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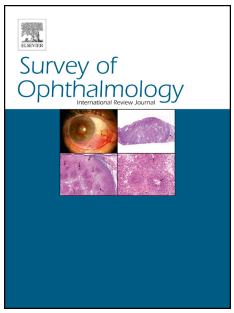

Optical coherence tomography angiography (OCTA) and retinal microvascular ramification in AMN and PAMM

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The recent review by Bhavsar and colleagues [2] on Acute macular neuroretinopathy (AMN) makes important points drawing on the availability of new multimodal imaging modalities. We agree with the concept that capillary plexus ischemia causes AMN and Paracentral acute middle maculopathy (PAMM). In this context the authors make a convincing point for the future role optical coherence tomography (OCT) angiography (OCTA) likely will play in the diagnostic work up.

Whilst we share the enthusiasm, a word of caution may be helpful as the clinical spectrum of AMN and PAMM keeps expanding and very subtle changes on OCT/OCTA are used to make a diagnosis. Second, as the authors correctly state causality of the presumed microvascular mechanisms still requires to be proven and we illustrate this with a own case.

First, it is relevant to be aware of pitfalls using OCT and OCTA. Artifactual hyperreflective bands may be seen close to the foveola due to oblique entrance of the OCT measurement beam which influences layer thickness data.[5] This is caused by asymmetric light back scattering from Henle fibres. Therefore one needs to be careful to keep the OCT B-scan strictly horizontally aligned during imaging.[1] Next, segmentation failure using automated algorithms may cause atrifactual thickness changes. For this reasons careful manual revision of segmented OCT scans is mandatory.[3] The same applies to OCTA which also requires careful revision of layer segmentation and localization of the capillary plexi. Finally, saccadic intrusions during OCTA may cause artifactual cutoff of vessels. A multimodal retinal imaging approach demonstrating consistent findings across modalities will contribute to optimize diagnostic specificity and sensitivity.
Such an approach should also consider Scanning Laser Polarimetry as probably one of the earliest signs for retinal tissue damage.[4]

Second, changes in the retinal microvasculature may exist with and without associated INL thinning and corresponding scotoma. A 68 year old man presented to our clinic on the 6th of May 2015, two months after sudden onset of a scotoma in his left eye inferotemporal to fixation (Figure 1A). He has a past medical history of hypertension, hypercholesterolaemia. He was a non-smoker and did not consume caffeine containing drinks. His best corrected Snellen visual acuity was 6/6 bilaterally. The fundus was normal, arterioles were not attenuated with no emboli seen. Spectral domain OCT showed thinning of the INL.