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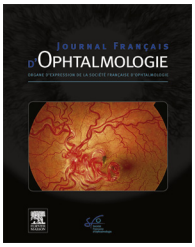

LETTER TO THE EDITOR

Traumatic incomplete posterior vitreous detachment associated with a form of non-arteritic anterior ischemic optic neuropathy

Décollement postérieur du vitré traumatique et incomplet associé à une forme clinique de neuropathie optique ischémique antérieure non artéritique

Introduction

Non-arteritic anterior ischemic optic neuropathy (NAION) is an important cause of visual loss in middle-aged and elderly patients. It results from damage to the prelaminar region of the optic disc. However, its physiopathology and even its etiology is still controversial [1,2].

This unique case shows a temporal association with a traumatic and incomplete posterior vitreous detachment (PVD) in a 17-year-old high hyperopic boy and the clinical picture of a NAION.

Case description

A 17-year-old Caucasian boy was referred to our department for an acute loss of vision in his left eye (LE) for a week. He accidentally received a finger in his LE from his brother. He immediately noticed a severe visual loss and an absolute defect of the superior visual field of his LE. His past medical history is significant for high hyperopia (+ 7.00 in both eyes) without amblyopia. Visual acuity was 20/20 in the right eye (RE) and 20/200 in the chin-up position and with searching movement in the LE. A mild relative afferent pupillary defect (RAPD) was present in the LE. Intraocular pressure was 12 mmHg in RE and 10 mmHg in LE. Anterior slit-lamp examination was unremarkable in RE and only showed a nasal subconjunctival hemorrhage in LE. The Humphrey 24—2 SITA-standard visual field was normal in his RE but in the LE showed an absolute superior altitudinal defect (Fig. 1). Fundoscopy of the LE showed an infero-papillary hemorrhagic PVD (Fig. 2) with an optic disc edema, predominant in the inferior half with retrovitral and peripapillary hemorrhages [3,4], and inner retinal folds [5]. Fundoscopy of the RE was unremarkable. The optical coherence tomography (OCT) clearly showed an incomplete PVD in the LE, with a persistent vitreopapillary attachment superiorly, as medially with a perpendicular adhesion, but inferiorly a complete detachment of the vitreous from the optic disc (Fig. 3). Macular OCT area showed interpapillomacular inner retinal folds [5]. Fluorescein angiography showed an optic disc diffusion especially in the inferior part. The magnetic resonance imaging (MRI) was normal. At the time of patient presentation, because of the severe visual loss and disc edema, the patient was started on intravenous bolus of 500 mg of methylprednisolone for three days but with poor success. The vision response was weak and passed from 20/200 to 20/100. At three-month follow-up, the superior visual field defect is unchanged, with an inferior optic nerve pallor and atrophy (Fig. 4).

Discussion

We report an unusual cause of a severe, irreversible visual loss following a non-penetrating eye injury in a 17-year-old patient. A direct, violent finger impact on the LE caused an immediate central and superior visual field defect. A NAION due to traumatic inferior PVD was diagnosed. The temporal association between the eye trauma and the visual loss allowed to link the two events. The OCT, demonstrating the incomplete PVD in the inferior half of the optic disc, as the fluorescein angiography, illustrating the inferior optic disc diffusion, strongly sustain the relation between the PVD and the optic disc insult.

This case supports the theory of Parsa and Hoyt [1] and Modarres et al. [2] that vitreous traction contributes to so-called NAION. As suggested by Parsa and Hoyt, we think that the traumatic incomplete inferior PVD causes a mechanical traction of the inferior optic nerve head leading to a direct axon injuries and ischemic insult from microvascular involvement [1]. Presence of numerous retinal and pre-retinal hemorrhages along the inferior temporal arcade supports the hypothesis of a direct traction, separation and shearing on vessels from the detached vitreous. These types of hemorrhages in PVD alone does not cause visual loss [3,4]. Mechanical shear damage to axons is implicated in visual loss [1]. This inferior optic nerve damage and edema match with the superior visual field defect.

The peripapillary vitreous cortex is the strongest adhesion site, followed by the macular region [6,7]. In vitreopapillary traction syndrome, the amount and generated force of the remaining vitreopapillary adhesion cause different types of complications [6].

Interestingly, in a study where surgical vitrectomy was performed in young adult monkey eyes, a similar clinical picture with damage to the optic nerve and loss of axons and small superficial optic disc and retinal hemorrhages were observed with an expected visual defect (based on primate retinotopic organization) [8]. In this study, vitrectomy may mimic a traumatic/brisk PVD as in our case and support the

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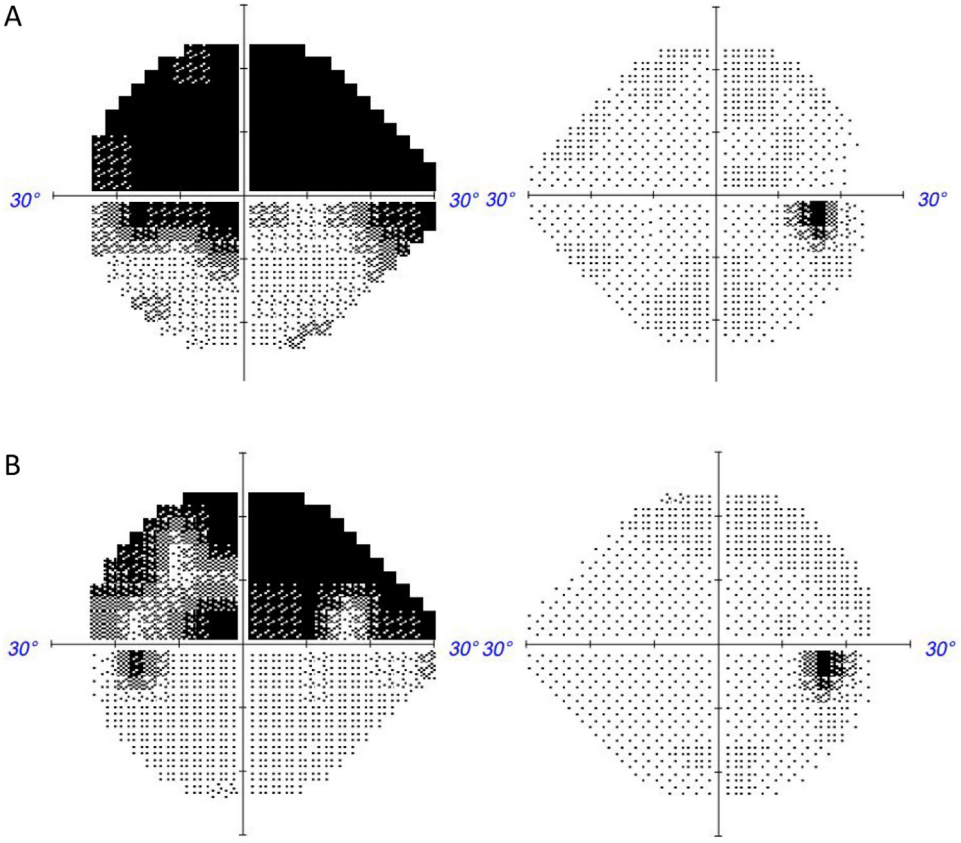

Figure 1. A. Visual field (VF) at presentation. B. At three-month follow-up.

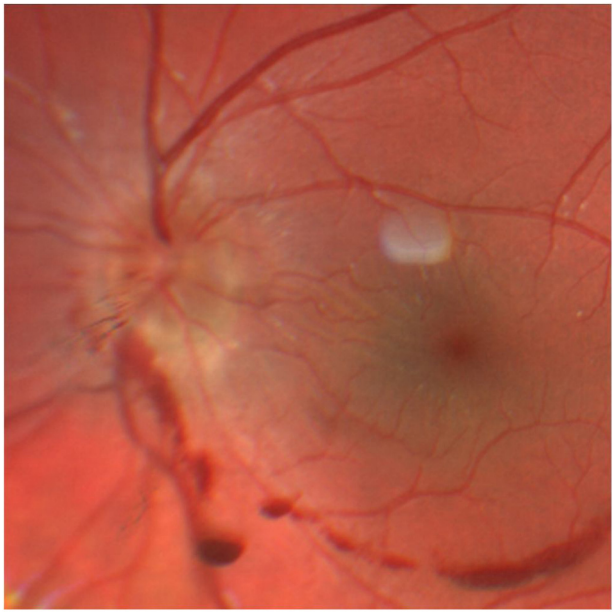

Figure 2. Fundus photography focused on the incomplete papillary vitreous detachment seen in the infero-papillary region and optic neuropathy edema. Retrovitreal and peripapillary hemorrhages are noted with inner retinal folds in the interpapillomacular area.

link between the vitreopapillary separation and the acute visual loss.

Furthermore, the young age and the high hyperopic refractive error of the reported case have probably contributed to the severe and permanent visual loss.

Usually, vitreous adhesions weaken with ageing [7], and, with the vitreous syneresis, the PVD spontaneously occurs [9]. In young patient, the posterior vitreous cortex is strongly attached to the peripapillary region [7]. Furthermore, the reported patient has a severe hyperopia, which

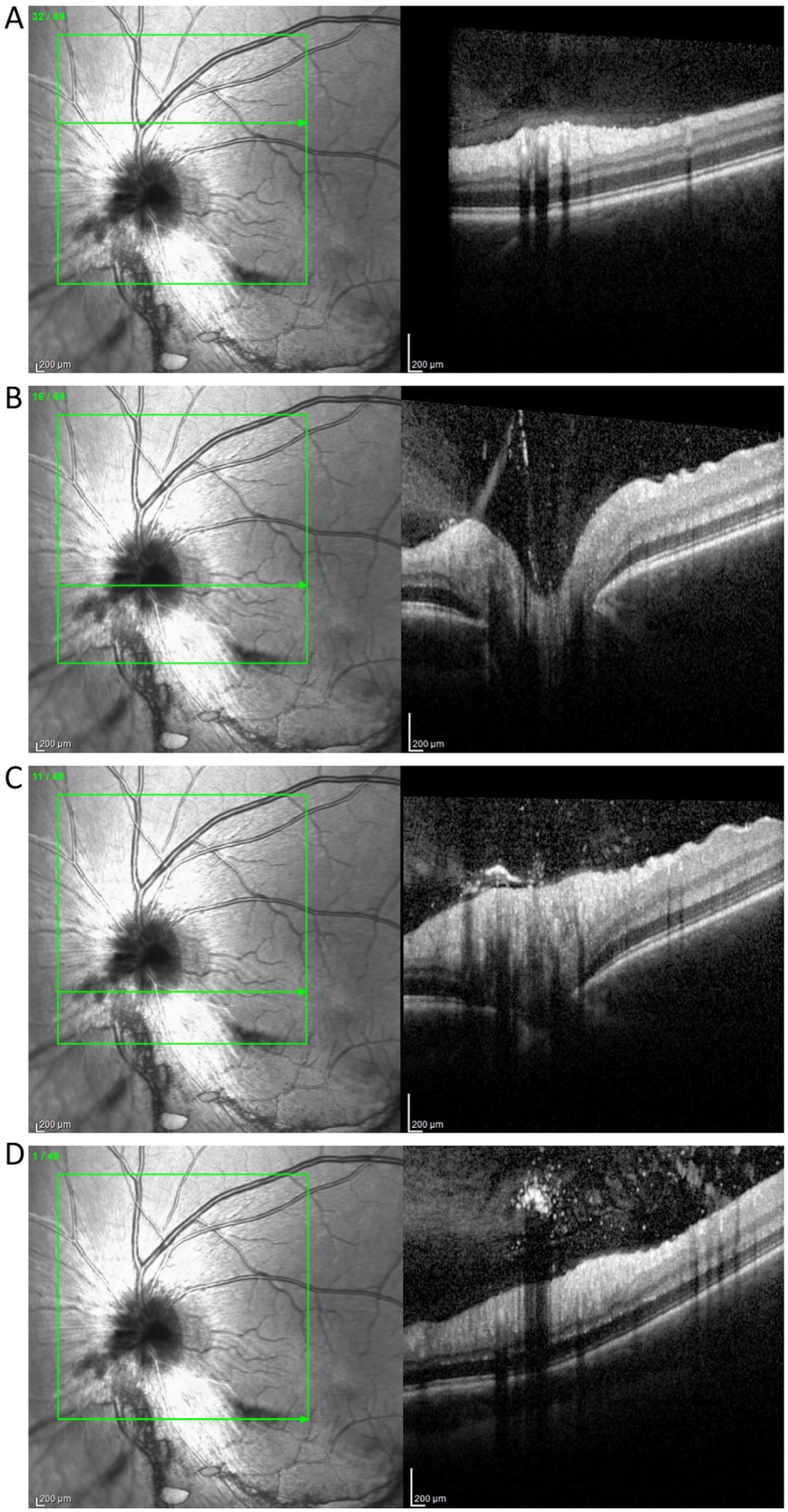

Figure 3. Optic nerve optical coherence tomography at presentation showing: A. Attached vitreous superior to the optic neuropathy (ON). B. Vitreous attached at the middle part of the ON with perpendicular adhesion. C. Interpapillomacular inner retinal folds secondary to traumatic papillary vitreous detachment. D. Detached vitreous inferior to the ON.

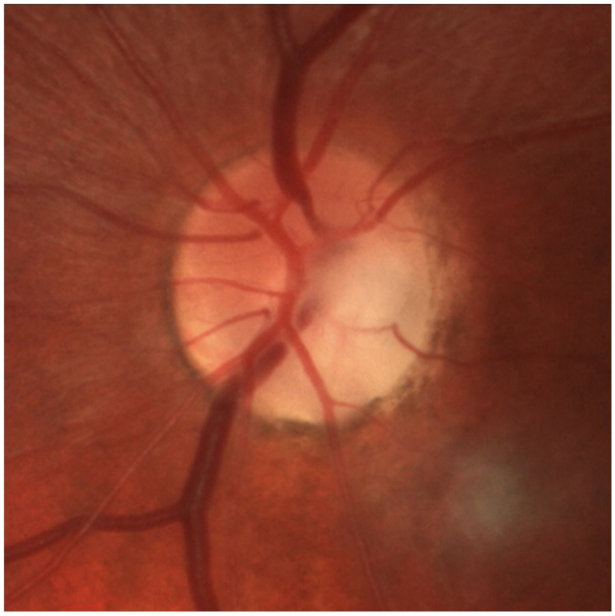

Figure 4. Inferior optic nerve pallor and atrophy with arteriolar narrowing at three-month follow-up.

suggests the presence of a larger and strengthen vitreopapillary adhesion [9]. Hyperopic eyes have a lower prevalence of PVD [10], and the higher the myopia, the younger is the onset age of PVD [11].

The young age (17 years old) and the high hyperopic refractive error (+ 7.00 D) in our case make us believe that our patient had a very strong peripapillary and macular posterior vitreous attachment. It probably explains the severe visual loss caused by the traumatic insult of optic disc axon fibers and vessels from the traumatic detachment of a very strongly attached vitreous.

Conclusion

This clinical case strongly supports the suggested hypothesis of a link between visual loss or visual field defect and vitreopapillary separation. This entity could be called papillary vitreous detachment neuropathy (PVD-N) [1].

Disclosure of interest

The authors declare that they have no competing interest.

References

[1] Parsa CF, Hoyt WF. Non-arteritic anterior ischemic optic neuropathy (NAION): a misnomer. Rearranging pieces of a puzzle to reveal a non-ischemic papillopathy caused by vitreous separation. Ophthalmology 2015;122:439—42.
[2] Modarres M, Sanjari MS, Falavarjani KG. Vitrectomy and release of presumed epipapillary vitreous traction for treatment of non-arteritic anterior ischemic optic neuropathy associated with partial posterior vitreous detachment. Ophthalmology 2007;114:340—4.
[3] Cibis GW, Watzke RC, Chua J. Retinal hemorrhages in posterior vitreous detachment. Am J Ophthalmol 1975;80:1043—6.
[4] Katz B, Hoyt WF. Intrapapillary and peripapillary hemorrhage in young patients with incomplete posterior vitreous detachment. Signs of vitreopapillary traction. Ophthalmology 1995;102:349—54.
[5] Kupersmith MJ, Sibony PA, Dave S. Non-arteritic anterior ischemic optic neuropathy induced retinal folds and deformations. Invest Ophthalmol Vis Sci 2017;58:4286—91.
[6] Gabriel RS, Boisvert CJ, Mehta MC. Review of vitreopapillary traction syndrome. Neuroophthalmology 2020;44:213—8.
[7] Sebag J. Anatomy and pathology of the vitreo-retinal interface. Eye 1992;6:541—52.
[8] Russell SR, Hageman GS. Optic disc, foveal, and extrafoveal damage due to surgical separation of the vitreous. Arch Ophthalmol 2001;119:1653—8.
[9] Johnson MW. Posterior vitreous detachment: evolution and complications of its early stages. Am J Ophthalmol 2010;149:371e1—82e1.
[10] Shao L, Xu L, You QS, Wang YX, Chen CX, Yang H, et al. Prevalence and associations of incomplete posterior vitreous detachment in adult Chinese: the Beijing Eye Study. PLoS One 2013;8:e58498.
[11] Yonemoto J, Ideta H, Sasaki K, Tanaka S, Hirose A, Oka C. The age of onset of posterior vitreous detachment. Graefes Arch Clin Exp Ophthalmol 1994;232:67—70.

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