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Authors
Gabriel, Rami
Boisvert, Chantal
Mehta, Mitul

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REVIEW

Review of Vitreopapillary Traction Syndrome

Rami S. Gabriel [a], Chantal J. Boisvert[b], and Mitul C. Mehta [c]

[a]School of Medicine, University of California, Irvine, California, USA; [b]Department of Ophthalmology, Duke Eye Center and Duke University Medical Center, Durham, North Carolina, USA; [c]Gavin Herbert Eye Institute, University of California, Irvine, California, USA

ABSTRACT

Vitreopapillary traction (VPT) syndrome is a potentially visually significant disorder of the vitreo-papillary interface characterised by an incomplete posterior vitreous detachment with the persistently adherent vitreous exerting tractional pull on the optic disc and resulting in morphologic alterations and a consequent decline of visual function. It is most commonly unilateral but bilateral reports have also been described. The cause of the condition may be unknown or idiopathic, although the histology of traction shows proliferation of fibrous astrocytes, myofibroblasts, fibrocytes, and retinal pigment epithelial cells. It is theorised that VPT may induce a congested optic disc with neuronal dysfunction as well as decreased prelaminar flow. The present study reviews and summarises the features, diagnosis, and management of VPT.

ARTICLE HISTORY
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Vitreopapillary traction; optic neuropathy; optic disc; posterior vitreous detachment; visual field loss

Disease entity

International Classification (ICD)

ICD-9:

377.39 Other Optic Neuritis

377.49 Other disorders of optic nerve

ICD-10:

H46.9 Unspecified Optic Neuritis

H47.09 Other disorders of the optic nerve, not elsewhere classified

H47.39 Other disorders of the optic disc

Disease

Vitreopapillary traction (VPT) syndrome, is a disorder of the vitreo-papillary interface characterised by anteroposterior traction exerted by the fibrocellular vitreal membrane pulling at adherent sites on the optic disc or an incomplete posterior vitreous detachment (PVD) causing anatomic and possible functional defects.

History

Moeschlin Sandoz first described effects of traction on the papilla in 1942 with tearing of the glial tissue from the peripapillary region by the shrinking vitreous gel. However, it was Schephens who described the effects of prolonged traction on the papilla causing "pseudopapilledema."[1] Katz and Hoyt then detailed the effects of vitreous traction on the optic papilla in 1994. Their article summarised the condition by (1) intrapapillary haemorrhage, (2) subretinal peripapillary haemorrhage, (3) elevation of the disc, (4) posterior vitreous detachment without complete separation from the disc, and (5) preservation of optic nerve function.[2]

Definitions

Advancements in imaging techniques have allowed better characterisation and definition of VPT. The Katz and Hoyt definition described earlier has been forgone as traction can be objectively apparent on OCT imaging.

Pathophysiology

Senescence leads to age-related liquefication of the vitreous gel as collagen fibres begin to break down. This process is both gradual and abrupt.[3] This uneven liquefication leads to small pockets of fluid surrounded by vitreous, leading to condensation or contraction of the vitreous. Furthermore, fluid may seep through the face of the now porous

CONTACT Mitul C. Mehta mcmehta@uci.edu Gavin Herbert Eye Institute, 850 Health Sciences Road, Irvine, CA, USA.

hyaloid face causing the remaining vitreous to collapse into itself.

As the vitreous loses volume, a tractional pull exerted at remaining sites of attachment occurs. Normally, the forces of traction do not cause significant pathology as the vitreoretinal and vitreopapillary interface undergo changes leading to weakening of attachment sites. It is proposed that detachment first occurs in the superior portion of the retina, along the super and inferior arcades, fovea, and finally at the optic nerve head.[4–6]

VPT has been postulated to occur when adherence to the optic disc site remains despite tractional forces. Histopathological examination on 13 eyes with vitreopapilary traction studied by Roth and Foos showed vitreous fibrils under and within epipapillary membranes, insinuating between glial cells, in every eye studied.[7] Adherence of the posterior hyaloid to the optic disc can be a primary anomaly or secondary to cellular proliferation from diseases that potentiate fibrovascular scaffolds for cellular proliferation and contraction.[2,8–11] Generally, traction specimens under histopathology show proliferation of fibrous astrocytes, myofibroblasts, fibrocytes, and retinal pigment epithelial cells.[12]

The optic nerve head is known to be the strongest site of vitreous adhesion and is typically the last area that separates from the retina in a posterior vitreous detachment (PVD).[13–16] Peripapillary vitreoretinal traction brought on by this process can induce a number of complications at the vitreoretinal interface including optic disc haemorrhage.[2,13–17] More specifically, VPT has been reported to cause isolated intrapapillary, peripapillary, or subretinal haemorrhages, with subretinal haemorrhages being the most common.[13,14] Pathologic features at the optic disc as a result of this condition can differ based on the size and strength of the residual vitreoretinal attachment.[13,17]

Vitreopapillary traction has been shown to cause both temporary or chronic visual impairments.[18] It is theorised that VPT may induce a congested optic disc with neuronal dysfunction as well as consecutive decreased prelaminar flow, causing a nonarteritic anterior ischaemic optic neuropathy (NAAION). Distortion of optic disc structures can lead to stretching and thinning of ganglion cell axons, decreasing the axoplasmic flow.[9]

Diagnosis

History

VPT syndrome generally affects ageing populations undergoing PVDs and patients with diabetes mellitus (likely due to accelerated detachment from earlier vitreous syneresis). There is no known sex predilection for VPT; however, women may be at increased risk as changes in the vitreous in a postmenopausal low-oestrogen state that cause premature vitreous liquefaction.[19]

Physical examination

Blurring of the optic disc margins with an elevated appearance of the disc was pictured by funduscopic examination by Meyer et al. (Figure 1a). The adhesions can extend beyond the disc as seen by the green arrows in Figure 1a.[20] The

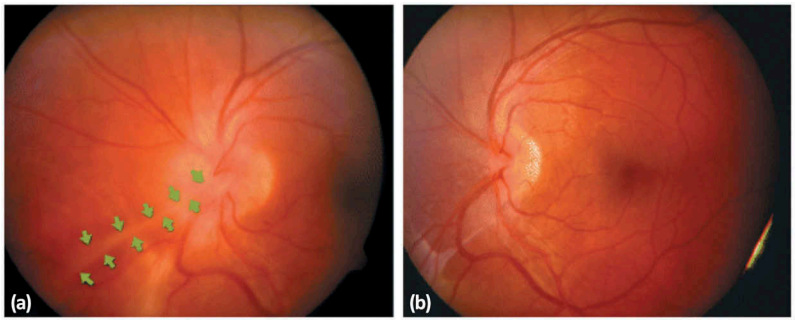

Figure 1. Fundus photographs demonstrating vitreopapillary traction. (a) Photographs of the left eye showing adhesions extending beyond the disc. (b) Postoperative photo showing improvement in retinal vasculature and disc margins.[20]

diagnosis of vitreopapillary traction can be easily overlooked, with multiple studies indicating misdiagnoses prior to the correct diagnosis; clinicians should be well informed of this disease process to direct patients with this diagnosis to the proper care. Meyer et al. show a postoperative image in the same eye showcasing improvement in retinal vasculature without visible traction.

Signs and symptoms

A table from Katz and Hoyt summarises findings from eight case reports of patients with VPT. The signs of VPT can vary from asymptomatic to film over full vision to visual field defects as "smudges or dark spots." A visual field of a patient with VPT is presented, showcasing her complaint of a "black kidney bean smudge" in her vision (Figure 2). Generally, symptoms are temporary but in some cases, especially in those with severe anatomic traction, permanent defects occur.[2]

Clinical diagnosis

Thanks to the micrometre resolution of optical coherence tomography, diagnosis is generally confirmed through imaging of the optic disc; however, ultrasonography detailing the traction can also be used.

Optical Coherence Tomography (OCT)

OCT allows for noninvasive visualisation and imaging of the vitreopapillary interface and has become an important tool in the assessment and management of VPT syndrome (Figure 3). The posterior hyaloid appears as a hyperreflective band marking the end of the hyporeflective vitreous. The hyperreflective band is suggested to be attributable to the bridging fibrocellular proliferative tissue.[22]

OCT has become an extraordinary tool in imaging the optic nerve head in patients with disc haemorrhage as it can differentiate between disc haemorrhages from VPT versus those from progressive optic nerve damage such as glaucoma.[21,23]

Disease monitoring

Routine self-assessment in visual fields may be advised for patients. Repeat testing with OCT of the optic nerve and visual field testing are generally warranted to confirm resolution and to monitor for progression.[9]

Management

Disease monitoring

Observation

The natural history of vitreopapillary traction has only been studied in a few series of collected case reports. Katz and Hoyt followed eight patients presenting with VPT and peripapillary haemorrhage over 6 months. None of the patients developed sequela of visual impairment but none had a full vitreous detachment at the end of that timeframe.[2] Likewise, Hedges et al. also described no long-term sequelae for patients with isolated VPT syndrome.[24] However, in patients with VPT and associated vitreomacular traction (VMT) or other pathology such as central retinal vein occlusion (CRVO), macular hole, NAAION, epiretinal membrane (ERM), or diabetic retinopathy, long-term visual field defects can be observed.[25] Thus, observation should be closely followed with attention to comorbidities as well as severity of the traction.

Surgical management

Surgical management is typically done with a pars plana vitrectomy with separation of the posterior hyaloid causing release of the vitreal traction on the optic nerve head. The release can be completed by cutting vitreous/hyaloid attachments rather than peeling, thereby preventing unnecessary risk of retinal trauma. This has led to good surgical outcomes with one report showing improvement in VA from 20/80 to 20/25 three weeks after surgery.[20] Furthermore, Modarres et al. reported 16 cases of NAAION secondary to VPT that underwent surgical correction with 93.75% having improvement in BCVA.[26] Although not reported in the literature for VPT, 0.3 ml of C3F8 gas has been used with 86% improvement in VMT after a single gas injection and this could be a promising therapeutic option for VPT.[27] While the permanent complications of VPT are more likely to be secondary to continued deprivation of blood or axoplasmic flow, the potential damage to axons

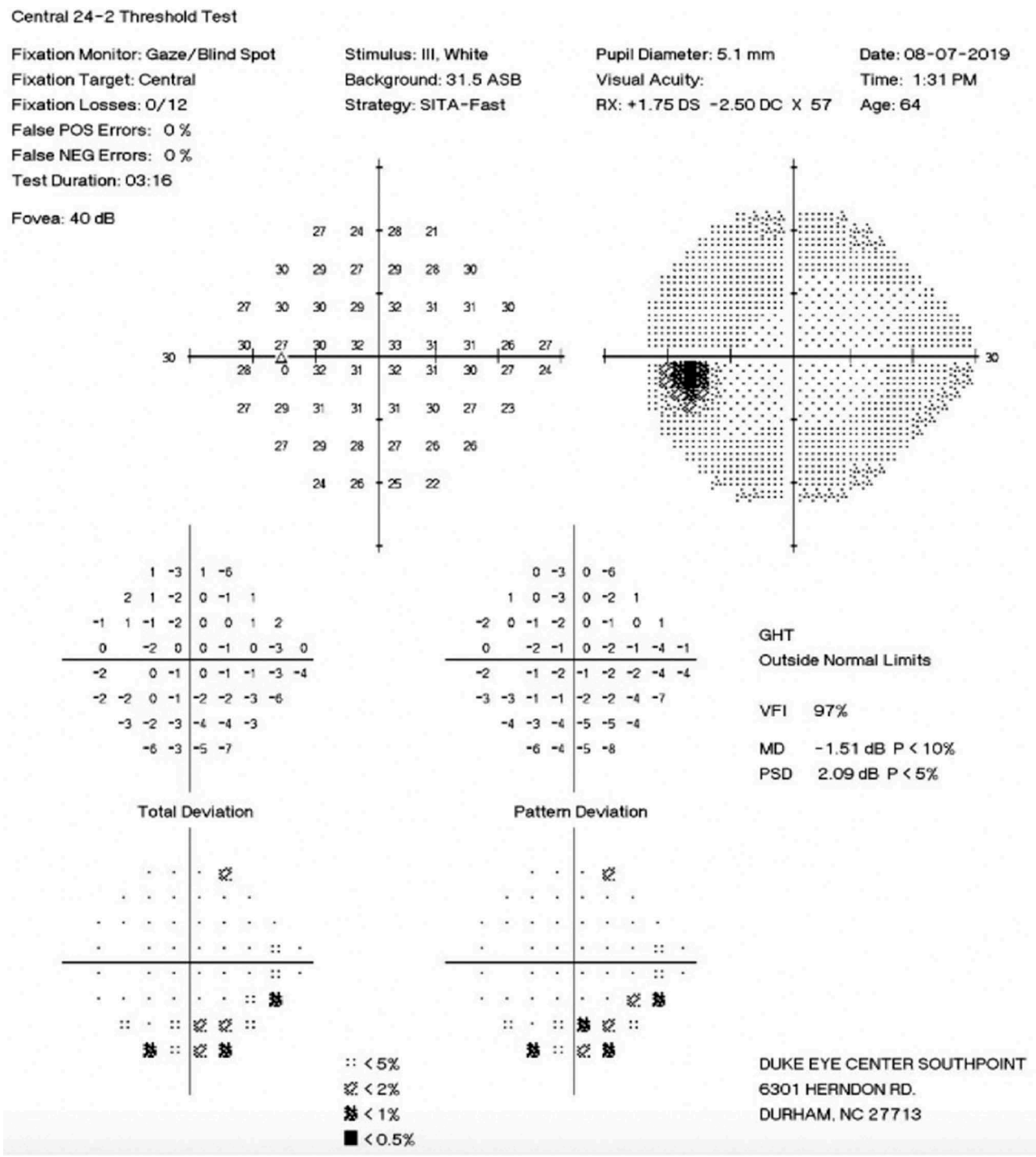

Figure 2. A 24–2 visual field of a patient who presented with the complaint of a "black kidney bean smudge" in her vision. The patient was found to have a VPT.

induced by C3F8 during separation may potentially be outweighed by the long-term benefits.

Surgical release of the VPT syndrome may seem logical to reestablish normal anatomical and functional potentials. There are multiple case reports showing anecdotal evidence that the release of traction improved outcomes and resulted in improvement in visual field loss and no changes in peripapillary or foveal thickness.[19,25] However, surgical management versus observation is still

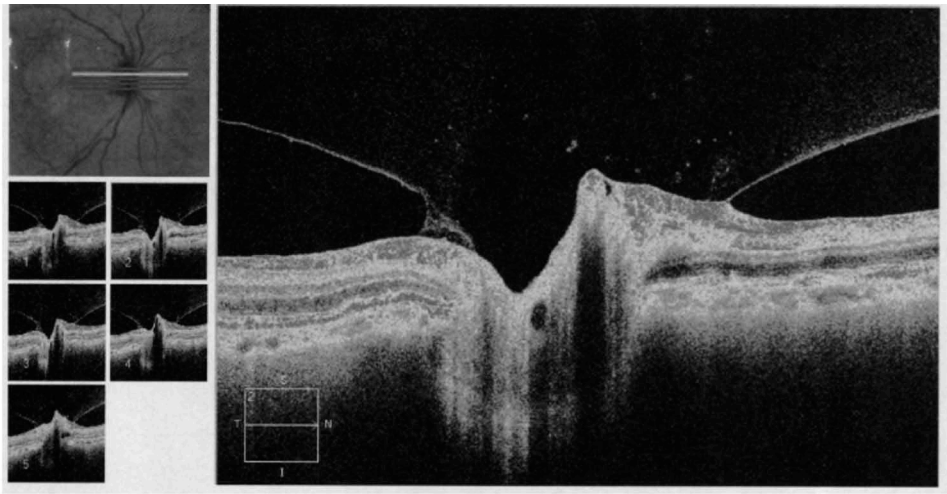

Figure 3. Optical coherence tomography showing various horizontal slices of the vitreopapillary traction centred on the optic nerve.[21]

a contentious point and the majority of the literature suggests observation unless severity or other comorbidities warrant tractional release.

Medical therapy

New research has been ongoing in developing pharmaceutical agents that cause liquefication of the vitreous. Ocriplasmin (formerly microplasmin) is a recombinant form of human plasmin that contains proteolytic activity on proteins anchoring the vitreoretinal interface as well as lacking activity against type IV collagen-an essential component of the inner limiting membrane. This allows for targeted activity in the vitreous while minimising toxic effects to the retina.[19] Trials have been conducted using this agent for VMT including two large randomised controlled phase 3 trials. Comparison of ocriplasmin injection to saline (placebo) has shown resolution of vitreomacular adhesion in 26.5% of eyes compared to 10.1%, respectively. Though these results are promising, many retinal specialists have abandoned the use of ocriplasmin due to its toxicity. Furthermore, its use in VPT would be limited due to the histopathological differences between VMT and VPT.

Other pharmacologic agents that have been investigated include enzymatic agents such as collagenase, chondroitinase, hyaluronidase, dispase, nattokinase, autologous plasmin, plasminogen activators, and non-enzymatic agents such as RGP peptides.[28] Currently, there are no known studies concerning pharmacologic agents for VPT syndrome. However, depending on the results of future studies for VMT, pharmacological therapeutics could prove a promising avenue for the treatment of VPT cases on the border of requiring surgery.

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Declaration of interest statement

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ORCID

Rami S. Gabriel http://orcid.org/0000-0003-0511-435X
Mitul C. Mehta http://orcid.org/0000-0002-5467-6220

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