Vitreous Hemorrhage as the Presenting Sign of Idiopathic Intracranial Hypertension

Amir R. Vosoughi\textsuperscript{a}  Jonathan A. Micieli\textsuperscript{b, c, d, e}

\textsuperscript{a}Max Rady College of Medicine, Rady Faculty of Health Sciences, University of Manitoba, Winnipeg, MB, Canada; \textsuperscript{b}Department of Ophthalmology and Vision Sciences, University of Toronto, Toronto, ON, Canada; \textsuperscript{c}Division of Neurology, Department of Medicine, University of Toronto, Toronto, ON, Canada; \textsuperscript{d}Kensington Vision and Research Centre, Toronto, ON, Canada; \textsuperscript{e}Department of Ophthalmology, St. Michael’s Hospital, Unity Health, Toronto, ON, Canada

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Abstract
Vitreous hemorrhage from papilledema has rarely been reported in the literature. It likely results from an acute increase in intracranial pressure leading to venous compression and rupture. We herein describe a 32-year-old woman who presented with flashes and floaters and was found to have vitreous hemorrhage due to idiopathic intracranial hypertension (IIH). She was treated with acetazolamide, and at 6-month follow-up, regained normal visual function with resolution of optic nerve swelling and hemorrhage. Our case demonstrates that patients with IIH may rarely present with floaters due to vitreous hemorrhage from papilledema. This case also re-iterates that the extent and presence of vitreous and optic disc hemorrhage likely does not confer a prognostic value in patients with papilledema.

Introduction
Vitreous hemorrhage from papilledema is very unusual. It likely results from venous compression and rupture secondary to an acute increase in intracranial pressure (ICP). The extent and presence of hemorrhage has been positively correlated with the degree of papilledema; however, it did not confer any prognostic value after controlling for the severity of papilledema.
in a previous study [1]. Rarely, severe hemorrhaging in idiopathic intracranial hypertension (IIH) may appear similar to Terson’s syndrome seen in patients with subarachnoid hemorrhage [2]. Here, we describe a rare case of vitreous hemorrhage, which was the presenting sign of IIH in an otherwise asymptomatic individual.

Case Report

A 32-year-old woman was referred urgently to ophthalmology for new floaters in the right eye. She had a past medical history of morbid obesity (body mass index 54.9 kg/m²), depression, and her only medication was venlafaxine. She denied regular headaches, pulsatile tinnitus, or transient visual obscurations. She denied any recent weight gain. On examination, her visual acuity was 20/20 in the right and 20/25 in the left eye. Pupils were equal and reactive; there was no RAPD. Dilated fundus exam showed severe, 360-degree peripapillary nerve fiber layer hemorrhages in both eyes (Fig. 1a). Additionally, there was a vitreous hemorrhage in the right eye emanating from the inferior portion of the right optic nerve. Humphrey 24-2 SITA-Fast visual fields were performed and were normal (Fig. 1b).

Her presentation with bilateral optic disc edema and preserved visual function was most consistent with papilledema. She underwent a CT/CTV of the head that was reported as normal apart from signs of raised ICP including an empty sella and distal transverse sinus stenosis. There were no signs of a subarachnoid hemorrhage. The complete blood count and coagulation profile were normal. Lumbar puncture showed normal cerebrospinal fluid (CSF) contents and an opening pressure of 34 cm of water. She was treated with acetazolamide 500 mg PO BID and her optic disc edema improved at the 3-month follow-up. At 6 months, her visual function remained normal, and the optic disc edema and hemorrhages had completely resolved.
Discussion

Vitreous hemorrhage in the setting of IIH has rarely been reported. There was no vitreous hemorrhage reported in the 165 patients studied in the IIH Treatment Trial [3]. In a study that aimed to look at unusual hemorrhages in IIH, 402 consecutive patients with papilledema were assessed and only 5 patients (1.2%) were noted to have a vitreous hemorrhage. Three of these patients had an intracranial malignant process: breast cancer metastasis, glioblastoma, and metastatic tumor, whereas only 2 patients had IIH [4]. We were only able to retrieve three other patients with IIH and vitreous hemorrhage reported in the literature [2, 5, 6]. Raevis and Elmalem [2] described a 39-year-old woman who presented with headache, transient visual obscurations, and blurry vision including floaters. This patient had very good visual function at presentation and was managed with pharmacologic treatment without any loss of vision. The two additional cases were in the pediatric population. Fraser et al. [5] reported a 15-year-old woman with papilledema and optociliary shunt vessels who developed a vitreous hemorrhage and worsening vision. She was treated with optic nerve sheath fenestration and had a final visual acuity of 20/20 OD and 20/25 OS. Watson and Sandford-Smith [6] reported a 14-year-old boy who was otherwise asymptomatic and presented with vitreous hemorrhage and a central retrohyaloid collection of blood. This patient also had normal visual function at final follow-up.

Acute and chronic papilledema can lead to vitreous hemorrhage through various proposed mechanisms. The widely accepted theory is that vitreous hemorrhage results from an acute rise in intracranial pressure, which is then transmitted to the CSF in the optic nerve sheath. The central retinal vein and choroido-retinal vascular anastomosis subsequently become compressed. This leads to venous stasis as blood is not able to exit through the anastomotic channels, which may result in subsequent rupture. Severe hemorrhage may break through the internal limiting membrane and spread to the vitreous. The proposed etiology of an acute rise in ICP leading to hemorrhage is particularly evident in cases where vitreous hemorrhage develops following a subdural injection [7]. Furthermore, in these patients, the risk is dependent on the volume of injection – and therefore the rise in CSF pressure – as vitreous hemorrhage only occurs with large volume injections [7]. A second possibility is that severe optic nerve swelling results in mechanical rupture of small vessels. This is consistent with studies which show nerve fiber layer hemorrhage to correlate with the severity of papilledema as measured using the Friesen scale [3]. In patients with papilledema, vitreous hemorrhage, and subarachnoid hemorrhage (Terson’s syndrome), another possibility is direct expansion of hemorrhage to retinal vessels through perivascular spaces (Virchow-Robin spaces) [8]. In patients with chronic papilledema, vitreous hemorrhage can result from peripapillary subretinal neovascularization [9] or through ruptured optociliary shunt vessels [5]. While raised intracranial pressure is an important diagnostic consideration, many diseases can lead to spontaneous vitreous hemorrhage. Therefore, at the minimum, a complete ophthalmic examination to look for the common causes of spontaneous vitreous hemorrhage must be performed. Common considerations include proliferative diabetic retinopathy, hemorrhagic posterior vitreous detachment, and neovascularization after ischemic retinopathies such as central retinal vein occlusions [10]. It is important that a thorough history is obtained since this may point to a specific cause (e.g., Preceding flashes and floaters would suggest a hemorrhagic posterior vitreous detachment). Both eyes should be examined carefully to look for signs of diabetic retinopathy and other retinal changes. When the vitreous hemorrhage precludes a view of the retina, a B-scan ultrasound should be performed to ensure there is no retinal detachment or other posterior pole pathology.

Patients in the IIH Treatment Trial with nerve fiber layer hemorrhages were found to be more likely to have treatment failure and poor visual outcomes [3]. However, a study
by Micieli et al. [1], showed that after controlling for the severity of papilledema, optic disc hemorrhages do not confer an increased risk. While there is a paucity of data on the prognosis of IIH patients with vitreous hemorrhage, all reported patients have had a visual acuity of 20/25 or greater at final presentation. The preserved final visual acuity is in keeping with the proposed venous mechanism of hemorrhage secondary to papilledema, as opposed to the predominantly arterial hemorrhage seen in conditions such as glaucoma, which is associated with a worse prognosis. In keeping with the previous literature, our patient had preserved visual acuity and complete resolution of hemorrhage at her final follow-up.

In conclusion, patients with IIH may rarely present with floaters from vitreous hemorrhage. The presence of these findings does not necessarily confer a negative prognostic value despite the concerning imaging features, as this case and previous studies demonstrated.

Statement of Ethics

This research was conducted ethically in accordance with the World Medical Association Declaration of Helsinki. Ethical approval is not required for this study in accordance with local or national guidelines. Written informed consent was obtained from the patient for publication of the details of their medical case and any accompanying images.

Conflict of Interest Statement

We have no conflict of interest to disclose.

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Author Contributions

Conception and design, acquisition of data, analysis and interpretation of data, drafting the manuscript and revising it for intellectual content, and final approval of the completed manuscript: Amir R. Vosoughi and Jonathan A. Micieli.

Data Availability Statement

All available data are included in the case description. Further inquiries can be directed to the corresponding author.

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