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RESPONSE TO LETTER

# Prevalence and Distribution of Macular Fluid with Central Retinal Artery Occlusion and Anterior Ischaemic Optic Neuropathy [Response to Letter]

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## Dear editor

We thank Plant et al for their interest in our article "Prevalence and Distribution of Macular Fluid with Central Retinal Artery Occlusion and Anterior Ischemic Optic Neuropathy".[1] In our study, 6 out of 11 eyes with acute non-arteritic anterior ischemic optic neuropathy (AION) had intraretinal fluid on optical coherence tomography (OCT). All 6 eyes had fluid involving the outer nuclear layer, 4 eyes had fluid within the inner nuclear layer (INL), and 3 eyes had fluid within the ganglion cell layer (GCL).

We agree with the authors of the letter on the importance of distinguishing the acutely developing microcystic macular edema from the later developing cystic degeneration of the INL (referred to as "retrograde maculopathy"). In a prior article by the authors,[2] it was hypothesized that the former develops due to "axonal swelling and further vascular compromise" that leads to "leakage of the peripapillary vessels" saturating Müller cell capacity to remove fluid. To support their hypothesis, the authors cited a study[3] that quantified INL cells across the retina and suggested that the decrease in Müller cell density with eccentricity from the fovea explains why fluid is observed in the peripapillary area and not the fovea in eyes with AION. It is important to note that the cited article by Masri and colleagues[3] actually found the peak Müller cell density to be at 0.8 mm eccentricity from the fovea, with lower density noted in the central fovea and peripheral macula, and with a higher proportion of Müller cells in the peripheral compared to the central retina. Further, the overwhelmed Müller cells by the leakage from the peripapillary vessels would not explain why in some instances fluid cysts could be noted within the GCL even away from the peripapillary region (an example from our cohort is given in Figure 1).

The authors of the letter also address the description of central retinal artery occlusion (CRAO) cases included in our study. They suggest that the term "cloudy swelling" should be used instead of whitening when describing the colored fundus photographs of CRAO cases, and that the term also serves the OCT appearance of the GCL in such cases. We would like to point out that CRAO has different grades and may evolve over time.[4] For example, paracentral acute middle maculopathy with INL infarcts and without GCL involvement may present as greyish-white lesions within the macula and can be the sole presentation in milder cases of CRAO.[5] That is why we believe that not all cases of CRAO involve cloudy swelling of the GCL (although the case depicted in our article had full-blown CRAO and did indeed show cloudy swelling). It is also important to distinguish between increased macular thickness with CRAO due to swelling of the inner retinal layers[6] from actual cystic fluid accumulation within the middle and outer retinal layers.[7] While the former does represent tissue edema (cytotoxic edema) as the authors accurately point out, the latter may evolve during the disease course[7] and could possibly have a different pathophysiological mechanism of Müller cell dysfunction. Again, we thank the authors for their interest in our article, congratulate them on their prior studies and look forward to their future work on this important topic.

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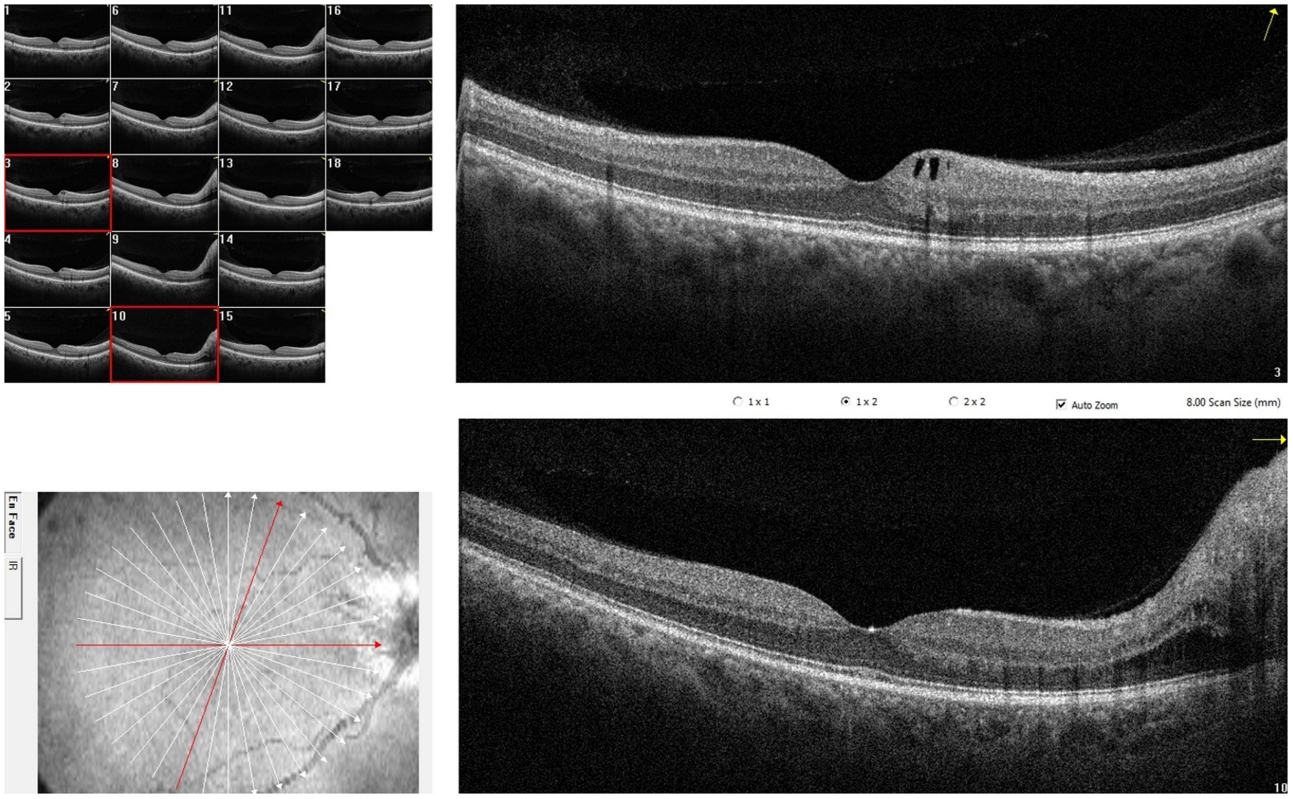


**Figure 1** Optical coherence tomography scans depicting different distribution of intraretinal fluid in an eye with acute anterior ischemic optic neuropathy. A horizontal macular radial scan (lower B-scan) shows peripapillary accumulation of fluid within the outer and inner nuclear layers and a swollen optic disc. An oblique macular radial scan (upper B-scan) shows cystic cavities within the ganglion cell layer in the upper nasal parafovea.

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