Unilateral subclinical optic disc edema secondary to vitreopapillary traction syndrome

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A 57-year old male patient reported a 15-min episode of transient amaurosis 2 years back, with persistent blurry vision in the right eye (OD), associated with sporadic headaches over the following 2 years. Visual acuity was 20/20 in both eyes. Fundoscopy of OD revealed mild optic disc edema (ODE) in the nasal sector and vascular changes in the optic disc compatible with venous shunting, corresponding to a prepapillary vascular loop. Fundoscopy of the left eye was normal. Imaging was performed to rule out intracranial hypertension and optic nerve meningioma. Optical coherence tomography revealed vitreopapillary traction syndrome (VTS) in OD, with preservation of the nerve fiber layer and ganglion cells. VTS is characterized by incomplete detachment of the posterior vitreous and adhesion of the vitreous to the optic disc, producing a persistent tractional pull. The main purpose of this report is to highlight the importance of including VTS in the differential diagnosis of patients with chronic ODE.

Key words: Optic neuropathy, posterior vitreous detachment, prepapillary vascular loop, subclinical optic disc edema, vitreopapillary traction syndrome

Vitreopapillary traction syndrome (VTS) is characterized by incomplete detachment of the posterior vitreous and adhesion of the vitreous to the optic disc, producing a persistent tractional pull.[1] VTS can cause chronic subclinical optic disc edema (ODE) and axonal loss of ganglion cells. Symptoms include transient amaurosis and persistent low visual acuity (VA).

The main purpose of this report is to highlight the importance of including VTS in the differential diagnosis of patients with chronic optic ODE.

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Case Report

A 57-year old male patient reported experiencing transient amaurosis in the right eye (OD) for 15 min in 2022. Subsequently, VA in OD was not fully restored, but blurry vision reportedly worsened during sporadic episodes of headache over a period of 2 years.

In May 2023, upon examination at an external ophthalmology service, the affected eye presented ODE and hemorrhage around the disc. No other complaints or previous comorbidities were reported.

Upon examination at our service in April 2024, the patient had a best-corrected VA of 20/20 in both eyes. On fundus examination, the optic disc and cup in OD were normal and without pallor, but edema was observed in the nasal region, with vascular changes resembling venous shunting. The left eye (OS) was normal. The 24-2 visual field test yielded normal findings for OD (except for a slight blind spot enlargement) and for OS. Retinal autofluorescence revealed hypoautofluorescent changes around the nasal sector of the disc, secondary to reduced retinal pigment epithelium density on the nasal side of the optic disc [Figs. 1 and 2].

In view of the presence of chronic ODE in OD and the reported concomitance of headache and transient blurry vision, two diagnostic hypotheses were initially considered: i) intracranial hypertension (IH) with unilateral ODE (despite the fact that ODE is rarely unilateral in IH), and ii) meningioma of the optic nerve associated with chronic ODE, vascular changes resembling venous shunting, and changes compatible with vitreopapillary traction.

Nuclear magnetic resonance of the cranium and orbits yielded normal findings, with no sign of IH or meningioma of the optic nerve in OD. Optical coherence tomography (OCT) scanning of OD revealed edema in the nasal sector, vitreopapillary traction in the nasal region of the optic disc, elevation of Bruch's membrane around the disc, and normal peripapillary nerve fiber layers (NFL) and ganglion cells [Figs. 2-4].

Based on the clinical findings and multimodal analysis, the patient was tentatively diagnosed with VTS in OD. Expectant management was chosen, with ophthalmological follow-up and perimetry. After 1 year of follow-up, the patient maintained the same VA and a discrete campimetric defect maintained in the OD.

Discussion

Posterior vitreous detachment (PVD) involves the separation of the posterior vitreous cortex from the internal limiting membrane of the retina, most often without visual loss.[2] PVD usually starts in the perifoveal macula and progresses toward the peripheral retina, with the optic disc as the final fixation point

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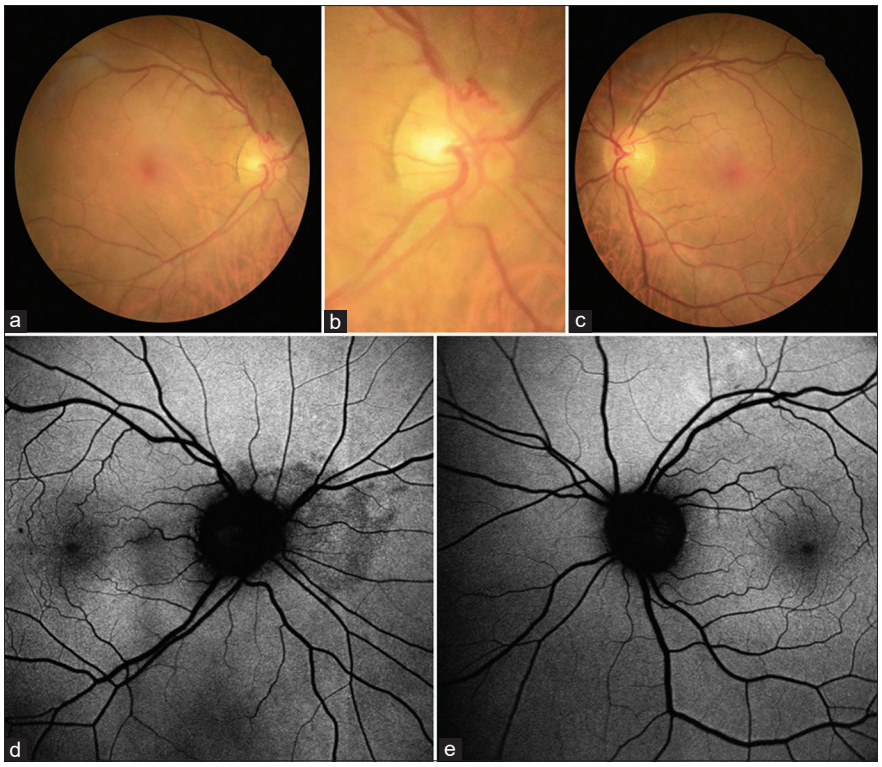

Figure 1: Retinography of the right eye showing optic disc edema in the nasal sector and vascular changes in the upper sector of the optic disc compatible with a prepapillary vascular loop (a, b). Retinography of the left eye, with normal findings (c). Autofluorescence of the right eye showing hypoautofluorescent areas nasally to the optic disc, secondary to reduced retinal pigment epithelium density on the nasal side (d). Autofluorescence of the left eye, with normal findings (e)

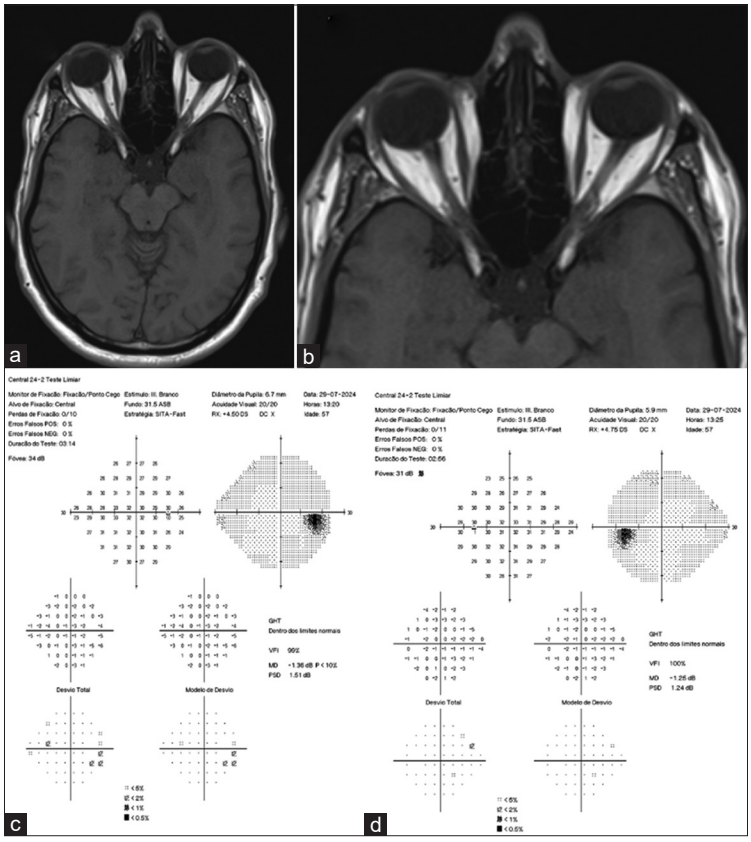

Figure 2: Nuclear magnetic resonance of the orbits, with normal findings (a and b). 24-2 visual field test of the right eye showing slight blind spot enlargement (c). 24-2 visual field test of the left eye, with normal findings (d)

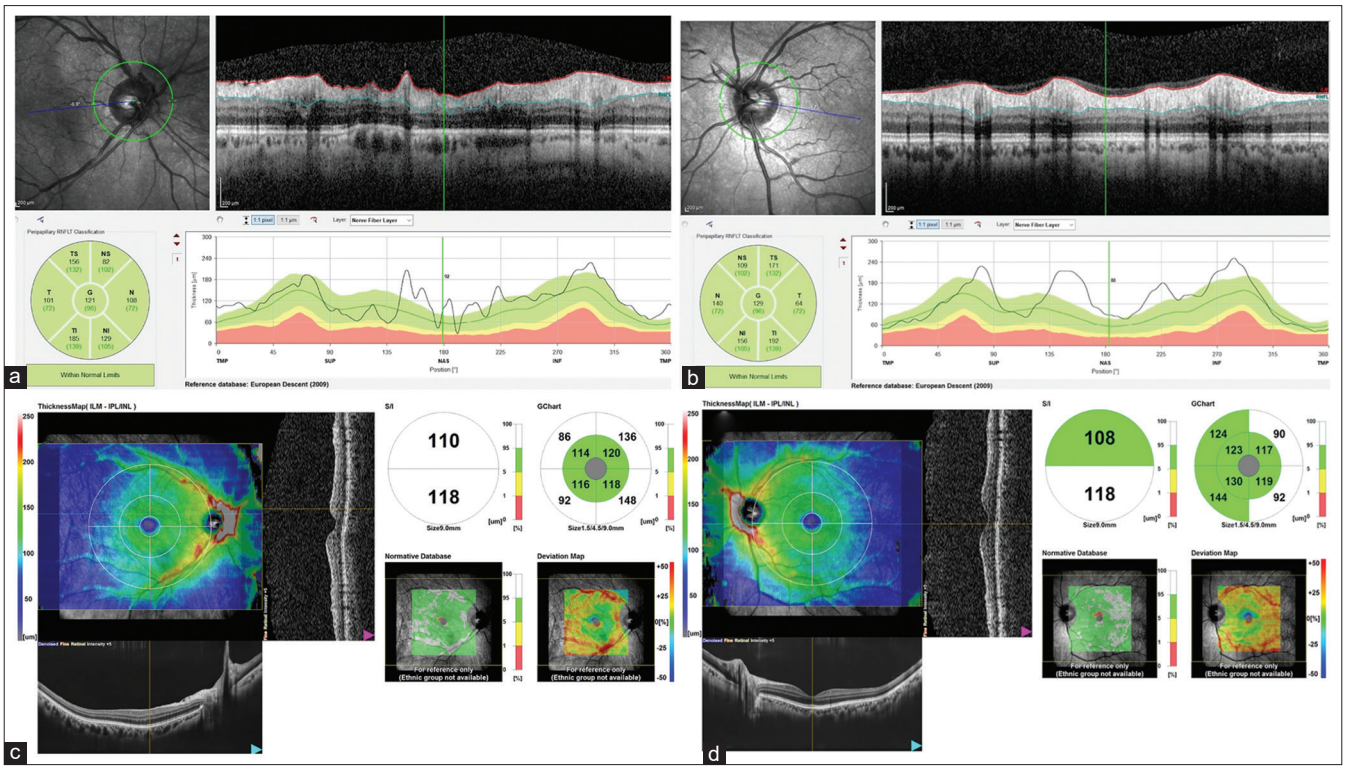

Figure 3: Optical coherence tomography of the right (a) and left eye (b), with normal findings for the peripapillary retinal nerve fiber layer. Optical coherence tomography of the right (c) and left eye (d) showing normal ganglion cells

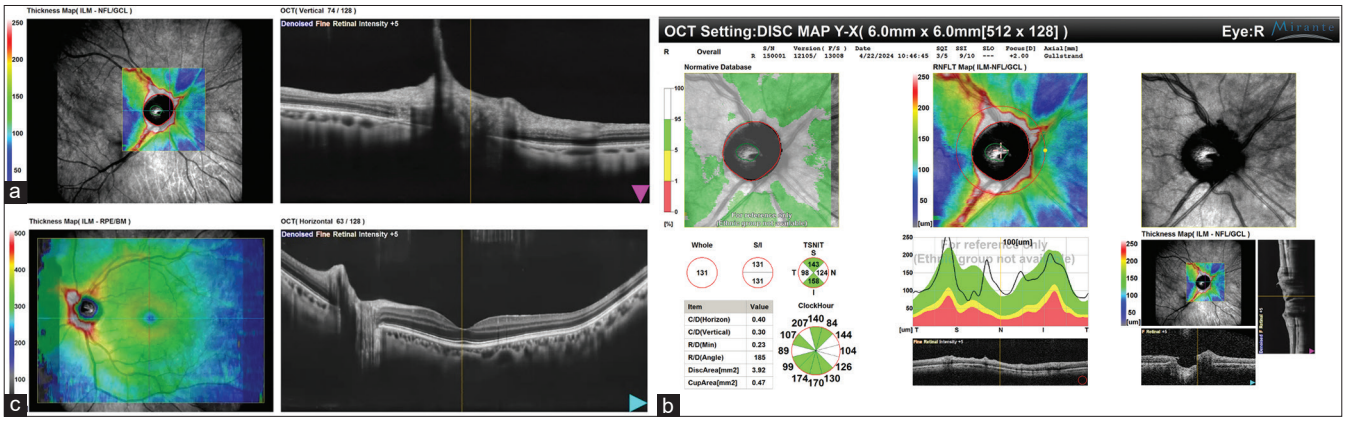

Figure 4: Optical coherence tomography of the right eye showing vitreopapillary traction (a). OCT of the right eye showing mild optic disc edema in the nasal region (b). OCT of the left eye, with no sign of vitreopapillary traction (c)

in most cases.[3] Incomplete PVD, combined with significant dislocation of the vitreous during eye movement, may result in additional traction. The site of vitreous adhesion varies and, depending on the location, may be associated with retinal hemorrhage, vitreomacular traction, retinal breaks, and VTS.[2-4]

VTS can lead to transient or chronic visual loss,[5] and persistent disc traction can cause ODE with neuronal dysfunction. This distortion of the optic disc structures tends to stretch and thin the axons of the ganglion cells, reducing axoplasmic transport.[4]

Risk factors for PVD include old age (usually >50 years), myopia, and previous cataract surgery. In myopic individuals, PVD occurs on average 10 years earlier than in emmetropic and hyperopic subjects.[2] VTS has been observed in association with subclinical ODE or "pseudopapilledema,"[4] diabetic retinopathy,[6] including diabetic macular edema,[7] other macular disorders (macular hole, age-related macular degeneration, macular pucker),[8] epiretinal membrane,[9] and central retinal vein occlusion.[10]

Some VTS patients are asymptomatic, while others are afflicted with photopsia, blurry vision, and shadows or spots in the visual field.[11] Gaze-evoked amaurosis secondary to VTS has also been described.[5]

VTS patients generally have a best-corrected VA of 20/25 or better, mild or absent relative afferent pupillary defect, and sometimes mild dyschromatopsia. Fundoscopy may reveal incomplete PVD, small or hypoplastic disc, disc elevation, juxtapapillary or peripapillary hemorrhage, and focal pallor of the NFL.[5,11]

VTS may be visualized with noninvasive imaging techniques, such as OCT. This technique can show the elevation of the optic disc and the thickening of the NFL and confirm the presence of PVD, with the thickened and hyperreflective posterior hyaloid membrane attached to the optic disc or to its margin.[1,12] In the case reported here, the visualization of VTS on OCT spared the patient further and more invasive examinations, such as lumbar puncture.

The vascular changes observed in the optic disc of OD of our patient were suggestive of a prepapillary vascular loop. This finding has been classified into six morphological subtypes, of which type VI is associated with VTS.[13]

The presence of VTS should be suspected in patients with persistent subclinical ODE and generally preserved visual function. The concomitance of a prepapillary vascular loop and ODE supports the hypothesis of VTS.

Authors' Contributions

AJDG wrote the case report, reviewed the bibliographic references, selected images, and critically reviewed the versions of the report. RSC performed the bibliographic search and contributed to capturing the images for the report. TCS contributed to the bibliographic search, selection of articles, and preparation of the first version of the introduction to the report. FCM critically reviewed the first and subsequent versions of the report. All authors approved the final version of the manuscript.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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