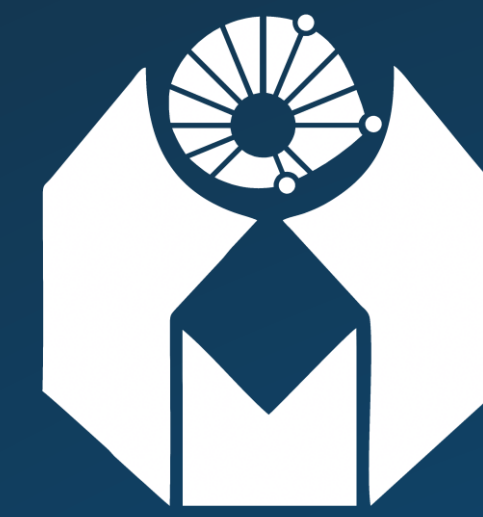


Central Retinal Vein Occlusion and Paracentral Acute Middle Maculopathy: a Case Report

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Background

Paracentral Acute Middle Maculopathy (PAMM) is characterized by hyperreflective bands and focal or diffuse lesions at the level of inner nuclear layers (INL), patients typically present with a negative central scotoma with acute onset. With a probable vascular etiology, risk factors include arterial hypertension, diabetes mellitus, sickle cell disease and retinal vascular occlusions. In here, we reported a case of PAMM secondary to Central Retinal Vein Occlusion (CRVO).

Case Report

A 70-year-old female presented with sudden vision loss in the right eye. Her previous clinical history included hypertension and diabetes mellitus, she was also under treatment for Primary Open Angle Glaucoma with three topical IOP-lowering drugs.

On examination, the best corrected visual acuity was 20/70 in the right eye and 20/30 in the left eye. Fundoscopic examination of the right eye revealed scattered intraretinal haemorrhages in four quadrants, peripapillary haemorrhage and dilated and tortuous retinal veins. The cup-to-disc ratio of the same eye was 0,7x0,6 (Figure 1). Examination of the left eye was unremarkable, besides the glaucomatous optic disc. Anterior segment and intraocular pressure were normal bilaterally.

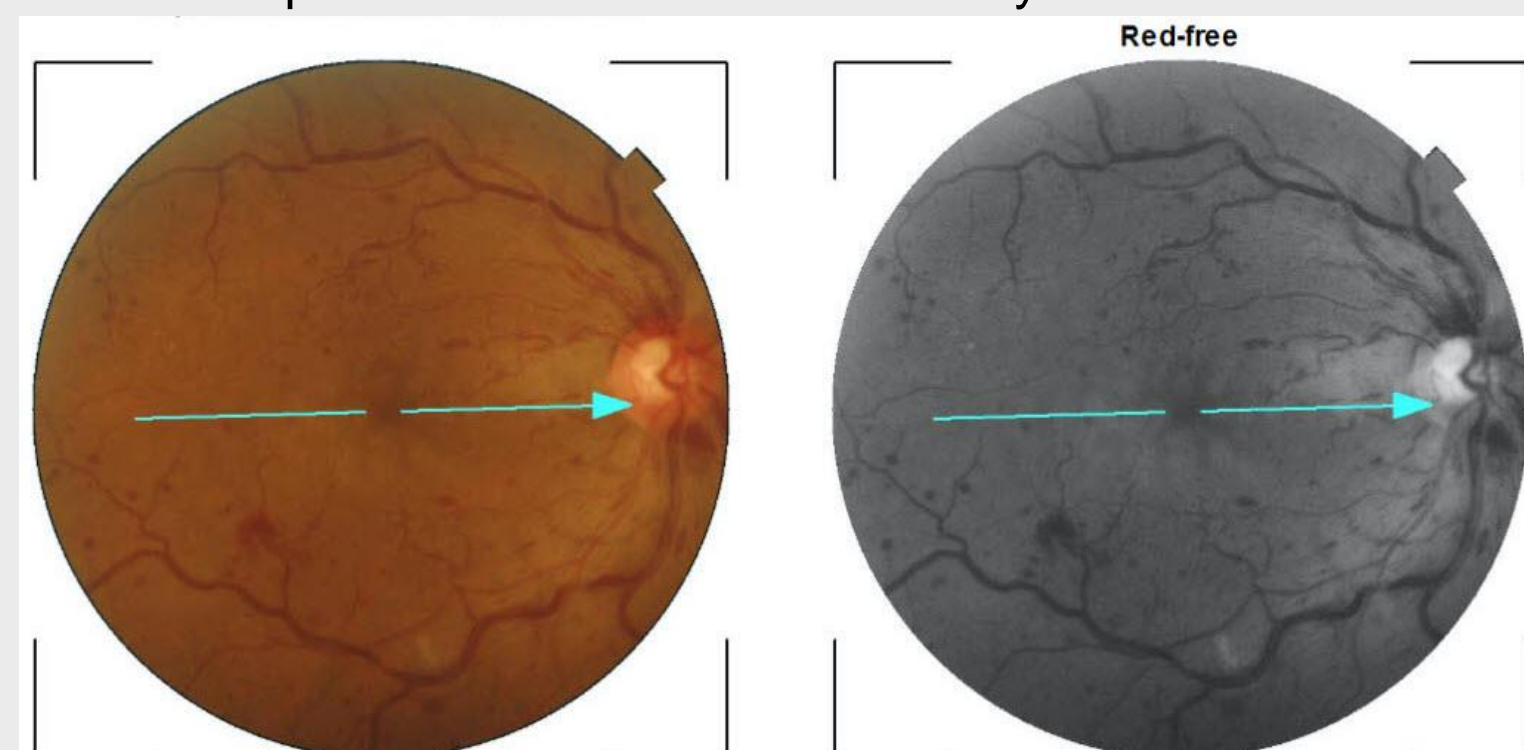


Figure 1. Retinography showing diffuse haemorrhage and tortuous retinal veins.

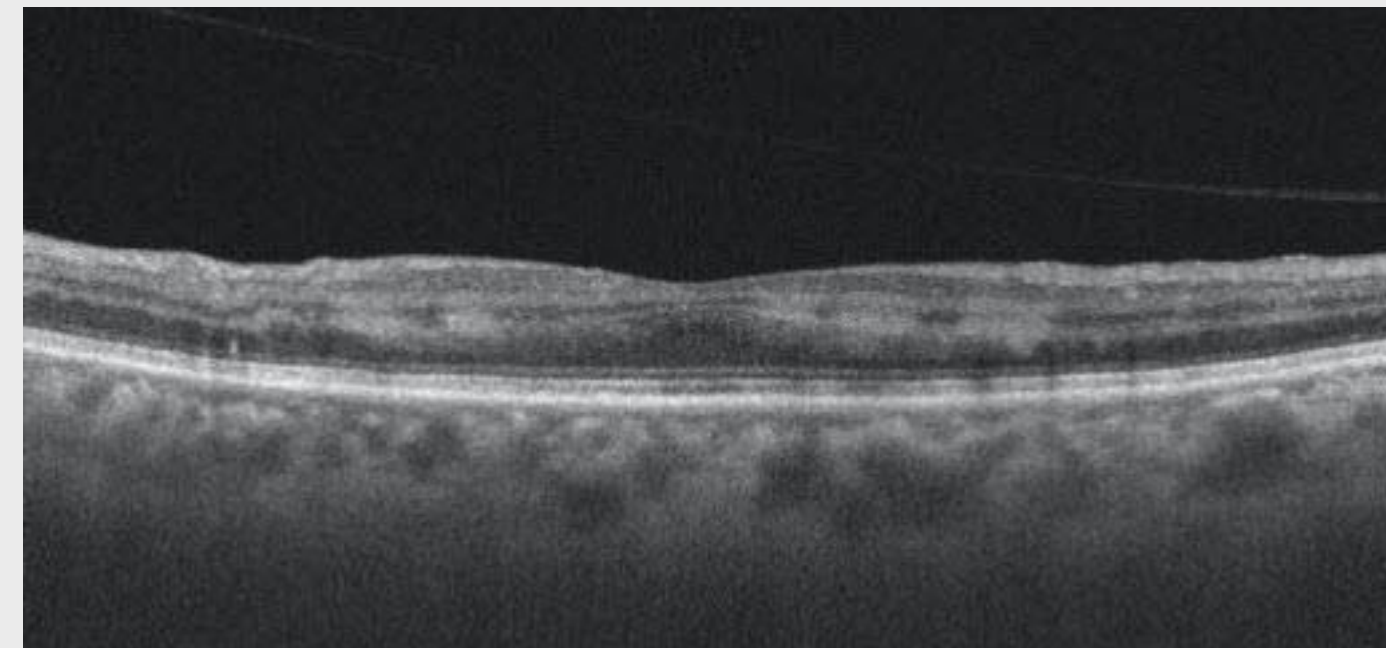


Figure 2. SS - Optical Coherence Tomography (vertical slicing) evidencing hiperreflectivity bands along the foveal slopes.

The patient referred previous allergic reaction to contrast, reason why fluorescein angiography could not be performed. SS-OCT of the right eye revealed absence of edema (Central Foveal Thickness of 270 μ m), hyperreflective band-like in the inner nuclear layer suggestive of Paracentral Acute Middle Maculopathy (Figure 2).

OCT angiography 6,0 x 6,0 mm showed attenuation of the vascular flow signal in the superficial capillary plexus (SCP) and vascular flow void in the deep capillary plexus (DCP) of the right eye (Figure 3).

A 30-day follow-up was sheduled and the patient was referrred to anti-VEFG treatment.

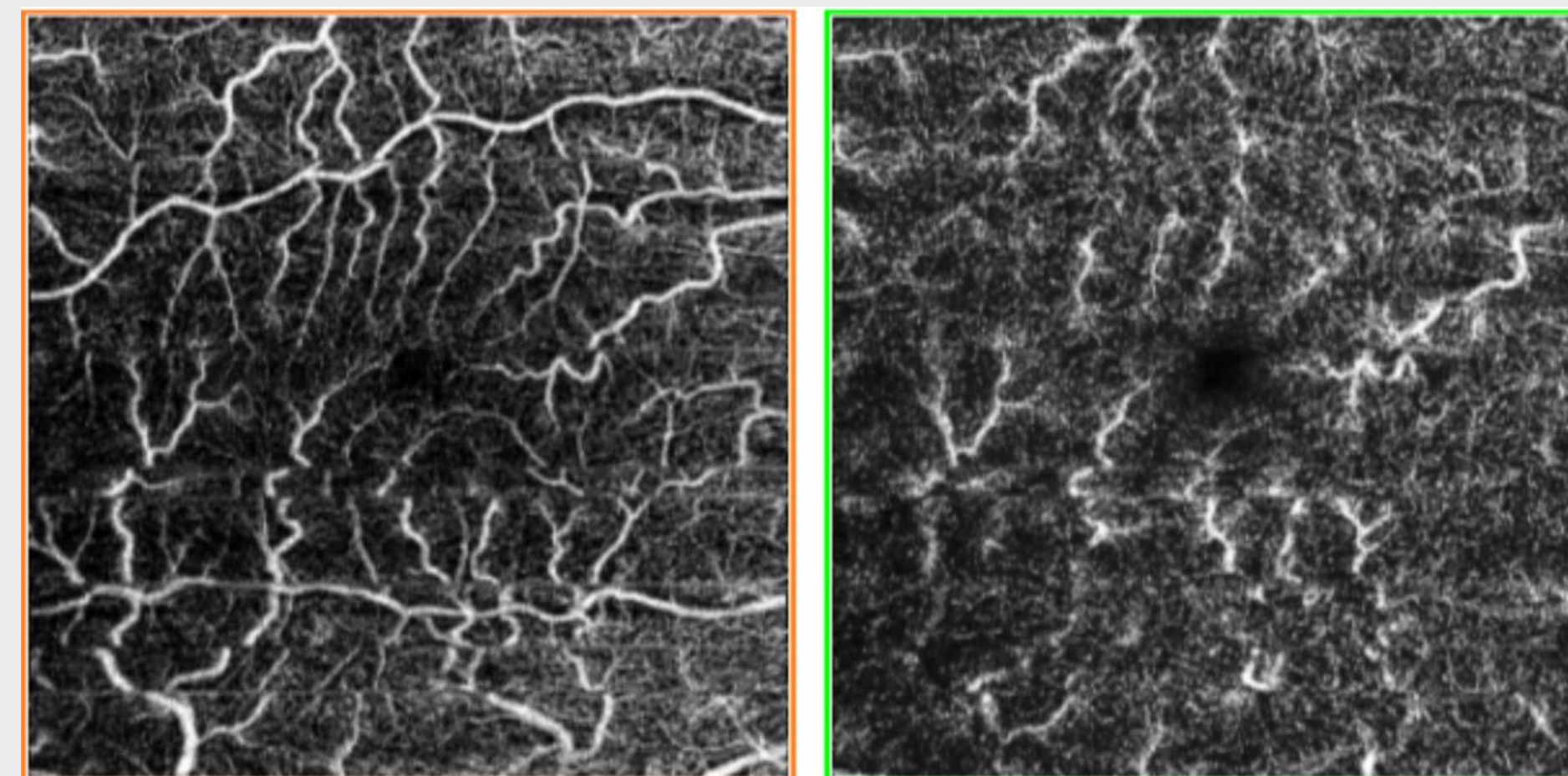


Figure 3. OCTA reveals an area of capillary dropout in deep retinal capillary plexus. On the right, SCP; on the left, DCP.

Discussion

In 2014, Rahimi et al. described a group of patients with nonischemic CRVO associated with PAMM at initial presentation. The pathogenesis is until now not quite understood, but it is believed that a transient hypoperfusion of the DCP is associated with PAMM manifestations. In the setting of CRVO, oxygen saturation of blood diminishes, affecting the most susceptible tissues, particularly, the posterior pole, where high metabolism levels are required. Macular edema was also an uncommon finding in this context, probably because it limits the ability to identify PAMM on OCT.

In summary, CRVO can cause significant vision loss due several mechanism, as macular edema, macular ischemic changes, or as a sequela of complication related to retinal neovascularization. In the absence of these findings, it is important to exclude features suggestive of PAMM, as it can be a cause of vision loss. Nowadays, there is no treatment for PAMM, however, patients should be investigated for cardiovascular risk and systemic ischemia.

References

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