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Angle Recession in Traumatic Hyphema: A Case Report

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Abstract

Introduction: *Ocular trauma is the one of the primary causes of unilateral vision impairment. It can damage the trabecular meshwork, resulting in post-traumatic glaucoma.* ***Case Report:*** *A 26-years old male complained pain and blurred vision in right eye in the last 4 days prior. Patient was hit by shuttlecock 6 days ago. Right eye visual acuity was hand movement. The intraocular pressure was 50,6 mmHg. There was minimal coagulum in one-third of anterior chamber volume. The anterior chamber was deep with middilated pupil. Patient was given loading CAI and hyperosmotic agent, topical β blocsa Bker, oral and topical steroid, and anti-fibrinolytic agent. First day of treatment, IOP was decreased to 11,2 mmHg. Third day of treatment, funduscopy showed hyperemic optic disc and gonioscopy showed widening of ciliary body band almost 180 degrees. Then, on the fifth day the visual acuity became 5/5 with 20 mmHg IOP.* ***Discussion:*** *Hyphema caused by ocular blunt trauma is typically the consequence of a tear in the iris or ciliary body, which damages the major arterial circle and its branches. Angle recession in this case was treated conservatively without drugs and the IOP remained stable. On gonioscopy, angle recession is observed as an expansion of the ciliary body band. It has been shown that angle recession glaucoma can develop within first few weeks to years after trauma.* ***Conclusion:*** *Ocular blunt trauma can induce elevated IOP due to inflammation, tear in iris or ciliary body and hyphema. Early diagnosis and prompt treatment can prevent irreversible optic nerve damage.*

Keywords: *Ocular blunt trauma, traumatic hyphema, angle recess*

INTRODUCTION

Ocular blunt trauma or ocular contusions, is frequently seen in the emergency room. Ocular injury is considered as important cause of unilateral visual impairment and visual loss. Approximately 1.6 million cases of blindness and 2.3 million cases of vision impairment globally are related to eye injuries. According to a study by, trauma frequently results in the majority of unilateral visual loss in developing countries (accounting for up to 5% of all blindness). Injury to or blockage of the trabecular meshwork can result in the development of post-traumatic glaucoma[1,2,3.]

The elevated IOP within 2 weeks to 1 months after injury can be caused by inflammation, hyphema and lens dislocation. One of the main effects of trauma is inflammation, which can lead to elevated intraocular pressure because it might obstruct the anterior chamber's outflow of inflammatory cells, debris, or proteins. It can also potentially cause inflammation in the trabecular meshwork. The clinical presentation experienced by each patient will be different according to the time of examination and diagnosis[4, 5, 6]

The incidence of posttraumatic glaucoma developing within six months can be as high as 3.4% and 2.7% in the case of blunt and penetrating ocular trauma, respectfully. Traumatic glaucoma has been linked to anterior segment hyphema as well as damage to the iris or angle. The most frequent injuries resulting from blunt ocular trauma are angle recession, iridodialysis, and cyclodialysis. A radial tear in the ciliary muscle that

divides the circular and longitudinal fibers is what is known as angle recession. It appears as an enlargement of the ciliary body band on gonioscopy.[5,14,15]

Trauma causes the lens-iris junction to shift backward, resulting in the widening of the equatorial sclera. The ciliary body's blood vessels, the recurrent choroidal arteries and veins, and the primary arterial circle of the iris are all under tension as a result of this growth. Vascular damage in the anterior ciliary body, iris sphincter, or peripheral iris causes hyphema[6,16,17,18].

Angle recession glaucoma has been demonstrated in research and literature to occur as quickly as a year or even decades following ocular trauma. The specific time period of development of angle recession glaucoma still cannot be defined. According to Mohammed's study, between one and five years of age, 56% of patients will develop angle recession glaucoma. Thus, the longlife follow up of patient with history of ocular blunt trauma is needed[2,19,20.]

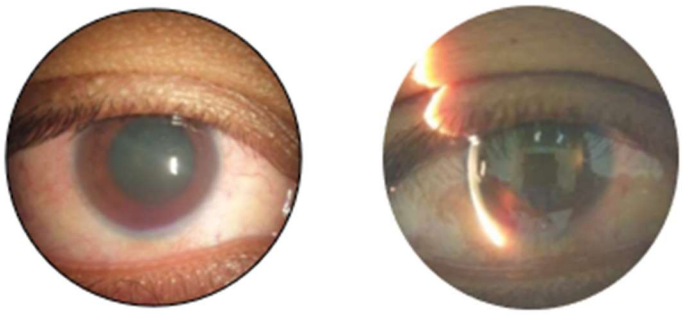
Angle recession, hyphema, low initial visual acuity, age, lens damage, high baseline IOP, and angle recession greater than 180 degrees are some of the predictive indicators that can be used to identify patients who may be at higher risk of developing angle recession glaucoma. The management of posttraumatic glaucoma is essential, as complications from elevated intraocular pressure can often be avoided with appropriate care. Damage to the optic nerve cannot be reversed by treating angle recession glaucoma and diagnosing it early[21, 22]. In this case study, a 26-year-old man who had physical injuries to his right eye is described as having angle recession glaucoma.

CASE ILUSTRATION

A 26-year-old man came with a history of blunt trauma to his right eye six days prior from being injured by a shuttlecock. Post injury, he complained blurred vision accompanied by redness and photophobia in the right eye. 2 days after injury, he complained painful in right eye and blurred vision was getting worse. He was non-hypertensive and non-diabetic patient back then. He has no history of blood abnormality.

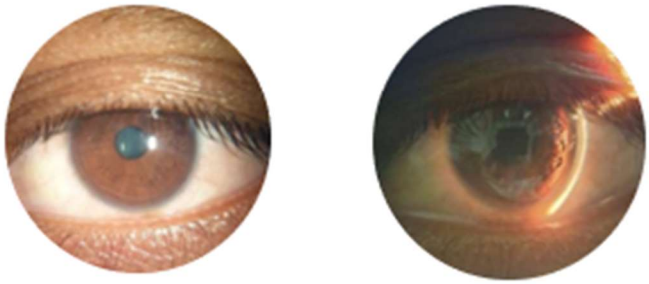
The general examination revealed blood pressure and pulse was within normal limit. The only outward injury was to the right eye. A normal systematic examination occurred. The results of an ocular examination revealed that the right eye's visual acuity was hand movement and the left eye's was 6/6. The intraocular pressure was 50,6 mmHg (RE) and 14,7 mmHg (LE). From anterior segment of RE showed conjunctiva hyperemia with corneal edema and deep anterior chamber depth to grade III-IV Van Herick. Minimal coagulum was noted in one third of anterior chamber volume. The right pupil was mid-dilated and measured 6 mm. The lens was crystal-clear. Because of ocular edema, the fundus could not be seen. The left eye's fundus and anterior portion were also within normal limits.

The patient had treatment at the prior hospital for six days, during which time they were given injections of Ceftriaxon (1 g every 12 hours), Ketorolac (30 mg every 12 hours), Tranexamic acid (500 mg every eight hours), MP (8 mg every eight hours), and Acetazolamide (250 mg every eight hours), Xitrol eyedrop 6 times a day RE, Fluorometholon eyedrop 6 times a day RE, Betaxolol 0,5% eyedrop every 12 hours RE and tropin eyedrop every 12 hours RE.



(A) (B)

Figure 1. (A) Initial presentation of right eye showed minimal coagulum with middilated pupil and (B) depth anterior chamber depth grade III-IV Von Herrick



(A) (B)

Figure 2. (A) Left eye showed normal anterior segment and (B)depth anterior chamber depth grade III-IV Von Herrick

Patient was administered loading 500 mg Acetazolamide but the IOP remained high. Then patient was given oral Glycerin 1 cc/kgW which lowered the IOP to 11,2 mmHg after 2 hours instillation. The initial diagnose of the patient was angle recession glaucoma of the right eye (RE) with differential diagnosis vitreous haemorrhage of RE. Patient then given acetazolamide 250 mg every 6 hours, KSR 600 mg once a day, methylprednisolon 4 mg every 8 hours, tranexamic acid 500 mg every 12 hours along with topical Timolol 0,5% every 12 hours and topical steroid every 6 hours. Patient suggested to bed rest and head up 30 degrees.

The next day, the vision in the right eye was recorded at 6/20, IOP 19.6 mmHg and the presence of corneal edema. We performed a B-scan ultrasonography on right eye and showed echogenic lesion particle shaped with 30-50% echospike rcs complex, low mobility indicating vitreous haemorrhage or inflammation.

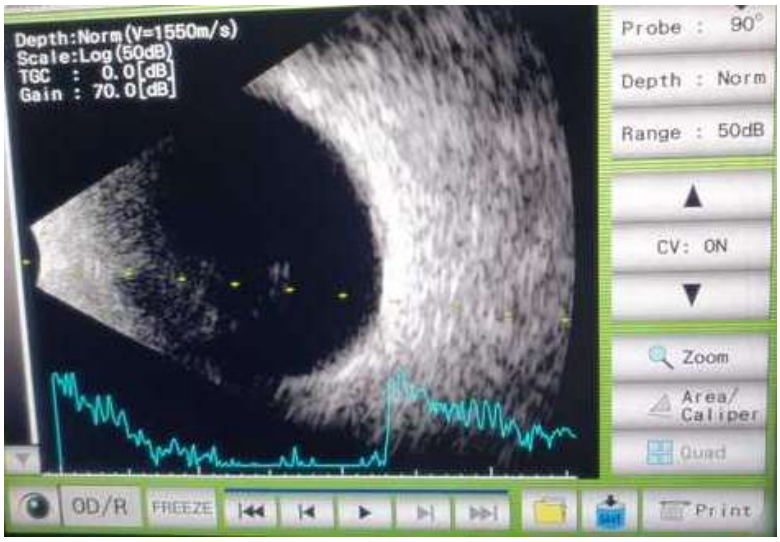
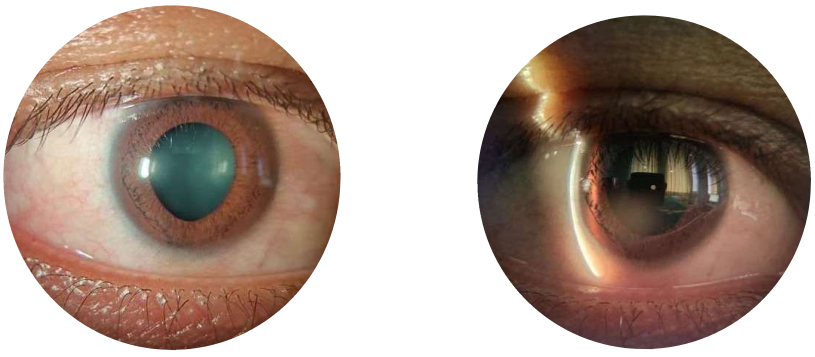


Figure 3. B-scan USG indicating inflammation process or vitreous haemorrhage RE

Visual acuity of 5/5 on both eyes with IOP both eyes was 13,4 mmHg after 5 days hospitalized. Traumatic mydriasis was observed. The inspection of the fundus was normal. A ciliary body widening of less than 180 degrees was discovered during a gonioscopy on the right eye. In the left eye, a gonioscopy revealed grade 4 open angles. Retinal nerve fiber layer (RNFL) optical coherence tomography (OCT) results were found normal. The patient's condition was identified as angle recession glaucoma RE. Patient was administered to continue the therapy with additional therapy steroid topical every 4 hours RE.

After 5 days of hospitalized, patient then discharged and was given acetazolamide 250 mg every 8 hours, KSR 600 mg once a day, methylprednisolon 4 mg every 8 hours, timolol 0,5% every 12 hours RE, topical steroid every 4 hours RE. He recommended that he recover as much as possible and was advised to not engage in any strenuous activities and to sleep at an angle of approximately 30°. He was provided with a non-pressure eye patch to help ease his photophobia when outside or in bright light.

The last examination of the patient after 2 months injury revealed that visual acuity of RE was 5/6.5 with pinhole 5/5 and 5/5 LE. The IOP of RE was 20 mmHg and 15 mmHg of LE. From anterior segment of RE showed middilated pupil 6 mm with irradier iris.



(A) *(B)*

Figure 4. Two months after injury (A) Right eye showed irradier iris with middilated pupil and (B)depth anterior chamber depth grade III-IV Von Herrick

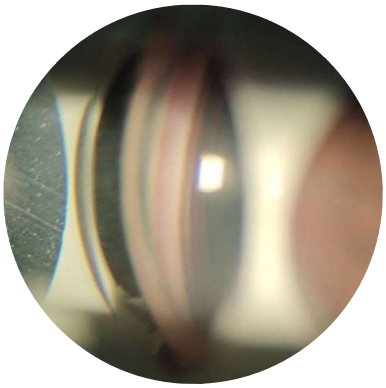


Figure 5. Gonioscopy of the right eye showed widening of ciliary body

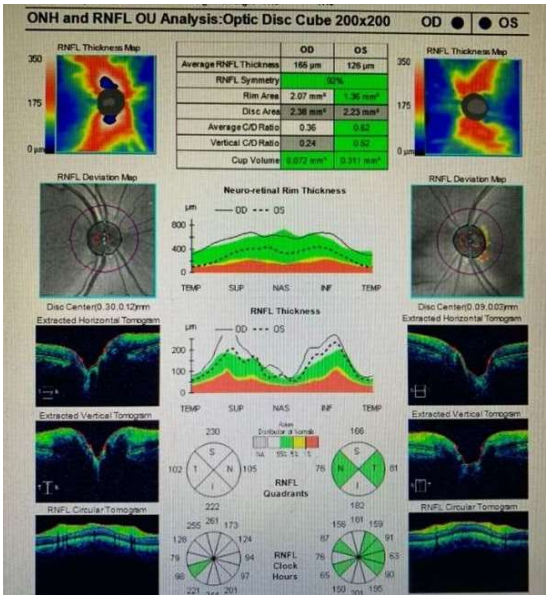


Figure 6. Optic Nerve Head Analysis

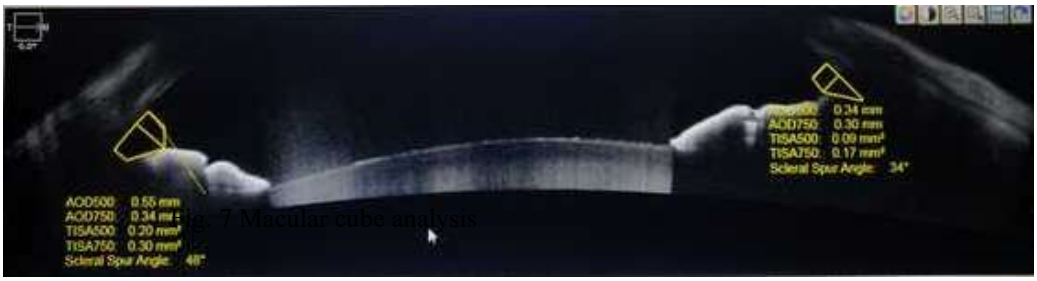


Fig. 7 Macular cube analysis

Figure 7. Anterior OCT of the right eye

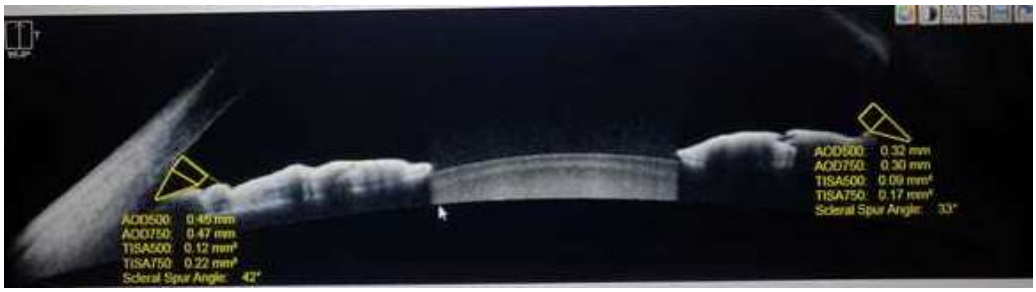


Figure 8. Anterior OCT of the left eye

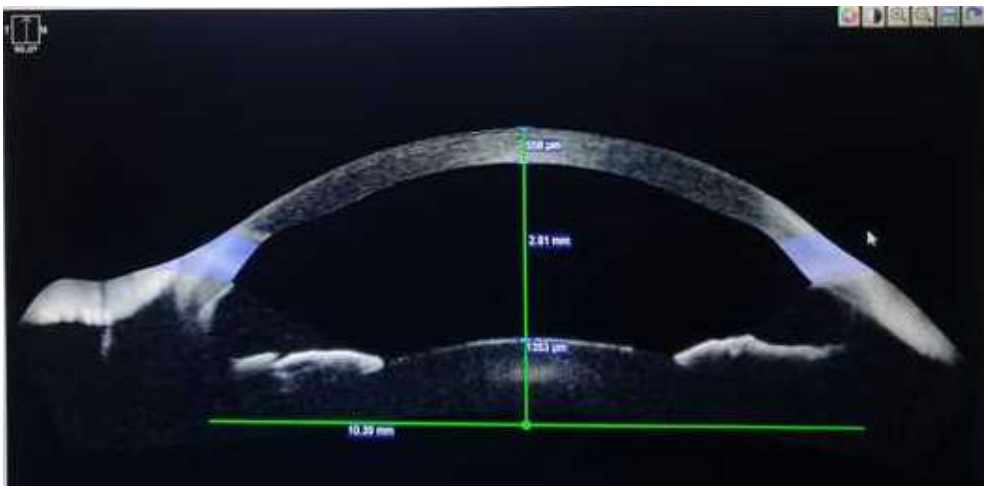


Figure 9. Anterior OCT of the right eye showed deep anterior chamber

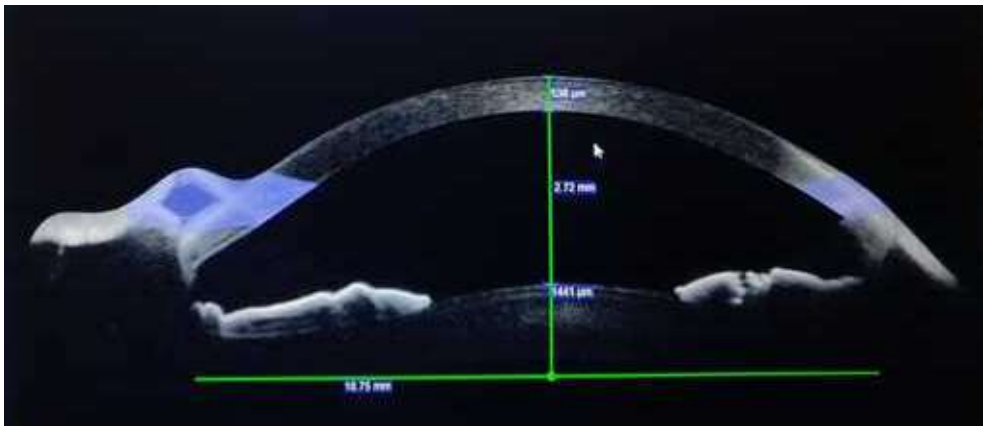
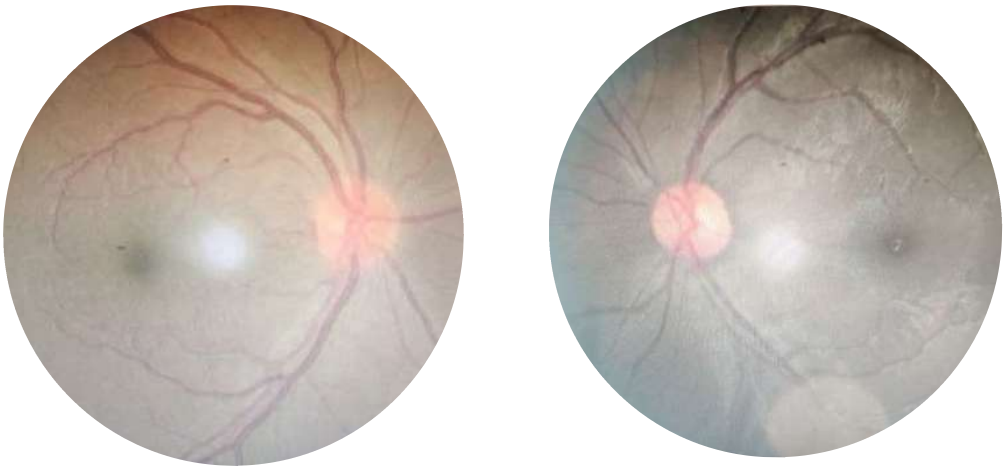


Figure 10. Anterior OCT of the left eye showed deep anterior chamber



(A) *(B)*

Figure 11. (A) Fundus Picture of right eye showed normal fundus with cup disk ratio 0.3 and (B) normal fundus of the left eye

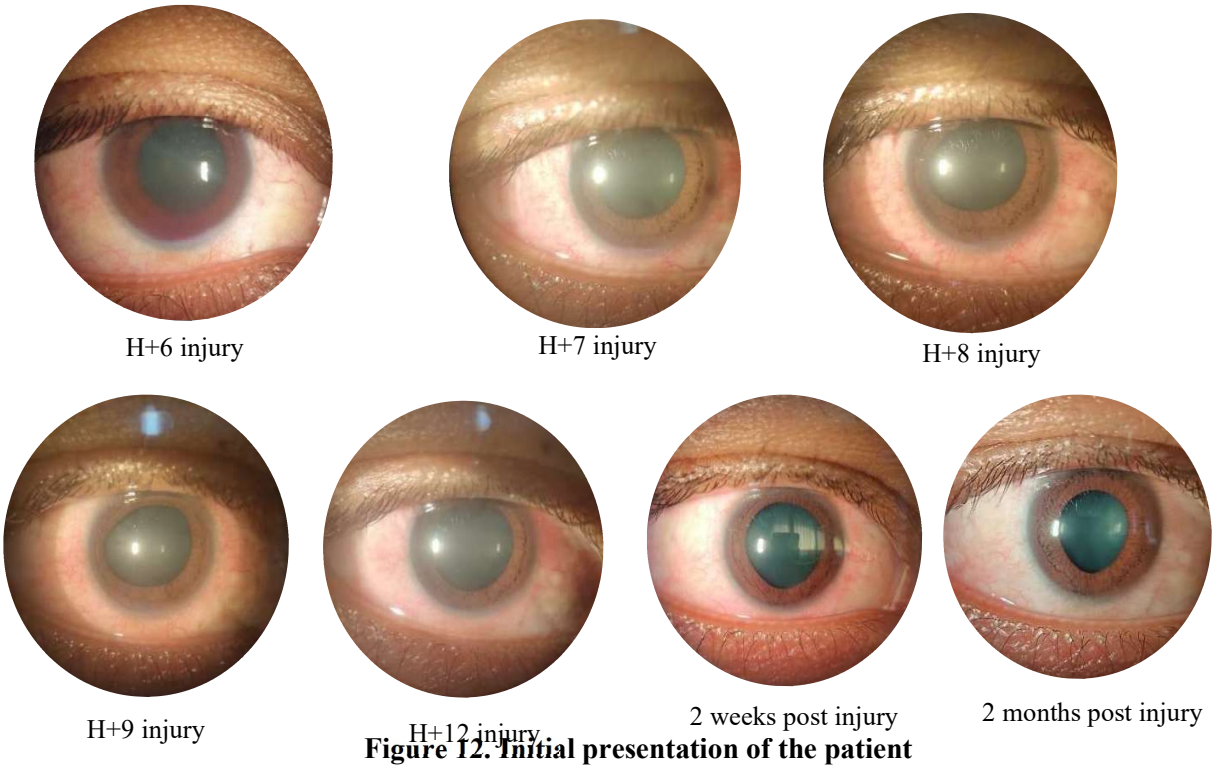


Figure 12. Initial presentation of the patient

DISCUSSION

Two significant outcomes result from the impact of a blunt object. First, force vectors extend from the area of contact between a blunt item and the eye, resulting in a direct transfer of energy. Second, ocular shape changes due to elongation along the equatorial plane resulting from globe compression in an anterior-

posterior orientation. The consequent predictable injury as described by Campbell were include sphincter pupil, iris base, anterior ciliary body, attachment of the ciliary body muscle fibers to the scleral spur, trabecular meshwork, zonules, and retina to the ora serrata[3,23,24.]

Bai et al.'s study revealed that the interval between ocular trauma and the onset of elevated intraocular pressure (IOP) varied from 2 to 60 months, with an average duration of 23 months. Other side, the elevated IOP within 2 weeks to 1 months after injury can be caused by inflammation, hyphema and lens dislocation. Inflammation is a significant consequence of trauma and can result in elevated intraocular pressure from outflow obstruction caused by inflammatory cells, detritus, or protein in the anterior chamber, as well as potentially due to inflammation at the trabecular meshwork.[3,4,25]

The first few hours to days after trauma can result in an intermediate phase of glaucoma that lasts one to several months. This phase can be caused by a steroid response, ghost cells from vitreous hemorrhage, angle closure from synechiae formation due to severe inflammation, and phacoanaphylaxis from a ruptured capsule. Glaucoma is often associated with red blood cells, inflammation, and pupil obstruction due to anterior lens dislocation[7,26,27]. Glaucoma may develop later (typically after six months) as a result of angle recession and siderosis. Hemolytic glaucoma, ghost cell glaucoma or both may be resulted from traumatic vitreous hemorrhage and history or preexisting retinal disease. The RBCs that have lost their intracellular hemoglobin obstruct the trabecular meshwork and cause elevated IOP[8,28,29.]

Following ocular blunt trauma, there is usually a brief rise in intraocular pressure (IOP) that resolves on its own; in most cases, medication is the only treatment required. Years after trauma, the late rising intraocular pressure (IOP) is more challenging to cure medically and may need surgery[9,30,31]. The exact mechanism remains unknown. It is believed to result from a chronic, progressive failure of outflow in trabecular meshwork, resulting in a gradual increase in intraocular pressure. Blunt trauma causes the aqueous humor to be pushed laterally and posteriorly against the iris. This hydrodynamic force can cause a tear between the ciliary body's circular and longitudinal muscles, pushing tension on the iris root. A hyphema can result from breaking the ciliary arteries with enough force. An early IOP spike could result from damage to Schlemm's canal and the trabecular meshwork caused by this initial assault. Aqueous outflow may decrease when Schlemm's canal and the hyaline membrane narrow as a result of reduced ciliary muscle tension on the scleral spur [10,32,33].

In this case, the patient got elevated IOP in 2 days after initial injury. Patient then given acetazolamide 250 mg every 8 hours, betaxolol eye drop every 12 hours RE and atropine eye drop every 24 hours RE but the IOP still remained high for 4 days. Patient then referred and was given therapy hyperosmotic agents glycerin 1cc/kgW and IOP reduced to 11,2 mmHg after 2 hours instillation.

After an ocular blunt trauma, glaucoma may result from inflammatory scarring, direct inflammation, inflammatory debris, lens fragments, coagulated blood components, dead red blood cells from prolonged vitreous hemorrhage, or red blood cells from hyphema. These factors can disrupt the trabecular meshwork. Pupilary block may result from anterior displacement of the lens caused by lens displacement or damage[1,34.]

Traumatic secondary glaucoma can arise from a single cause, but it is generally the result of multiple contributing factors. Variations in the mechanism of traumatic secondary glaucoma are dependent upon the severity and expanse of tissue damage[4,35.]

Hyphema is a manifestation that is frequently observed following ocular blunt trauma. Most of the time, with conservative care, the blood clears up on its own in a few days, but clot lysis and retraction can cause bleeding to occur again in the initial weeks following trauma. The extent of the hemorrhage after traumatic hyphema affects the likelihood of developing glaucoma. In a research, glaucoma developed in 52% of eyes with a complete hyphema and 13,5% of eyes with a hyphema involving less than half of the anterior chamber [5,3.]

Traumatic hyphema is commonly resulted from injury of the major arterial circle and branches of the iris. Blunt trauma results in simultaneous equatorial expansion and antero-posterior compression of the globe. This growth may result in a tear of the iris stromal vessels or ciliary body. Increased pigmentation in the angle during gonioscopy, A higher baseline intraocular pressure and the absence of a cyclodialysis cleft can both aid in identifying ocular blunt trauma linked to chronic glaucoma particularly in those who have a history of hyphema, angle recession, and lens injury[11,12,36.]

According to reports, angle recession is seen in more than 60% of eyes with non-penetrating traumatic injuries. Between 56% and 100% of patients with traumatic hyphema exhibit variable degrees of angle recession, according to careful gonioscopy. According to Sharma and Rao, 20–94% of blunt trauma cases result in angle recession as a sequela. Angle recession was apparent in 71–100% of cases of blunt trauma with hyphema identified. Glaucoma is expected to develop in about 1-2% of eyes with angle recession over time[9,10,37].

Following physical trauma, traumatic or angle recession glaucoma is frequently observed. The ciliary muscle experiences a radial tear during angle recession, separating the longitudinal and circular fibers. It manifests as a ciliary body band widening on gonioscopy.

Traumatic glaucoma is generally chronic and usually occurs in one eye. It can arise right after the eye injury or manifest months to years afterward[8,5,38].

In a retrospective analysis of ocular contusions that extend a 12-year period from 1967 to 1979, angle recession was shown to be the most common finding (80.5%) following anterior segment blunt trauma, with iris trauma (37.3%) and lens injury (24.5%) following immediately behind. The most common clinical sequelae, according to another study conducted by Ng et al., were angle recession (81,4%) and early glaucoma (37,1%). Within six months of their presentation, all patients (97,2%) who received IOP lowering medication eventually stopped receiving it and their IOP decreased to < 21 mmHg. Surgical techniques such as anterior chamber paracentesis, washout, or trabeculectomy were not utilized. The average duration from the injury to the end of treatment was 38.1 ± 39.2 days, ranging from 4 to 122 days[1,7].

According to Sihota *et al* study about 1 year evaluation of closed globe injury revealed that A higher incidence of traumatic glaucoma was associated with a greater amount of angle recession. About 93% of the eyes in the group with traumatic glaucoma had angle recession glaucoma, compared to 54% of the eyes in the group without glaucoma.[12.]

The iridocorneal angle often decreases following physical trauma, nevertheless glaucoma will only develop in 6% to 7% of these eyes over time. After angle recession, there are two significant peaks in the incidence of glaucoma: the first peak appears weeks to years after the damage, and the second peak appears ten years or more later[9,39].

In a 2018 study by Maity, anterior chamber angle recession was identified as the most frequent complication, occurring in 80.5% of subjects. Additionally, the incidence of angle recession after blunt trauma in patients with hyphema was reported to be 71.4% within the first month[13,40.]

It seems that eyes with an angle recession of less than 180 degrees are less likely to acquire glaucoma, but eyes with an angle recession of 180 to 360 degrees are more likely to develop late-onset glaucoma. Blanton found that every patient who developed angle recession glaucoma ten years after trauma had more than 180 degrees of recession. Furthermore, the non-traumatized fellow eye in these patients has a reported 50% likelihood of developing open-angle glaucoma. Identifying the eyes that are susceptible to chronic traumatic glaucoma is crucial in order to ensure that the right therapy is executed [9,12].

Patients who may develop angle recession glaucoma may be identified with the use of a number of predictor characteristics, such as poor initial visual acuity, advanced age, lens damage, angle recession, hyphema,

elevated baseline IOP, and angle recession more than 180 degrees. A study conducted by Ng et al. found that 75.8% of patients developed angle recession with microhyphema, similar to Blanton *et al*.'s earlier report of 71% among 182 patients with gross traumatic hyphema[2,7.]

In this case, we found that there was microhyphema and minimal coagulum in the anterior segment of the right eye in day 6 after initial injury. It means that after the injury, the patient got hyphema and had been absorbed. Two days after injury, patient got elevated IOP and thus we indicated that the patient got traumatic hyphema. From the gonioscopy revealed widening of ciliary body right eye less than 180 degrees.

The management of hyphema, regardless of the cause, aims to prevent secondary hemorrhage, minimize further trauma to the eye, facilitate the settling of blood in the anterior chamber, and manage traumatic uveitis. Management involves using plastic or metal shields for eye protection, limiting physical activity, elevating the head, and avoiding aspirin and other non-steroidal anti-inflammatory medications. If the hyphema fails to show improvement after 5 days, surgical intervention may be suggested to remove the blood and decrease the elevated intraocular pressure[4, 11,41.]

Beta blockers and carbonic anhydrase inhibitors (CAIs) are typically the first-line treatments for patients whose intraocular pressure (IOP) is more than 25 mmHg. However, topical CAIs should be used with caution in individuals with sickle cell hemoglobinopathies, as these drugs can decrease aqueous pH and lead to increased sickling of blood cells. Eyes with hyphema need thorough examination and follow-up. If the hyphema does not improve after 5 days, surgical intervention is necessary to remove the blood and decrease the elevated intraocular pressure (IOP). Anterior chamber irrigation is usually the initial approach. If this method fails to lower the high IOP, trabeculectomy, either with or without mitomycin C, may be considered[4, 42.]

In this case, the patient came to our hospital in 6 days after initial injury. The anterior segment of the RE was found mycrohyphema with minimal coagulum. It showed that the hyphema has been absorbed and thus didn't need any surgical intervention. Patient was given therapy with acetazolamide 250 mg every 6 hours, timol eye drop every 12 hours RE and methylprednisolone 4 mg every 8 hours, tranexamic acid 500 mg every 8 hours and showed reduced IOP and better visual acuity.

Only 7 eyes (18%) with traumatic glaucoma and 18 eyes (35%), without glaucoma, had cyclodialysis found by Sihota et al. A cyclodialysis cleft results from the disinsertion of the longitudinal fibers of the ciliary muscle from the scleral spur and the underlying sclera. While cyclodialysis is generally associated with low intraocular pressure (IOP) during the initial examination, the IOP can later increase spontaneously as the cleft closes. A shallow anterior chamber, hypotony, opaque media, or atypical anterior segment anatomy can make it challenging to identify a cyclodialysis cleft in recently injured eyes[12.]

Traumatic glaucoma is treated medically in accordance with the severity of the initial injury and variations in the course of the condition. Effective topical aqueous suppressants include beta-blockers, carbonic anhydrase inhibitors, and alpha agonists. Prostaglandin analogs, due to their pro-inflammatory nature, should be avoided during the acute phase but can be employed afterward, as they may facilitate uveoscleral outflow by circumventing the dysfunctional trabecular meshwork. Pilocarpine should be avoided as it can worsen angle recessions. If lens dislocation leads to a pupillary block, cycloplegics may be helpful until surgery is conducted[10.]

It is important to inform the patient about increasing risk of glaucoma in the future, particularly for patients with angle recession greater than 180°.

CONCLUSION

Mechanism of traumatic glaucoma are complex. It often results from a combination of several factors. The mechanisms for different types of traumatic secondary glaucoma differ based on the severity and extent of

tissue damage. Recognizing the risk factors for traumatic glaucoma is vital to prevent ocular morbidity with prompt and suitable management.

Patients with a history of traumatic hyphema should be advised to have follow-up visits and undergo gonioscopic evaluation of the angle to enable early diagnosis. Irreversible optic nerve damage can be avoided by treating angle recession glaucoma early on.

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