



Prevalence and Distribution of Macular Fluid with Central Retinal Artery Occlusion and Anterior Ischemic Optic Neuropathy [Letter]

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Dear editor

We read with interest the article by Fouad et al regarding macular fluid in CRAO and AION.¹ The results obtained in 11 cases of AION are generally in agreement with the acute phase findings in our own study,² emphasizing that early OCT imaging is crucial to detect retinal fluid accumulation and tissue oedema in AION. However, we disagree with the comment that in our article we reported that the finding in the inner nuclear layer (INL) of microcystic macular oedema (MME) “constitutes inner retinal cysts presumably associated with impaired axonal transport”. We hypothesized that this change seen *acutely* in the inner nuclear layer (which is peri-papillary rather than macular in distribution) occurs in situations wherein the various mechanisms for elimination of retinal tissue fluid are overwhelmed. Impaired axonal transport accounts for the axonal swelling which contributes to the elevation of the optic disc.

As we had the advantage of follow-up data we were able to demonstrate that: (1) this change is reversible; and (2) the later finding of INL cystic change which is related to ganglion cell loss (often referred to as MME although we prefer the term “retrograde maculopathy” (RM)) is very strictly limited to the macula, likely permanent and not related to tissue oedema.³ The point we are trying to make is that the use of the term MME is misleading in cases of RM which are unrelated to tissue oedema as such. However, the appearance on OCT of the two findings is similar as a consequence of the anatomical structure of the INL.

The comparison with CRAO provided in this study is indeed of great interest: the oedema is arising from ischaemic inner retina rather than the optic disc. The authors make the point that the “macular edema associated with acute CRAO has long been suggested to be distinct from other retinal vascular disorders in being intracellular swelling rather than extracellular fluid accumulation”, citing a recent study.⁴ We would like to point out that this has been understood for more than a century and that the change in appearance of the *en face* retinal image that is referred to in the authors' Fig 1 as “whitening” has been better described as “cloudy swelling” for nearer two centuries. The term was introduced by Virchow⁵ in the mid-19th century (as “Trübe Schwellung”) in histopathological cellular studies of many organs and was soon taken up by ophthalmologists following the invention of the ophthalmoscope. We would suggest that “cloudy swelling” is therefore the most appropriate terminology with a long history corresponding to what is now generally referred to as cytotoxic oedema. This change is of course not seen in AION where the loss of retinal ganglion cells follows retrograde degeneration rather than direct ischaemic damage. The outer retinal changes and subretinal fluid in CRAO are tissue oedema. The 2/3 CRAO cases in the study not showing cloudy swelling of the GCL may have been examples of border-zone retinal ischaemia.

Disclosure

The authors report no conflicts of interest in this communication.

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