned by the authors. In contrast to the studies of Fard et al.¹ and Akbari et al.,² we previously reported that NAION eyes had a significant thinning of the ganglion cell inner plexiform layer minimum (GCIPL min) as early as 2.2 days after symptoms onset (P = 0.017). In fact, the GCIPL min was abnormally thinned in more than 50% of eyes (56.3%) within a week of symptom onset.³

Recently, we analyzed the superficial peripapillary vascularization in eyes with acute NAION (<1 week) and at 3 months (atrophic stage) using an OCT-A device (AngioPlex; CIRRUS, HDOCT-5000, 10.0; Carl Zeiss Meditec, Jena, Germany) and two analysis strategies (the automatically Angioplex metrix field analysis and a customized analysis to remove the peripapillary large vessels).⁴ Both strategies demonstrated a significant peripapillary microvascular dropout in NAION. Although the GCIPL thickness significantly thinned from acute to atrophic stages, all GCIPL values (average, minimum and all sectors) already were significantly reduced at acute episode compared to healthy eyes.

Several reasons can explain these differences regarding the macular thickness at acute NAION. First, different OCT devices and combinations of macular inner layers were used: Avanti SD-OCT (Optovue, Inc., Fremont, CA) to map the GCC by Fard et al.,¹ and Cirrus OCT (Carl Zeiss Meditec) to quantify the GCIPL thickness by De Dompablo et al.³ and Rebolleda et al.⁴ This point is relevant because a significant macular RNFL thickening develops almost immediately in NAION, which may be an extension of peripapillary edema.⁵ Considering that GCC analysis includes the combined thickness of the GCIPL and macular RNFL, the macular RNFL thickening could mask the neuronal loss at the acute phase, which would be better detected by the analysis only focused on GCIPL. Second, Fard et al.¹ included eyes with papilledema, apart from eyes with acute NOIAN. By contrast, we only analyzed NAION eyes. Third, the time that defined the acute NAION was longer in their study than in ours (within 2 weeks vs. 1 week).

Although, as Fard et al.¹ suggested, the vessels dropout could be a primary result of ischemia in NAION and the impaired vascular supply eventually leads to neuronal loss; conversely, microvascular dropout might be a secondary change accompanying retinal ganglion cell loss and diminished metabolic needs. This last hypothesis could be supported by the significant correlation we found between the mean
GCIPL thinning and the microvascular reduction from the acute to the atrophic stages. Therefore, we consider that, before concluding if vascular changes precede structural changes or vice versa, larger longitudinal studies are needed to determine the temporal relationship between the structural damage and retinal microvasculature dropout observed in prior studies.


References