Clinical Case Report

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Abnormal increase of intraocular pressure in fellow eye after severe ocular trauma

A case report

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Abstract

Background: An ocular injury can lead to secondary glaucoma in the traumatized eye in 3% to 20% of cases. Literature on the risk of developing elevated intraocular pressure in the nontraumatized fellow eye is scant. Clinicians treating ocular traumas should also bear in mind sympathetic ophthalmia, a rare bilateral granulomatous panuveitis following accidental or surgical trauma to 1 eye.

Case report: We report a case of high-pressure glaucoma of the fellow eye without any signs of uveitis. The left eye of a 24-year-old man was injured in an inadvertent movement during a free-time table-tennis match. The eye was severely crushed, leading to blindness. His right eye developed medically uncontrolled high-pressure glaucoma only 1 month after the injury.

Conclusion: To the best of our knowledge, there are no previous reports of post-traumatic glaucoma in the nontraumatized eye after open-globe injury.

Abbreviations: BMI = body mass index, IOP = intraocular pressure.

Keywords: fellow eye, glaucoma, intraocular pressure, trauma

1. Introduction

It is well known that an ocular injury can lead to secondary glaucoma in the traumatized eye. The mechanism of pressure elevation is often multifactorial.[1] One of the main reasons is tearing of the anterior chamber angle and iris root, which causes posterior angle synechias and angle recession formation, leading to a decrease in the outflow of the aqueous humor and thus an increase in intraocular pressure (IOP).[2–5] The IOP may increase even years after the injury. The probability of developing elevated IOP varies between 3% and 20%. Blunt trauma underlies the majority of trauma-related glaucomas.[3,4,6–9] There are only a few reported cases on the risk of developing glaucoma in the nontraumatized fellow eye.[9] Sympathetic ophthalmia is a rare bilateral granulomatous panuveitis following accidental or surgical trauma to 1 eye. According to the literature, the risk of sympathetic ophthalmia after open-globe injuries is estimated to be 0.2% to 0.5%, but the increase in IOP is not related to this.[10]

This case report is published with the patient's permission. It describes an unusual elevation of IOP in a young man after severe perforating ocular trauma in the fellow eye.

2. Case report

A 24-year-old male student injured his left eye in a table-tennis match when bending over the ball and hitting his head and eye on a metal balk. He is myopic and he had undergone strabismus operations in early childhood due to intermittent exotropia with otherwise healthy eyes. There were no heritable eye diseases among his near relatives. He had not previously had long-term corticosteroid treatments. He is obese (BMI [body mass index] = 41 kg/m^2) and under treatment for diabetes (oral therapy) and arterial hypertonia. In addition, he has symptoms of stress asthma. He was using contact lenses (–6.0 cyl –1.25 ax 10/–3.5 cyl –1.25 ax 170) at the time of the accident. His left eye was seriously crushed, leading to a severe perforating open-globe trauma, for which he was immediately hospitalized.

2.1. The left eye

A large superotemporal scleral perforation with prolapse of the uvea was observed, the cornea was clear and intact, but the lens was luxated and he had a choroidal hemorrhage. Cefuroxim 1.5 g × 3 i.v. was initiated immediately and the perforated sclera sutured the following morning. The CT scan of the globe and brain taken a day after the trauma revealed soft orbital contusion with no bone damage. Eighteen days later a reoperation was required due to late detachment of the retina; pars plana vitrectomy and injection of silicon oil and retinal endophotocoagulation were performed. In spite of all efforts, retina reablation was detected 1 month later, and no further operations were indicated due to poor prognosis. The left eye is permanently blind.

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As in all trauma cases, the patient's injured eye was intensively medicated with topical corticosteroid drops. A dose of 1.0% prednisolone was administered topically every second hour, overlapping with antibiotic drops (0.5% levofloxasin) during the first 4 days. Thereafter a combination steroid–antibiotic eye drop (0.1% dexamethasone–0.2% chloramphenicol) was used 6 times per day during the following 2 weeks until the vitrectomy. Postoperatively the dose of topical combination eye drops (0.1% dexamethasone–0.2% chloramphenicol) was reduced to 4 times daily. After 6 weeks, the corticosteroids were discontinued. Several months later 0.1% fluorometolone twice a day was started by reason of a slight anterior chamber reaction.

An elevated IOP of 38 mm Hg (normal range 9–21 mm Hg) was measured once in the traumatized left eye a day after the reoperation and injection of silicon oil. The eye pressure rapidly normalized during the first postoperative weeks with topical drugs (a timolol–dortzolamide combination), and antiglaucomatous medication was thereafter discontinued. Since then the IOP has been constantly <21 mm Hg.

2.2. The right eye

The fellow right eye was carefully examined after the first emergency operation on the injured eye. Visual acuity was normal 1.0 (−8.0 cyl −4.50 ax 5). The IOP was 21 mm Hg. The conjunctiva was normal and the anterior chamber showed no inflammatory cells, the lens was clear, there were no signs of active diabetic retinopathy, the optic nerve head was considered physiologic with extensive pigmentation around the optic nerve head. Images of the fundus and nerve layer were taken. Unexpectedly, 3 weeks after the trauma, on the day after the elective vitrectomy of the traumatized eye, the pressure in the fellow right eye was 42 mm Hg. Pressure-lowering medication was initiated with a timolol–dortzolamide combination drug. The IOP rapidly fell below 20 mm Hg. However, 2 months later it increased to 61 mm Hg. The optic nerve head still appeared clinically unchanged with practically normal visual field. Maximal topical glaucoma medication was implemented. The pressure was again lowered until the 2 months check-up, when it was again 62 mm Hg. The patient evinced no symptoms. Due to suspicion of noncompliance, the patient was hospitalized. However, during the following days, in spite of all various antiglaucomatous drugs, including mannitol infusion, the IOP was not reduced <40 mm Hg. There were no signs of inflammation, but iris processes were seen in the chamber angle.

A shunt device (Molteno 3 Implant GS Single Plate 175 mm^2, Molteno Ophthalmic Ltd, Dunedin, New Zealand) was placed in the superotemporal quadrant of the right globe 7 months after the initial trauma to the left eye. The shunt was ligated with resorbable thread (polyglactin 910, 6-0 Vicryl Rapide, Ethicon, Guaynabo, PR) and Sherwood slits were incised. Postoperatively the IOP was initially low due to the filtrating slits and the subsequent resorption of the Vicryl thread, when the shunt opened. Two months after the shunt operation, the IOP again increased to 56 mm Hg. Topical glaucoma medication and oral acetatsolamide were initiated whereas topical corticosteroid was tapered. The IOP normalized. When blood samples for diabetes were drawn, the thyroid tests appeared to be abnormal. P-TSH was 6.6 mU/L (0.27–4.2 mU/L), P-T4-V 18.4 pmol/L (11.0–22.0 pmol/L), and later thyroidea antibodies were seen to be abnormal P-TPOAb 113 kU/L (0–<34 kU/L). A consultant in internal medicine prescribed thyroxin treatment.

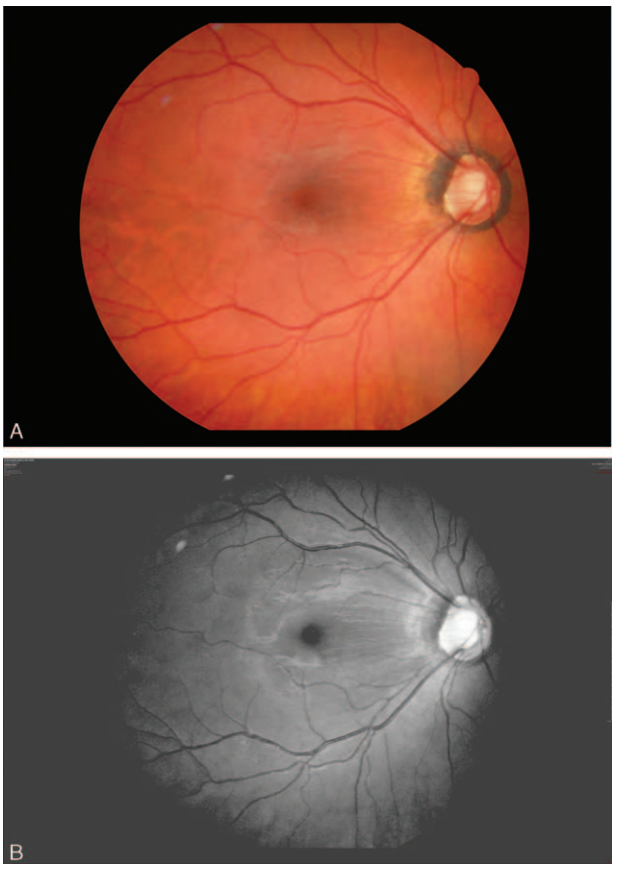

Figure 1. Fundus and nerve fiber layer images of the nontraumatized fellow eye 18 months after open-globe injury to the left eye.

A corticosteroid-containing drug (0.1% dexamethasone–0.2% chloramphenicol) was first time administered into the right eye 3 days before the glaucoma device operation. The dose of 0.1% dexamethasone was 1 drop 4 times daily. This medication was maintained for 8 weeks.

About 2 years after the initial trauma, the best corrected (−7.50 −3.50 ax 3) visual acuity in the right eye is still normal 1.0 and the ocular pressure is 19 mm Hg with submaximal antiglaucoma medication (timolol–dortzolamide combination drug and tafluprost). The visual field and the optic nerve head are considered unchanged (Fig. 1). The left eye is blind, with normal pressure. The left eye now shows a few cells in the anterior chamber and the patient therefore uses fluorometolone in his left eye. In addition, he takes metformin 500 mg 2 × 2 for his diabetes, ramipril 10 mg × 1 for arterial hypertension and thyroxin 0.1 mg 1 × 1 for his hypothyreosis. Although the patient's life changed completely due to the ocular trauma, he has successfully adapted and developed strategies to cope with having a chronic eye disease in the noninjured right eye, in addition to a blind left eye, which caused him to change his profession from mechanics to commerce.

3. Discussion

The patient has no family history of glaucoma, but has diabetes and myopia which are regarded as risk factors for glaucoma.[11] The role of obesity in relation to IOP and glaucoma would seem

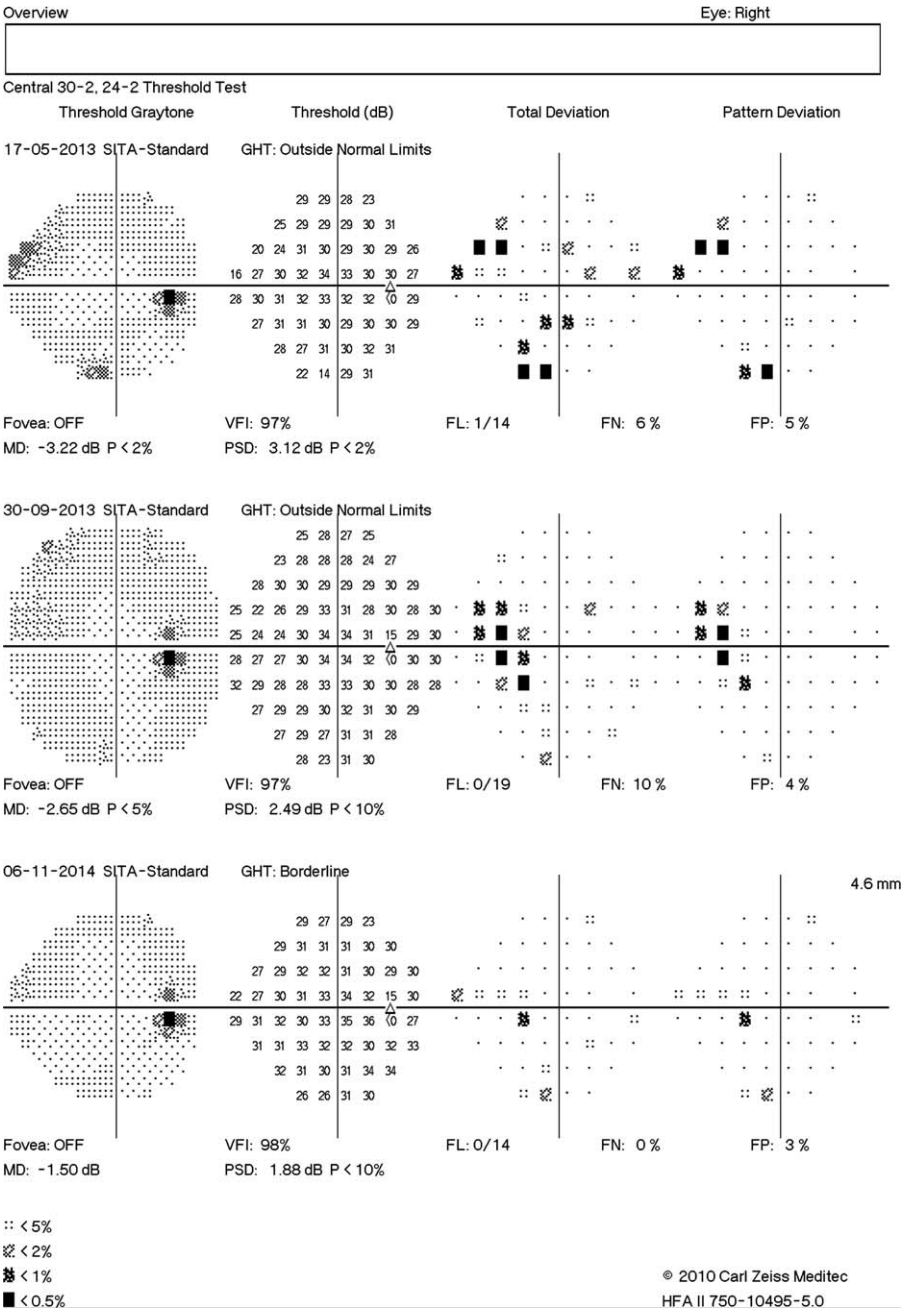

Figure 2. Visual fields in the right eye with long-term fluctuation interpreted as no progression.

to be controversial.[12–14] As the patient evinced no symptoms concomitant with IOP increases > 60 mm Hg, he may also have had symptomless IOP spikes before the left eye was injured. In fact, the first nerve fiber layer image was already abnormal even though it was taken only 3 days after the injury. A slight narrow superotemporal glaucomatous defect was already apparent. During follow-up this localized nerve fiber layer has not so far progressed (Fig. 1). His visual field showed only long-term fluctuation, interpreted clinically as no progression (Fig. 2). It is possible that the severe accident aggravated the course of otherwise symptomless high-pressure glaucoma.

According to the literature, the risk of developing angle recession glaucoma is 5% to 20% after eye injury. The risk is increased with the magnitude of recession, typically > 180 degrees. Among subjects developing glaucoma, up to 50% will eventually also develop glaucoma in the fellow eye.[9] Tesluk and Spaeth[9] have reported a study with a follow-up of 34 years after the trauma. Thirteen injured eyes and fellow nontraumatized eyes were diagnosed as glaucomatous by visual field and optic nerve head evaluation. The fellow eyes developed glaucoma or were in the process of developing glaucoma, during a mean of 4.7 years. There are, however, no reports in the literature on post-traumatic glaucoma in the nontraumatized eye after *open-globe* injury to the fellow eye.

It is well known that corticosteroids can raise intraocular pressure. The possibility of high cortisone response is more likely with certain risk factors such as type 1 diabetes, high myopia, primary open-angle glaucoma, or angle recession glaucoma. A

corticosteroid-induced increase in pressure may occur with any mode of administration, but is much more common with topical or intravitreal corticosteroids than with other modes.[15–18] The IOP usually rises 3 to 6 weeks after the introduction of topical steroids, but elevation of IOP can be found as early as the first or second week.[18] It has even been reported that when 1 eye is treated with topical steroids, the contralateral untreated eye may be affected by the systemically absorbed steroid.[19–20] In our case, the patient has several predisposing risk factors for cortisone response. In addition, the abnormally high intraocular pressure in the fellow eye (61 mm Hg) was first measured only 3 weeks after intensive corticosteroid treatment of the injured eye (23 mm Hg). Thereafter, the IOP in the fellow eye remained high, whereas the trauma eye was normotensive. In summary, it is possible that intensive topical cortisone medication to the injured eye induced a high-pressure glaucomatous cascade in the fellow eye. It is also possible that the injured eye was so seriously damaged that the IOP no longer reacted to steroids. In addition, we have to note that a glaucomatous defect was already seen in the nerve fiber layer image taken 3 days after the trauma, which would imply that the patient had possibly already had symptomless open-angle glaucoma.

Sympathetic ophthalmia has been associated with ocular contusion, open-globe trauma, glaucoma surgery and treatment of ocular malignancies.[10] The typical ocular signs of sympathetic ophthalmia are cells in the anterior chamber, mutton-fat keratic precipitates, small nodules at the level of the retinal pigment epithelium and thickening of the uveal tract in the nontraumatized fellow eye. The precise mechanism of action is not known, although as far back as 1840 MacKenzie and associates hypothesized inflammatory spread from the affected to the sympathizing eye via optic nerve chiasm.[21] More recent studies suggest that the condition is mediated by an autoimmune response to ocular self-antigens. The time between the ocular damage and the development of sympathetic ophthalmia varies considerably; the sympathizing eye usually presents with inflammation within 3 months (~80% of patients) after injury, the time-span ranging however from 5 days to 50 years.[10,21,22] It has been assumed that sympathetic ophthalmia can be prevented by enucleation of the injured eye within 2 weeks from injury.[21,23] However, once the disease process has set in, the benefit of enucleation remains a matter of controversy.[21,23,24]

In conclusion, according to the literature, the masked IOP elevation in the noninjured eye of the present case is not a sign of sympathetic ophthalmia. Instead, it is more probable that primary open-angle glaucoma was incidentally diagnosed at the time of the ocular injury. The accident may have aggravated the course of symptomless high-pressure glaucoma and the situation was worsened by the patient's high sensitivity to corticosteroids. The decision not to enucleate the blind symptomless left eye nor to initiate modern immunosuppressive therapy was thus justifiable.

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