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LETTER

A Comparison of Vessel Density in Primary Open-Angle Glaucoma and Normal-Tension Glaucoma Patients Using OCT Angiography [Letter]

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Dear editor

We read with interest the article by Zehavi-Dorin et al[1] comparing vessel density in Primary Open-Angle Glaucoma (POAG) and Normal-Tension Glaucoma (NTG). We commend the authors for their rigorous severity-matching using Hodapp-Parrish-Anderson criteria and thoughtful study design. We write to offer several methodological considerations that may warrant further discussion and help contextualize their conclusion that "vascular factors may play a more prominent role in the early stages of NTG".[1]

Statistical Significance and the Vascular Hypothesis

The authors report a "trend toward significance" ($p = 0.058$) for lower peripapillary vessel density in early NTG. While this is an intriguing observation, we note that after adjustment for multiple comparisons, this difference was not statistically significant ($p = 0.301$).[1] We respectfully suggest that this non-significant finding might alternatively indicate that global vessel density metrics are not sufficiently sensitive to distinguish between POAG and NTG when severity is matched. We would be interested to know the authors' perspective on the statistical basis for their conclusion regarding vascular factors in early NTG given the adjusted p-value of 0.301.

The authors note that "the RNFL thickness appeared to follow a similar trend to vessel density".[1] Given the well-established correlation between vessel density and RNFL thickness in both healthy[2,3] and glaucomatous eyes,[4] we wonder whether the observed patterns in vessel density might reflect parallel structural changes. We would be interested to learn whether a multivariate analysis adjusting for RNFL thickness was performed, as this could help clarify if the microvascular changes represent an independent finding.

Additional Data That May Inform Interpretation

The authors report similar IOPs on the day of OCTA (13.9 ± 3.4 mmHg for POAG vs 13.3 ± 2.4 mmHg for NTG),[1] suggesting patients were on treatment. Since Holló[5] demonstrated that significant IOP reduction can increase peripapillary vessel density, we wonder whether information on baseline untreated IOP, treatment regimens, and disease duration might be available. This additional context could help readers better understand whether the observed vessel density reflects underlying disease pathophysiology or potential treatment effects.

The authors acknowledge that "systemic hypertension, cardiovascular status, or topical and systemic medications" were not collected.[1] We appreciate this transparent acknowledgment. Given that NTG is associated with systemic vascular dysregulation[6,7] and that conditions such as hypertension, sleep apnoea, and carotid stenosis can affect retinal vessel density,[8–10] future studies incorporating these variables might help further elucidate the relationship between vessel density and NTG pathophysiology.

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The authors focused on global metrics, noting that "sectoral or focal changes" were not examined.[1] Since NTG frequently presents with focal neuroretinal rim notching[11] and global averaging might potentially mask regionally specific changes,[12,13] we wonder whether a sectoral analysis might reveal additional insights that complement the global findings reported in this study.

An Alternative Perspective

We find the authors' alternative hypothesis particularly compelling: "As ganglion cell death progresses, secondary vascular attrition takes over".[1] The statistical similarity in vessel density between severity-matched POAG and NTG groups across all stages appears consistent with this interpretation.

Building on this, we suggest that the absence of significant differences even in early disease might indicate that by the time glaucoma is clinically detectable, regardless of IOP phenotype, a common pathway of neurovascular degeneration may already be engaged. In this scenario, vessel density changes potentially represent consequences rather than primary causes of neuropathy in both subtypes. This interpretation would align with the parallel trends between RNFL thickness and vessel density that the authors observed.

If vessel density primarily reflects structural neuronal loss rather than independent vascular pathology, the findings of no significant difference between POAG and NTG when severity-matched would be expected and meaningful.

We thank Zehavi-Dorin et al for their valuable contribution of carefully severity-matched OCTA data to this important field. We offer these observations in the spirit of constructive scientific discourse and believe that future prospective studies incorporating treatment data, systemic hemodynamic profiling, and sectoral analysis would further advance our understanding of the vascular profile in NTG.

Disclosure

The authors report no conflicts of interest in this communication.

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