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 ophthalmic 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 in-
creased 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 diag-
nostic capabilities of spectral domain 
optical coherence tomography (SD-OCT) 
directed to the obstruction site to detect 
this traction. Although, the intimate me-
chanism is still unknown, vitreoretinal trac-
tion might produce angulation of the reti-
nal 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 vit-
reous 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 ret-
inopathy or retinal vein occlusion. They 
suggest several possible mechanisms in-
cluding vitreoretinal traction inducing chronic low-grade inflammation, retinal 
exposure to cytokines or free radicals in the 
vitreous gel, or by interfering with transvit-
real 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 inter-
face, and OCT might be a suboptimal op-
tion to detect posterior vitreous adhesion 
status (7).

Conflicts of interest
None declared.

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possible role of posterior vitreous detachment. 
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Figure 2: Findings at the examination after 
one month. A) The fundus had returned to its nor-
mal appearance, with complete resolution of reti-
 nal haemorrhages, venous tortuosity and conges-
tion. B) Fluoresceinography was also normal. C) SD-
OCT at the arteriovenous crossing site revealed a 
spontaneous vitreous relaxation and flattening of 
perivascular retina. D) SD-OCT color-coded maps 
confirmed the decreased retinal thickness at this 
point showing less intensity of red colour.