CASE REPORT

Persistent Post-Traumatic Ocular Hypertension with Extensive Angle Recession Following Paintball Injury

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ABSTRACT

Blunt ocular trauma may cause acute and persistent intraocular pressure (IOP) elevation through hyphema, inflammation, trabecular injury, and angle recession. A 36-year-old man presented after a paintball injury to the left eye with visual acuity of hand motions and an IOP of 45 mmHg. Examination revealed minimal hyphema, a Vossius ring with an associated membranous appearance, and dense vitreous hemorrhage. After hyphema resolution, gonioscopy demonstrated discontinuous angle recession involving all four quadrants, with a cumulative extent of approximately 240°. One month after injury, progressive traumatic lens opacity and persistent vitreous hemorrhage necessitated combined phacoemulsification, intraocular lens implantation, and pars plana vitrectomy. Early postoperatively, IOP increased to 32 mmHg and additional topical therapy was initiated after ocular inflammation had subsided. At the third postoperative month, IOP was 15 mmHg and all antiglaucomatous medications were discontinued; one month later, untreated IOP increased to 26 mmHg. Once-daily carteolol hydrochloride 2% reduced IOP to 17 mmHg at the 6-month postoperative visit. Final visual acuity was 20/400, limited mainly by traumatic macular and outer retinal damage. Serial retinal nerve fiber layer optical coherence tomography showed a persistent inferior abnormality without unequivocal progression, while visual field interpretation was limited by retinal damage and reduced vision. In the absence of definite glaucomatous optic neuropathy, the patient was classified as having persistent post-traumatic ocular hypertension associated with extensive angle recession. Long-term IOP and optic nerve monitoring are essential after blunt ocular trauma.

Keywords: angle recession, ocular hypertension, gonioscopy, ocular trauma, paintball injury

INTRODUCTION

Blunt ocular trauma may cause structural damage to the trabecular meshwork, ciliary body, and anterior chamber angle, resulting in acute or delayed elevation of intraocular pressure (IOP). Acute post-traumatic IOP elevation is frequently multifactorial and may be associated with hyphema, inflammatory cells, contusional trabecular dysfunction, and direct trabecular injury. In contrast, angle recession may lead to persistent or delayed aqueous outflow dysfunction and an increased long-term risk of secondary glaucoma [1–3].

Paintball projectiles can produce high-energy blunt ocular trauma and cause combined anterior and posterior segment injuries, including hyphema, traumatic cataract, vitreous hemorrhage, retinal contusion, retinal breaks, and secondary IOP elevation. Although paintball-related traumatic glaucoma has previously been reported, detailed descriptions of extensive angle recession accompanied by early and persistent post-traumatic ocular hypertension remain limited [4,5].

We present a case of paintball-related blunt ocular trauma associated with extensive angle recession, persistent post-traumatic ocular hypertension, progressive traumatic lens opacity, vitreous hemorrhage, and severe traumatic retinal damage. This case also illustrates the diagnostic difficulty of distinguishing glaucomatous structural and functional abnormalities from those caused by coexisting traumatic retinal pathology.

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CASE PRESENTATION

A 36-year-old man presented to the emergency department on the same day that a paintball projectile struck his left eye. Presenting visual acuity was 20/20 in the right eye and hand motions in the left eye. Intraocular pressure was 17 mmHg in the right eye and 45 mmHg in the left eye. Examination of the right eye was unremarkable. Anterior segment examination of the left eye revealed hyphema occupying less than one-tenth of the anterior chamber and a Vossius ring on the anterior lens capsule with an associated membranous appearance (Figure 1A). Fundus visualization was limited because of media opacity, and B-scan ultrasonography demonstrated dense vitreous hemorrhage (Figure 1B).

Initial medical treatment included 300 mL of intravenous 20% mannitol (4 cc/kg. Polifleks; Polifarma, Türkiye). Oral treatment consisted of acetazolamide 250 mg four times daily (Diazomid; Sanofi, Türkiye) and one effervescent tablet providing 40 mmol of potassium once daily (Kalinor; Farma-Tek, Türkiye).

Topical treatment to the left eye consisted of dorzolamide 2%–timolol 0.5% fixed combination, one drop twice daily (Tomec; Abdi İbrahim, Türkiye); brimonidine tartrate 0.15%, one drop twice daily (Alphagan P; AbbVie, Türkiye); cyclopentolate hydrochloride 1%, one drop three times daily (Sikloplejin; Abdi İbrahim, Türkiye); and dexamethasone sodium phosphate 0.1%, initially administered as one drop hourly (Dekort 5 mg/5 mL; DEVA, Türkiye).

At 1 week after trauma, visual acuity remained at the level of hand motions and the IOP had decreased to 18 mmHg. Progressive cortical lens opacification was observed. Topical antiglaucomatous treatment was continued, whereas dexamethasone was gradually tapered. Cyclopentolate, oral acetazolamide, and potassium supplementation were discontinued.

At 1 month after trauma, visual acuity remained at the level of hand motions and the IOP was 17 mmHg. The hyphema had completely resolved, and only occasional cells were present in the anterior chamber. Gonioscopy revealed a Shaffer grade 3 open angle in the right eye. In the left eye, the angle was Shaffer grade 4, with Mooney grade 2 angle recession distributed discontinuously across all four quadrants and involving a cumulative extent of approximately eight clock hours (240°). The affected segments were characterized by posterior displacement of the iris root, deepening of the angle recess, and marked widening of the ciliary body band (Figure 2A–D).

Because of progressive traumatic lens opacity and persistent dense vitreous hemorrhage that continued to prevent adequate fundus visualization, combined surgery was performed 1 month after the injury. The patient underwent phacoemulsification, posterior chamber intraocular lens implantation, and pars plana vitrectomy in the left eye. After clearance of the vitreous hemorrhage, a broad area of hemorrhagic retinal contusion with marked retinal irregularity was observed in the inferotemporal impact quadrant. Because an occult full-thickness retinal break could not be reliably excluded within this severely traumatized area, prophylactic barrier endolaser photocoagulation was applied along the posterior margin of the lesion, followed by fluid–air exchange.

At the early postoperative examination, visual acuity in the left eye was counting fingers at 1 m and the IOP was 32 mmHg. No significant active inflammation was observed on anterior or posterior segment examination. Because the patient could not tolerate oral acetazolamide and did not wish to undergo an additional surgical procedure at that stage, preservative-free bimatoprost 0.01%, one drop once daily, was added after confirming that ocular inflammation was controlled (Bimatocare; SABA, Türkiye).

At the first postoperative month, visual acuity was 20/400 and the IOP was 17 mmHg. Bimatoprost was discontinued, and treatment with dorzolamide 2%–timolol 0.5% fixed combination and brimonidine tartrate 0.15% was continued. Peripapillary retinal nerve fiber layer optical coherence tomography was performed using a spectral-domain OCT device (Cirrus HD-OCT; Carl Zeiss Meditec, Dublin, CA, USA), showed an average RNFL thickness of 103 µm in the left eye, with inferior quadrant thinning outside the normal limits (64 µm) (Figure 4A).

At the third postoperative month, the IOP was 15 mmHg, and all topical antiglaucomatous medications were discontinued. At the subsequent untreated examination 1 month later, the IOP had increased to 26 mmHg. Long-acting carteolol hydrochloride 2%, one drop once daily,

was therefore initiated in the left eye (Carteol LP; Bausch + Lomb, Türkiye).

At the 6-month postoperative examination, while receiving once-daily carteolol hydrochloride 2%, visual acuity in the left eye was 20/400 and the IOP was 17 mmHg. Fundus examination demonstrated central traumatic macular scarring, pigmentary changes, and temporal and inferior endolaser photocoagulation scars. Optical coherence tomography showed irregularity of the foveal architecture and marked disruption and atrophic changes predominantly involving the outer retinal layers, including the ellipsoid zone (Figure 3).

Repeat peripapillary RNFL-OCT at the 6-month postoperative examination showed an average RNFL thickness of 95 µm, with a persistent inferior quadrant abnormality (74 µm) (Figure 4B). Because the inferior RNFL thickness did not decrease and the optic disc parameters remained stable, unequivocal glaucomatous progression could not be demonstrated. The difference in average RNFL thickness between the examinations was interpreted cautiously because formal progression analysis was unavailable, scan centration differed, and extensive traumatic retinal pathology was present.

Humphrey 24-2C SITA Faster automated perimetry, performed using the Humphrey Field Analyzer (Carl Zeiss Meditec, Dublin, CA, USA), demonstrated marked diffuse visual field depression in the left eye at the 6-month postoperative examination with a visual field index of 32%, mean deviation of −22.28 dB, and pattern standard deviation of 11.47 dB. The false-negative rate was 33% (Figure 4D). Interpretation was limited by reduced visual acuity, central macular scarring, extensive traumatic retinal damage, and the high false-negative rate.

No unequivocal glaucomatous cupping or focal neuroretinal rim loss was observed. Although a persistent inferior RNFL abnormality was present, characteristic and reliable glaucomatous structural and functional damage could not be established because of the coexisting traumatic retinal pathology. The recurrence of untreated IOP elevation to 26 mmHg after resolution of the hyphema and active inflammation was therefore considered consistent with persistent post-traumatic ocular hypertension associated with extensive angle recession.

Glaucoma surgery was not considered necessary at this stage because the IOP was controlled at 17 mmHg with a single topical medication and no unequivocal glaucomatous optic nerve damage or progression was demonstrated. In addition, the patient did not wish to undergo another surgical procedure. The patient remains under long-term follow-up with serial IOP measurements, optic nerve examination, RNFL-OCT, and automated perimetry. The total follow-up period was 7 months after the injury and 6 months after surgery.

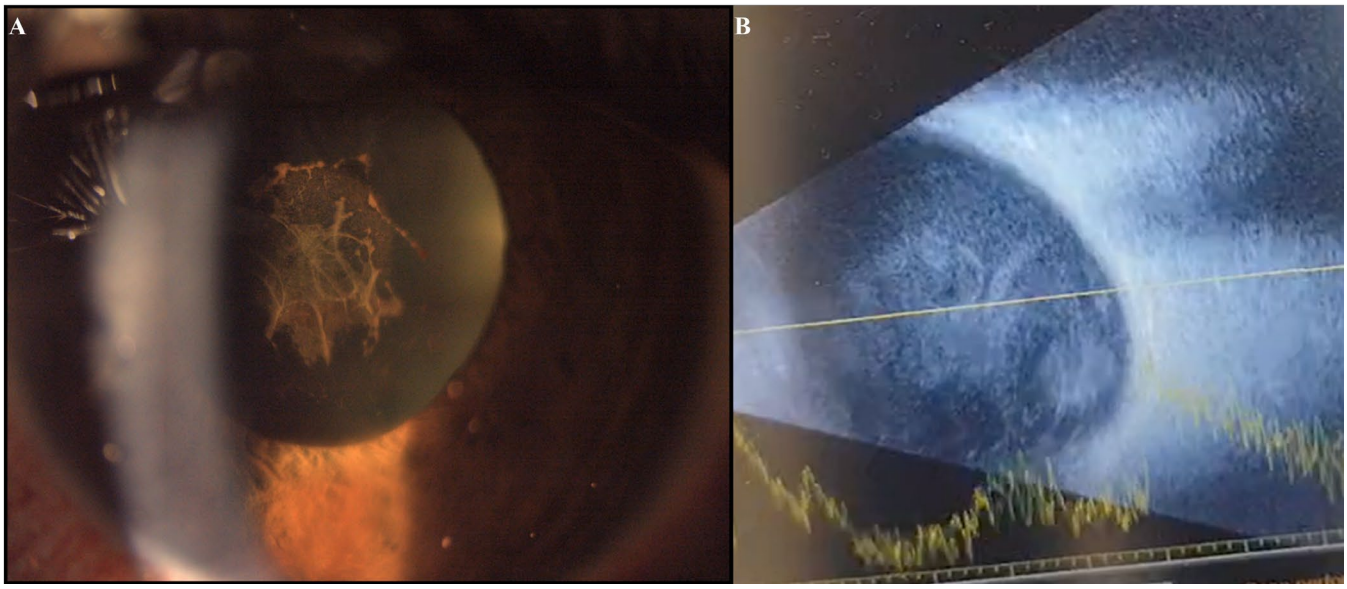


Figure 1. *Initial anterior and posterior segment findings of the left eye. (A) Slit-lamp photograph showing a Vossius ring on the anterior lens capsule with an associated membranous appearance. (B) B-scan ultrasonography demonstrating dense vitreous hemorrhage.*

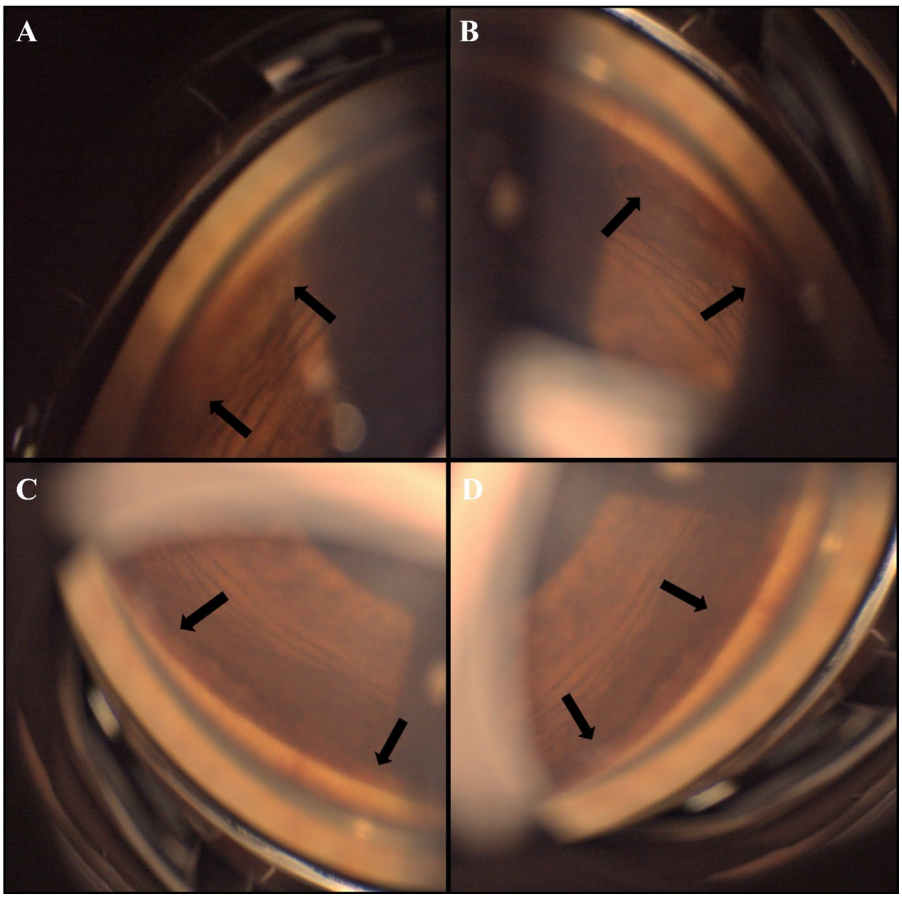


Figure 2. *Gonioscopic images obtained from different quadrants of the left eye. The arrows indicate areas of angle recession characterized by posterior displacement of the iris root, deepening of the angle recess, and marked widening of the ciliary body band. Gonioscopic examination demonstrated Mooney grade 2 angle recession extending over approximately eight clock hours (approximately 240°).*

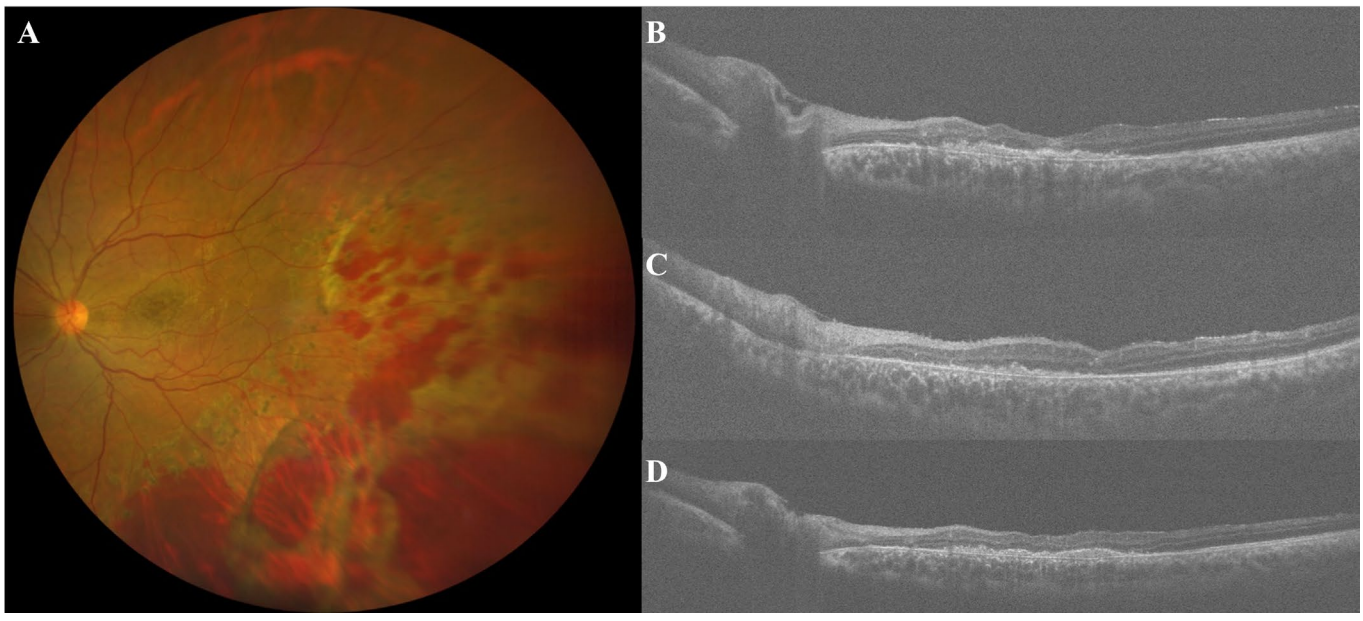


Figure 3. *Postoperative posterior segment findings of the left eye. (A) Color fundus photograph showing central traumatic macular scarring, pigmentary changes, and temporal and inferior endolaser photocoagulation scars. (B–D) Optical coherence tomography B-scans demonstrating irregularity of the foveal architecture and marked disruption and atrophic changes predominantly involving the outer retinal layers, including the ellipsoid zone.*

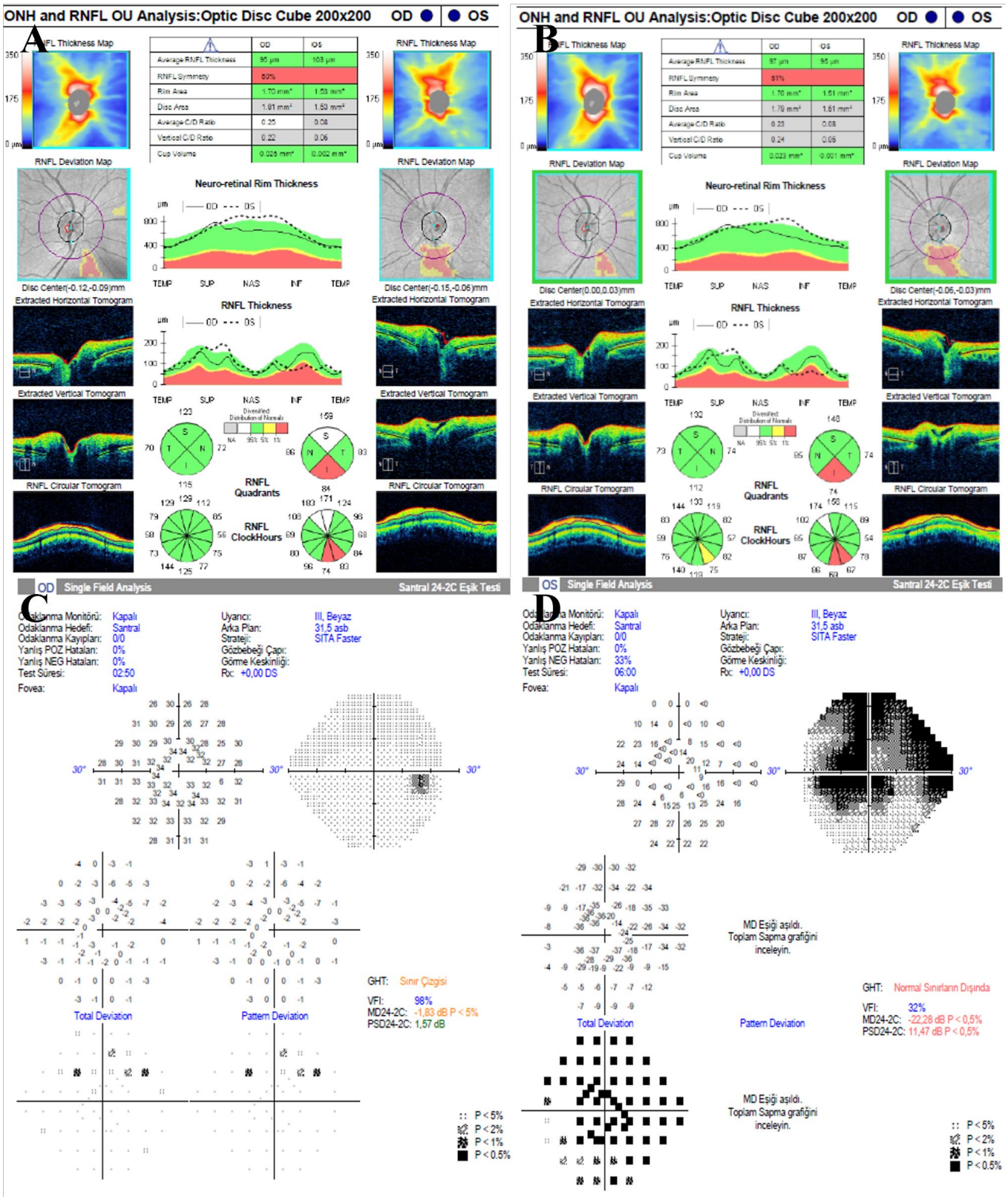


Figure 4. *Structural and functional assessment for glaucomatous damage. (A) Bilateral peripapillary RNFL-OCT performed at the first postoperative month, showing an inferior RNFL abnormality in the left eye. (B) Repeat RNFL-OCT at the sixth postoperative month demonstrating persistent inferior RNFL abnormality with stable cup-to-disc parameters. (C) Humphrey 24-2C SITA Faster automated perimetry of the fellow right eye (D) Humphrey 24-2C SITA Faster automated perimetry of the affected left eye, showing marked diffuse visual field depression. Interpretation of the left-eye visual field was limited by reduced visual acuity, central macular scarring, extensive traumatic retinal damage, and a false-negative rate of 33%.*

DISCUSSION

Ocular trauma is a potentially vision-threatening event, and visual impairment may result from both the primary injury and subsequent complications. Elevated IOP may occur during the acute post-traumatic period or may develop months to years after injury. Analyses from the United States Eye Injury Registry demonstrated that both penetrating and blunt ocular trauma are associated with secondary glaucoma, with a higher reported incidence following ocular contusion [1,2].

Acute post-traumatic IOP elevation may result from multiple mechanisms, including trabecular meshwork obstruction by erythrocytes and inflammatory cells, hyphema, contusional trabecular dysfunction, direct trabecular injury, and trabeculitis or iritis [3]. In the present case, the marked IOP elevation of 45 mmHg at presentation was considered multifactorial and was likely related to extensive angle recession together with trabecular meshwork obstruction by erythrocytes and inflammatory cells, contusional trabecular dysfunction, and direct traumatic damage.

Angle recession is defined as pathological separation between the circular and longitudinal muscle fibers of the ciliary body. Gonioscopy remains the first-line examination for its diagnosis and is recommended in the evaluation of traumatic angle abnormalities [6]. Ultrasound biomicroscopy may provide additional information when gonioscopic visualization is limited or when differentiation from iridodialysis and other traumatic anterior segment abnormalities is required [7,8].

Mooney classified angle recession into three clinical grades according to the gonioscopic depth and appearance of the ciliary body cleft [9]. The findings in our patient were consistent with Mooney grade 2 angle recession, with posterior displacement of the iris root, deepening of the angle recess, and marked widening of the ciliary body band. The recession extended over approximately eight clock hours, corresponding to approximately 240°. Ng et al. demonstrated that angle recession involving more than 180° was significantly associated with an increased long-term risk of glaucoma following traumatic microhyphema or hyphema [10]. The extensive angle involvement in our patient therefore indicates a substantial long-term risk of angle-recession glaucoma.

Nevertheless, elevated IOP and angle recession alone are insufficient to establish a diagnosis of glaucoma in the absence of characteristic glaucomatous optic neuropathy. In our patient, untreated IOP increased to 26 mmHg after resolution of the hyphema and active inflammation, supporting persistent traumatic aqueous outflow dysfunction associated with extensive angle recession. However, no unequivocal glaucomatous cupping or focal neuroretinal rim loss was observed.

Serial RNFL-OCT examinations demonstrated a persistent inferior RNFL abnormality. However, stable cup-to-disc parameters, variability between the two scans, the absence of formal progression analysis, and extensive coexisting retinal pathology precluded confirmation of progressive glaucomatous structural damage. Automated perimetry showed marked diffuse depression in the affected eye, but its interpretation was limited by reduced visual acuity, central macular scarring, widespread traumatic retinal damage, and the high false-negative rate. The patient was therefore classified as having persistent post-traumatic ocular hypertension associated with extensive angle recession rather than established angle-recession glaucoma.

Combined phacoemulsification and pars plana vitrectomy were performed 1 month after trauma because of progressive traumatic lens opacity and persistent dense vitreous hemorrhage. Vitrectomy may be considered in selected cases of traumatic vitreous hemorrhage that fail to improve after an initial observation period [11]. Intraoperatively, marked retinal irregularity was identified within a broad hemorrhagic contusion in the inferotemporal impact quadrant. Because an occult retinal break could not be confidently excluded, prophylactic barrier endolaser photocoagulation was applied as a case-specific precaution. Traumatic retinal breaks following ocular contusion are associated with a substantial risk of rhegmatogenous retinal detachment [12]. The limited visual recovery was attributed primarily to traumatic macular and outer retinal damage, consistent with evidence indicating that higher grades of macular commotio are associated with poorer visual outcomes after blunt ocular trauma [13].

Medical treatment is generally the first-line approach for post-traumatic ocular hypertension. Medication selection should take the degree of ocular inflammation into consideration. In the present case, preservative-

free bimatoprost 0.01% was initiated only after anterior and posterior segment examinations demonstrated no significant active inflammation. It was selected because the postoperative IOP remained elevated at 32 mmHg, oral acetazolamide was not tolerated, and the patient did not wish to undergo an additional surgical procedure at that stage. Bimatoprost was discontinued after the IOP decreased to 17 mmHg.

After all topical medications were discontinued at the third postoperative month, untreated IOP subsequently increased to 26 mmHg, further supporting persistent traumatic outflow dysfunction. The IOP was subsequently controlled at 17 mmHg with once-daily carteolol hydrochloride 2%. Glaucoma surgery was not considered necessary because satisfactory IOP control was achieved with monotherapy and unequivocal glaucomatous damage or progression was not demonstrated. The patient also did not wish to undergo another surgical procedure.

Several surgical procedures, including trabeculectomy with mitomycin-C, penetrating canaloplasty, and glaucoma drainage device implantation, have been used in medically uncontrolled angle-recession glaucoma [14–16]. However, there is no established consensus regarding the optimal surgical technique, and treatment should be individualized according to the degree of IOP elevation, the presence and severity of glaucomatous damage, previous ocular surgery, and patient-related factors.

Lee et al. reported four patients who developed glaucoma following paintball-related ocular injuries [4]. Two patients had elevated IOP at presentation, whereas the remaining two developed glaucoma during follow-up. Two underwent Baerveldt glaucoma drainage device implantation, one underwent cyclophotocoagulation, and one was controlled medically. Gonioscopic findings and the precise mechanisms of glaucoma were not reported. Similar to the present case, both the anterior and posterior segments were affected, and visual impairment was largely associated with posterior segment injury.

In a Turkish series of paintball-related ocular trauma, Keleş et al. reported secondary glaucoma in one of 12 patients, which was treated with trabeculectomy; however, the extent and characteristics of angle recession were not described [5].

This case demonstrates that marked and persistent ocular hypertension may occur early after high-velocity blunt ocular trauma in the presence of extensive angle recession. Acute IOP elevation may be multifactorial, whereas recurrent elevation after resolution of hyphema and inflammation may reflect persistent traumatic aqueous outflow dysfunction. In patients with concomitant retinal damage, RNFL and visual field abnormalities should be interpreted cautiously before a diagnosis of glaucoma is established. Careful gonioscopic examination and long-term monitoring of IOP, optic nerve appearance, RNFL thickness, and visual field findings are essential for the timely detection of angle-recession glaucoma.

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