Vitreoretinal traction in impending branch retinal vein occlusion: A pathogenetic role?

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A 25-year-old woman noticed floaters in her left visual field since a few days earlier. She was positive for the heterozygous A1298C mutation on MTHFR gene, beta-fibrinogen gene –455G/A polymorphism, and 4G/5G plasminogen activator inhibitor-1 (PAI-1). Blood test and a systemic workup did not reveal any abnormality. Blood pressure was 120/80 mmHg. Visual acuity was 20/20 in both eyes. Following a complete ophthalmologic examination an impending upper temporal branch retinal vein occlusion (BRVO) was diagnosed (Fig. 1). The patient was followed for one month without any particular treatment. Visual acuity remained unchanged and ophtalmic examination demonstrated improvement in the retinal vascular configuration with complete resolution of retinal hemorrhages, venous tortuosity and congestion (Fig. 2).

BRVO is the second most common vascular disorder of the retina, after diabetic retinopathy. It typically occurs at an arteriovenous (AV) crossing, where the retinal arteriole and venule share a common adventitial sheath (1). Although the pathogenesis of BRVO is not clear, there is evidence suggesting a multifactorial process including genetic, environmental and local factors. Mechanical narrowing of venous lumen may alter the normal blood flow downstream of the AV crossing, leading to turbulent flow. The preexisting endothelial vascular damage from different systemic conditions (arterial hypertension, diabetes mellitus, hyperlipidaemia and haematological disorders) creates a local environment favourable to intravascular thrombus formation, producing eventually occlusion (2). However, several observations suggest that vitreous attachments to retinal vessels and their subsequent traction might also have a significant role in some BRVO cases (1, 4). Coinheritance of three relatively

Figure 1: Findings at first presentation of the patient. A) Fundus photograph of the left eye showing a moderate tortuous and engorged upper temporal retinal vein (white arrows), with some small flame-shaped retinal hemorrhages along the territory of the affected vessel (black arrows). B) Fluoresceinography disclosed delayed venous filling and a localised partial obstruction in the venous lumen near optic nerve (arrow). C) SD-OCT directed to the site of obstruction revealed a vitreovascular traction (arrow) and tissue elevation (asterisks) by an obliquely directed posterior hyaloids. D) SD-OCT dense protocol of false-color maps provided qualitative information of perivascular edema, showing an intense red colour at the involved blood vessel segment.
common genetic conditions in our patient, which is not a rare event, probably increased the relative risk of thrombosis. However, present case points out the role of vitreous traction in the pathogenesis of an impending BRVO and the important diagnostic capabilities of spectral domain optical coherence tomography (SD-OCT) directed to the obstruction site to detect this traction. Although, the intimate mechanism is still unknown, vitreoretinal traction might produce angulation of the retinal vein at the traction site, changing the laminar blood flow, creating turbulence, and increasing the formation of thrombi. Likewise, recent studies have shown that the interaction between the posterior vitreous cortex and the retina seem to play a crucial role for the pathogenesis of a variety of retinal diseases, including exudative age related macular degeneration, diabetic retinopathy or retinal vein occlusion. They suggest several possible mechanisms including vitreoretinal traction inducing chronic low-grade inflammation, retinal exposure to cytokines or free radicals in the vitreous gel, or by interfering with transvitreal oxygenation and retinal nutrition (5, 6). Unfortunately, this patient did not undergo B-scan ultrasonography, which is the most reliable clinical technique to evaluate the posterior vitreous cortex interface, and OCT might be a suboptimal option to detect posterior vitreous adhesion status (7).

Conflicts of interest
None declared.

References