

## Mechanisms of post-radiation optic atrophy with neuroretinal rim thinning

Dear Editor,

We read with interest the article “Optic disc cupping after circumpapillary PD-103 slotted plaque radiation therapy.”<sup>1</sup> Finger et al. examined a cohort of patients managed with slotted plaque radiotherapy for peripapillary, juxtapapillary, or circumpapillary choroidal melanoma and found that treatment was associated with subsequent increase in cup-to-disc ratio.

We recently published a series of patients managed with round Collaborative Ocular Melanoma Study plaques and found that of 78 patients, 41 developed post-radiation optic atrophy, and 15 had concomitant neuroretinal rim thinning,<sup>2</sup> a phenotype matching that described by Finger et al. These findings are not unique to a slotted plaque design, but they could be more likely in the setting of greater radiation dose to the optic disc and posterior ciliary arteries. In our series, we found that all patients with neuroretinal rim thinning also developed some degree of optic disc pallor. Although Finger et al. found no significant increase in pallor after treatment, we suspect this may have been due to small sample size, especially given  $p = 0.051$  and a change in both the median and minimum pallor grade from zero to one after treatment.

In our study, we found an additional association between higher baseline intraocular pressure (IOP) and development of post-radiation optic atrophy, with a further association of higher maximum IOP and the phenotype of neuroretinal rim thinning. Finger et al. emphasized that higher post-treatment IOP was likely not responsible for neuroretinal rim thinning in their series, but our data suggest that higher IOP, even if within the “normal” range, may increase susceptibility to post-radiation optic atrophy, perhaps due to connective tissue stress and strain.<sup>3</sup>

We applaud Finger et al. for their application of optical coherence tomography angiography. Optic disc cupping in their study correlated to changes in vessel length and density, leading to the hypothesis that radiation-induced ischemia could be responsible for the optic disc changes. Overall, we were excited to see validation of findings we have observed in our own practice, and we agree that prospective studies including correlation of visual field defects are necessary to better understand how post-radiation optic atrophy compares with glaucomatous optic neuropathy.

**Lauren A. Dalvin, Gavin W. Roddy**

Mayo Clinic, Rochester, Minn.

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Correspondence to:

Lauren A. Dalvin, MD; [dalvin.lauren@mayo.edu](mailto:dalvin.lauren@mayo.edu).

## References

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## Footnotes and Disclosure

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