Clinical Case Report

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Optic disk hemorrhage and vitreous hemorrhage after phacoemulsification in a normal tension glaucoma patient

A case report

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Abstract

Rationale: Optic disk hemorrhage has been closely correlated with glaucoma for its development and progression. Phacoemulsification surgery results in large intraocular pressure (IOP) fluctuation. We report a case of optic disk hemorrhage and consequently progressive vitreous hemorrhage after an unsuccessful phacoemulsification surgery in an advanced normal tension glaucoma (NTG) patient.

Patient concerns: An advanced NTG patient of 82 years old with chronic hypertension underwent an unsuccessful phacoemulsification surgery complicated by posterior capsule rupture. During the postoperative 2 weeks, recurrent episodes of fresh hyphema occurred and B ultrasonography scan revealed the progressive vitreous hemorrhage. The IOP went out of control under the maximum tolerable IOP-lowering medications.

Diagnosis: Vitreous hemorrhage after phacoemulsification in an advanced NTG patient.

Interventions: Vitrectomy was performed to search for the cause of the progressive vitreous hemorrhage. After removal of the thick vitreous hemorrhage, a fresh spot of optic disk hemorrhage was detected at the nasal margin of the significantly-cupping disk.

Outcomes: Postoperatively, the hemorrhagic vitreous opacity gradually resolved and the IOP remained stable at 10 ~13 mmHg with topical prostaglandin analogue drops. Fundus examination revealed the dilated disk vessel with localized angiomatous change at the nasal disk margin.

Lessons: Severe optic disk hemorrhage may occur after phacoemulsification in advanced glaucoma patients. Systemic vascular factors, such as chronic hypertension and old age, and surgical complications, such as posterior capsule rupture and postoperative IOP elevation, would further increase the risk. For phacoemulsification in advanced glaucoma cases, extra care should be taken to control intraoperative IOP fluctuations and monitor postoperative IOP.

Abbreviations: BCVA = best corrected visual acuity, IOL = intraocular lens, IOP = intraocular pressure, NTG = normal tension glaucoma, ODH = optic disk hemorrhage, RNFL = retina nerve fiber layer, VH = vitreous hemorrhage.

Keywords: glaucoma, optic disk hemorrhage, phacoemulsification, vitreous hemorrhage

1. Introduction

Optic disk hemorrhage (ODH) has been closely associated with glaucoma and is considered as an important risk factor for both the development[1] and the progression[2,3] of the disease. Typically, ODH is splinter-shaped along the orientation of the retina nerve fiber layer (RNFL) and can be flame- or fan-shaped in severe cases. It may locate on the optic disk (laminar or prelaminar tissue), cross the disk margin, or on the peripapillary retina. The type of ODH on the disk has been correlated with the morphological change of lamina cribrosa pores as a result of tissue remodeling.[4] In fact, the prevalence and incidence rates of ODH can be higher in open angle glaucoma with normal tension than with high tension.[5–9] We report a case of ODH after an unsuccessful phacoemulsification surgery, which resulted in the postoperative progressive vitreous hemorrhage (VH) in an elderly advanced normal tension glaucoma (NTG) patient with chronic hypertension.

2. Consent

This study adhered to the tenets of the *Declaration of Helsinki* and was approved by the ethics committee of the First Affiliated

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Hospital, College of Medicine, Zhejiang University. Informed consent was signed by the patient for the publication of this report and its related images.

3. Case presentation

An 82-year-old man complained of blurred vision in both eyes, which had started 5 years ago. He had been diagnosed with NTG for about 10 years during a routine check-up and was followed at an outside hospital. He was treated with topical Timolol alone until 1 year ago, Travoprost combined with Dorzolamide hydrochloride was administered to replace Timolol. Other medical history was significant for chronic hypertension and prostatitis, both requiring medications.

On presentation, the best corrected visual acuity (BCVA) was 20/40 in the right eye and 20/100 in the left eye. Intraocular pressure (IOP) was 11 mmHg in the right eye and 14 mmHg in the left eye. On slit-lamp examination, the nuclear sclerosis grade 3 and 4 (LOCS III)[10] with asymmetric cortical opacity was noted, respectively in the right and left eye, and the cortical opacity was close to the visual axis in the left eye. Dilated fundus examination revealed the cupping of optic disk, generalized arteriolar narrowing, arteriovenous nicking, and slightly dilated veins, which were more significant in the left eye. Corneal endothelial cell density was 2061 cells/mm^2 in the right eye and was 1539 cells/mm^2 in the left eye. The optical coherence tomography scan revealed the significant thinning of mean RNFL thickness in the superior and inferior quadrants of the left eye, while only the localized thinning in the inferotemporal sector of the right eye. The standard automated perimetry (Humphrey Field Analyzer, 30–2 pattern) showed the significant visual field loss in the left eye and much less abnormality in the right eye, however, with poor test reliability in both eyes. The B ultrasonography scan revealed the posterior vitreous detachment in both eyes. Cataract extraction was recommended for his left eye and informed consent was signed. Routine preoperative examinations did not reveal any other systemic contraindication for surgery, such as the dysfunction of coagulation.

Phacoemulsification was performed in the left eye. During surgery, the patient complained of discomfort and became agitated as the operation progressed. The depth of anterior chamber was not maintained well due to the likely positive vitreous pressure. At the end stage of cortical aspiration, a small rupture of the posterior capsule was noted. The aspiration was immediately stopped and viscoelastic agents were instilled through the side port before removing the Irrigation/Aspiration needle to prevent further vitreous prolapse. After careful check of the capsule status, a 3-piece intraocular lens (IOL) was planned to insert into the ciliary sulcus. Just then unexpected hyphema occurred, which initially originated from the temporal anterior chamber angle and spread to the nasal angle. The main corneal incision was immediately closed with sutures and the hyphema stopped. The plan of IOL insertion was abandoned considering the risk of further hyphema. After partially removing the hyphema with gentle irrigation, the red reflex of the fundus was observed.

On postoperative day 1, the visual acuity was hand motion and the IOP was 33 mmHg. Thick hyphema clots were observed and B ultrasonography scan revealed the VH which mainly located in the anterior vitreous cavity (Fig. 1A, E). The patient was treated with topical tobramycin 0.3% and dexamethasone 0.1%, IOP-lowering medications, and tropicamide phenylephrine. On postoperative day 4, the hyphema clots resolved in part, however, mild fresh hyphema was noted. Carbazochrome sodium sulfonate tablets were prescribed to prevent further hyphema. The patient was discharged on postoperative day 5 with no fresher hyphema and controlled IOP of 20 mmHg.

On postoperative day 9 during follow-up, the BCVA was still hand motion and the IOP was 28 mmHg. Fresh hyphema was once again observed and B ultrasonography scan revealed the progression of VH which involved the posterior vitreous cavity with a hemorrhagic mass locating in front of the optic disk (Fig. 1B F). On postoperative day 15, the visual acuity went down to light perception and the IOP was 40 mmHg. The hyphema resolved partially without further fresh hyphema, however, B ultrasonography scan showed thick VH with changes of proliferation (Fig. 1C, G).

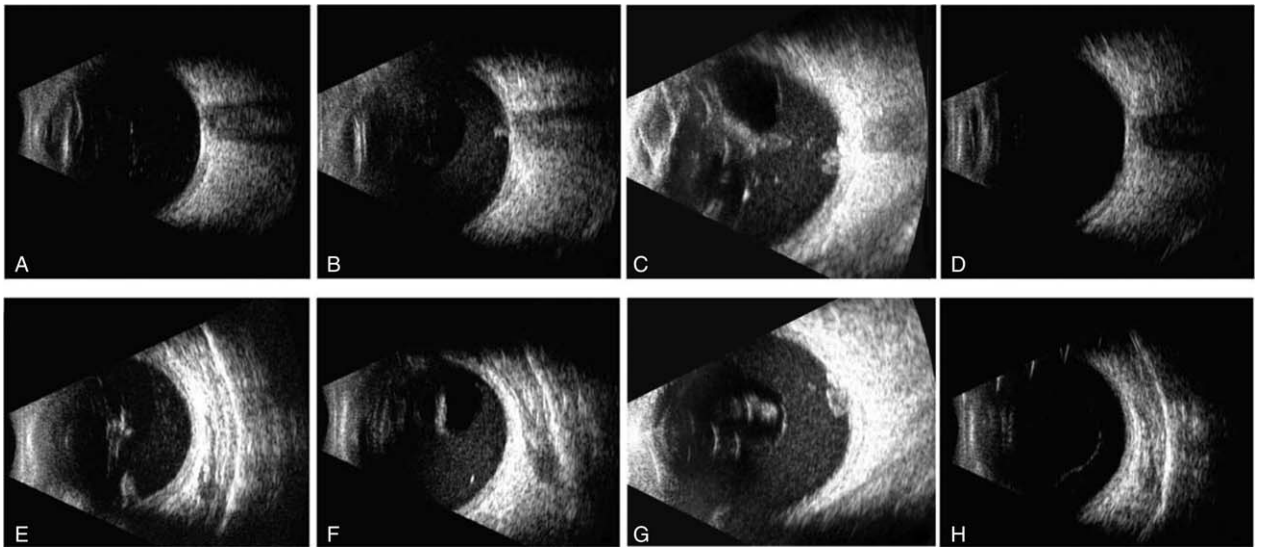

Figure 1. B ultrasonography images of the left eye at different time-points after surgery (A, E, at day 1 after phacoemulsification; B, F, at day 9 after phacoemulsification; C, G, at day 15 after phacoemulsification; D, H, at day 30 after vitrectomy). Upper panels (A, B, C, D) and lower panels (E, F, G, H) show the ultrasound images through the optic nerve and of anterior vitreous cavity, respectively.

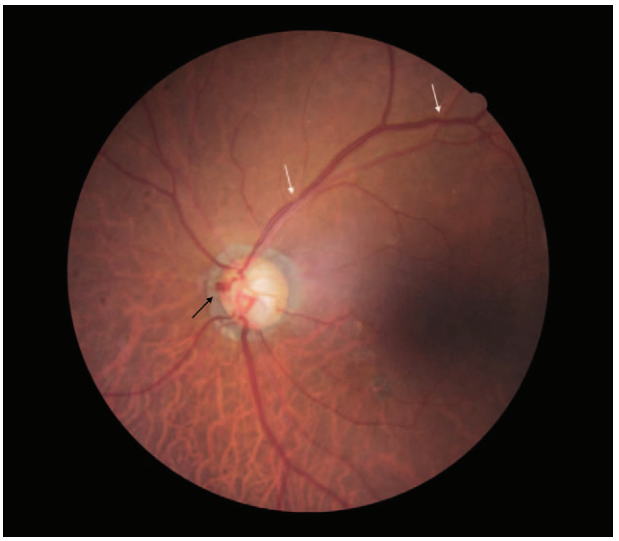

Figure 2. Fundus image of the left eye at day 30 after vitrectomy. Postoperative fundus examination revealed the dilated disk vessel with localized angiomatous change (black arrow) at the nasal disk margin, significant cupping of the disk, generalized arteriolar narrowing, arteriovenous nicking (white arrows), and slightly dilated veins.

With informed consent, vitrectomy was then performed to search for the cause of the progressive VH. After removal of the thick VH, a fresh ODH spot was detected at the nasal disk margin. There was not any other hemorrhagic lesion observed on the retina. A 3-piece IOL was gently implanted into the ciliary sulcus (ZA9003, Abbott Medical Optics). Diffuse oozing of iris capillaries was noted when the intraoperative IOP fluctuated.

On postoperative day 1, the visual acuity was finger-counting and the IOP was 15 mmHg. Hemorrhagic keratic precipitates and vitreous opacity were observed with the optic disk dimly seen. The hemorrhagic vitreous opacity gradually resolved (Fig. 1D, H) and the BCVA was 20/40 at the end of the first postoperative month. During follow-up, the IOP remained stable at 10~13 mmHg with topical Travoprost drops. Fundus examination revealed the dilated disk vessel with localized angiomatous change at the nasal disk margin, and significant cupping of the disk (Fig. 2).

4. Discussion and literature review

In the present case, unexpected progressive VH and recurrent episodes of fresh hyphema occurred during the first 2 weeks after an unsuccessful phacoemulsification surgery in an 82-year-old NTG patient with chronic hypertension. Vitrectomy was then performed to remove the thick proliferative VH and a fresh ODH spot was detected at the nasal disk margin. The dilated disk vessel with localized angiomatous change was further observed at the nasal margin of the significant-cupping disk when the hemorrhagic vitreous opacity resolved postoperatively. The etiology of the present case can be analyzed from the glaucomatous pathological factors and also the undeniable iatrogenic factors.

ODH is widely accepted as a prominent feature of glaucoma, however, its underlying pathogenesis has yet to be fully understood to date.[11] Several likely causes have been discussed, mainly the mechanical mechanism and the vascular mechanism. An early histologic investigation by Quigley et al[12] proposed that the posterior bowing of the lamina cribrosa resulted in the stretching of optic disk capillaries, and then some vessels consequently ruptured under such tension leading to ODH. By retrospective clinical investigations, Nitta et al[13] reported the widening or deepening of RNFL defects was closely associated with the ODH occurrence in NTG patients. Recent investigations with the enhanced depth imaging technology further revealed the strong spatial correlation between the ODH and the glaucomatous lamina cribrosa defect, although the controversy remains about whether ODH is always be precipitated by laminar damage.[14–17] On the other hand, the vascular mechanism discussed the possible correlations between systemic vascular diseases or hemodynamic abnormalities and ODH.[18–22] Kim et al[21] investigated various systemic and ocular variables in a group of 281 NTG patients, and reported the systemic hypertension was the only statistically significant risk factor for ODH. In the present advanced NTG case with the significantly-cupping disk, the deficient connective-tissue support from thin lamina crobrosa would cause small disk vessels more susceptible to mechanical damage. Furthermore, the 82-year-old patient suffered from chronic systemic hypertension and retinal vessels had been subject to long-term hypertensive damage. The advanced glaucomatous disk damage (thin lamina crobrosa for support and the stretching of disk vessels) and systemic vascular factors (chronic hypertension and old age) may have put the patient at high risk for ODH.

Phacoemulsification is performed in a relatively closed intraocular space with irrigation and aspiration. Large IOP fluctuations occur during surgery, which may repeatedly exceed 70 mmHg, while drop close to 0 mmHg at times.[23] Substantial elevation of IOP may also occur in the early postoperative period, which is more frequently noted in glaucoma patients.[24,25] Transient high IOP could cause the enlargement of optic disk cupping.[26] Animal model investigations reported short-term elevation of IOP resulted in the significant deformation of optic nerve head and posterior bowing of the peripapillary sclera in glaucoma eyes.[27] Evidence has shown the supraphysiologic short-term IOP fluctuation does not commonly lead to an increased incidence of ODH in glaucoma eyes,[28] however, the intraoperative posterior bowing of lamina cribrosa may inevitably increase the risk of disk vessel rupture. In our case, the intraoperative rupture of posterior capsule may even worsen the situation although immediate treatments were performed to maintain the anterior chamber. It is unclear whether the ODH occurred during the surgery as the VH of the anterior vitreous cavity in early postoperative period might simply originate from the hyphema. We speculate the additional postoperative elevation of IOP eventually led to the rupture of disk vessel at the nasal margin resulting in postoperative VH, while in turn, the VH further elevated the postoperative IOP resulting in the progressive ODH and VH. The IOP was finally under control after surgical removal of the VH. Unexpected episodes of intraoperative and postoperative hyphema may share the similar systemic and iatrogenic pathogenesis with the ODH. Large IOP fluctuations led to the mechanical stretching of sclerosing angle vessels and led to their ruptures.

5. Conclusions

Severe ODH may occur after phacoemulsification surgery in advanced glaucoma patients. Systemic vascular factors, such as chronic hypertension and old age, and surgical complications,

such as posterior capsule rupture and postoperative IOP elevation, would further increase the risk. Ophthalmologists should be aware of this potential complication and appropriately counsel their patients. For phacoemulsification in advanced glaucoma cases, extra care should be taken to control intraoperative IOP fluctuations and monitor postoperative IOP.

Author contributions

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