Angle recession glaucoma: A case report and review of the literature

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ABSTRACT

Ocular trauma is an important cause of visual loss and has varied manifestations. Here, we present the case of a 34-year-old male who presented with a history of trauma in the right eye (RE) 2 months when he was hit with a tennis ball while playing cricket. He had a visual acuity of counting fingers close to face in the RE. The slit-lamp evaluation revealed a hyphema of more than half of the anterior chamber volume. The left eye (LE) was normal. Intraocular pressure (IOP) was 48 mmHg (RE) and 17 mmHg (LE). The patient was started on anti-glaucoma medications (tablet Diamox, eye drop Timolol, and Brimonidine) and IOP reduced to 21 mmHg in the RE. After 1 month, the gonioscopic evaluation revealed an angle recession in 4 clock hours. Recorded IOP was 18 mmHg in the RE and 16 mm Hg in the LE. The optical coherence tomography retinal nerve fiber layer showed early glaucomatous changes. Based on these findings, the patient has been diagnosed as a case of angle recession glaucoma RE and kept on follow-up.

Key words: *Angle recession, Glaucoma, Gonioscopy, Ocular trauma*

Trauma is a preventable cause of ocular morbidity occurring commonly in young adults [1]. Many cases of traumatic glaucoma post-closed globe injury are not diagnosed and not followed up. They are diagnosed after irreversible glaucomatous optic nerve damage [2,3].

The rationale of reporting this case is to identify the features of angle recession, the risk factors for progression to glaucoma, and optimum management of the case. Early diagnosis and treatment of angle recession glaucoma can prevent irreversible optic nerve damage. Here, we present the case of angle recession glaucoma in a 34-year-male patient developed post blunt trauma in his right eye (RE).

CASE REPORT

A 34-year-old male patient presented with a history of trauma in the RE 2 months back when he was hit with a tennis ball while playing cricket. Post-injury, he complained of a painful diminution of vision with redness and photophobia in the RE. He was non-hypertensive and non-diabetic.

The general examination revealed pulse – 72/min regular and blood pressure – 122/78 mm Hg, and the patient was afebrile. There were no external injuries elsewhere except in the RE. The systemic examination was normal. Ocular examination revealed ecchymosis in the RE with visual acuity of counting fingers close to face. The left eye (LE) had visual acuity of 6/6 unaided. The slit-lamp (SL) evaluation revealed normal conjunctiva and clear cornea. Hyphema was noted which was more than half of the anterior chamber (AC) volume and there was traumatic mydriasis. The lens was clear and the fundus could not be visualized. The anterior segment and fundus examination were normal in the LE. The intraocular pressure (IOP) was 48 mmHg (RE) and 17 mmHg (LE).

The patient was administered IV mannitol 200 ml over 20 mins which lowered the IOP to 35 mm Hg. He was put on tablet Diamox 500 mg BD along with topical Timolol 0.5% BD and Brimonidine TDS. IOP got reduced to 23 mmHg in the RE. The hyphema had reduced in volume (<½ of AC volume). Tablet Diamox was stopped after 3 days and he was kept on topical Timolol and Brimonidine. He was called for a review after 1 month.

Ocular examination revealed visual acuity of 6/9 unaided (RE) and 6/6 unaided (LE). The AC revealed no hyphema. Traumatic mydriasis was present. The fundus examination was normal. Gonioscopy done in both eyes revealed grade 4 open angles with angle recession about 4 clock hours in RE (Fig. 1) with iris processes and grade 4 open angles in the LE with no evidence of angle recession. Perimetry showed early glaucomatous changes with MD – 4.8 RE with Glaucoma Hemifield Test (GHT) outside normal limits. LE fields were normal. Optical coherence tomography (OCT) retinal nerve fiber layer (RNFL) showed early glaucomatous changes. He was diagnosed as a case of angle recession glaucoma RE. The last IOP reading (6 months post-injury) was 18 mm Hg in the RE and 16 mm Hg in the LE. The patient kept on follow-up with Timolol and Brimonidine eye drops.

DISCUSSION

Traumatic glaucoma is a heterogeneous group of post-traumatic ocular disorders with various underlying mechanisms leading

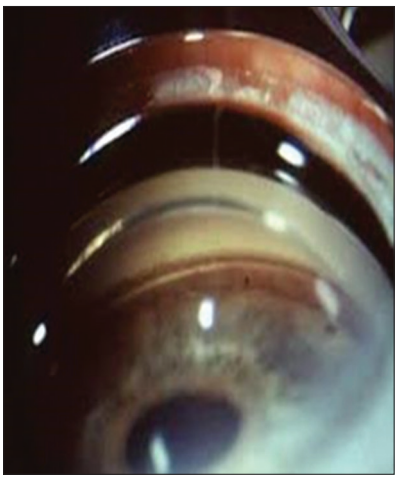

Figure 1: Gonioscopic view of angle recession in the right eye

to raised (IOP), thereby increasing the risk of optic neuropathy. It includes lens particle glaucoma, angle recession glaucoma, phacoantigenic glaucoma, and hemolytic or ghost cell glaucoma [4].

Angle recession was first described by Collins in 1892 [5]. Angle recession occurs due to a tear between the longitudinal and circular muscle fibers and often includes the trabecular meshwork (TM). Glaucoma can occur soon after ocular trauma or may develop over the course of months to years. Angle recession glaucoma is classified as a type of traumatic secondary open-angle glaucoma. The most common cause is from sports-related injuries such as baseball or boxing [4]. The reported frequency of angle recession as a complication of blunt trauma is 20–94%. It has been observed that the angle recession occurs in 71–100% of cases of blunt trauma with hyphema. Approximately 1–20% of eyes with angle recession will develop glaucoma [6]. Various studies have shown the incidence of glaucoma in 10% cases after 10 years of follow-up post-trauma [7]. The development of glaucoma in the fellow eye is seen in 50% of cases where the angle regression develops into glaucomatous optic atrophy [8].

The exact mechanism involved is not known. It is believed that it is due to the chronic progressive mechanism of trabecular outflow dysfunction, leading to pressure elevation over time. The proposed mechanism of recession is that blunt trauma forces aqueous humor laterally and posteriorly against the iris and angle. This exerts traction on the iris root leading to a tear between the longitudinal and circular muscles of the ciliary body. With enough force, the ciliary arteries can be broken, leading to a hyphema. This initial insult may damage the TM and Schlemm's canal leading to an early IOP spike. Long-term scarring and fibrosis of the TM/Schlemm's canal can lead to an elevated pressure year down the road [9]. Loss of tension of ciliary muscle on the scleral spur, thus narrowing Schlemm's canal and the hyaline membrane, has been reported to grow across the TM which may be another mechanism to explain decreased aqueous outflow [10].

Sihota *et al*. [11] found the presence of increased pigmentation at the angle, elevated baseline IOP, hyphema, lens displacement, and angle recession of more than 180° were significantly associated with the occurrence of chronic glaucoma after closed globe injury. Gonioscopy [12] is the only clinical procedure that must be performed before the angle of recession can be diagnosed. The key examination finding in angle recession is the widening of the ciliary body band which is seen on gonioscopy. Widening of the ciliary body band in the presence of elevated pressure and nerve damage leads to the diagnosis of angle recession glaucoma. Some normal eyes have a broad ciliary body band and therefore, comparison with the fellow eye is of utmost importance to avoid mistaking physiologic or even 360-° recession as normal. Use of a 1- or 3-mirror Goldman goniolens, which provides the greatest magnification of angle structures, is recommended.

Angle recession will manifest as a tear in the ciliary body between the longitudinal and circular fibers. Acutely, classic gonioscopic findings include brown-colored, broad-angle recess, glistening white scleral spur, and depression in the overlying TM. Chronically, findings include peripheral anterior synechiae at the border of the recession or anywhere in the angle and damaged iris processes. Comparison with the angles in the injured and uninjured eyes is important, particularly in cases with subtle findings.

Documented asymmetry supports the diagnosis. Detailed and meticulous Slit lamp (SL) examination should be undertaken to look for other evidence of ocular trauma such as sphincter tears, corneal scars, vossius ring, iridodialysis, iridodonesis, phacodonesis, and hyphema. Visual field analysis is the most important adjunctive diagnostic modality for detection and follow-up of the disorder. OCT RNFL is also important for documenting and monitoring glaucoma. High-frequency ultrasound bio-microscopy is effective for evaluating abnormalities of the angle in the AC in cases where gonioscopy is difficult or impossible due to corneal edema, corneal scarring, hyphema, synechia, or other opacity. Zonular deficiency and angle recession are the most common ultrasound biomicroscopy (UBM) findings in a closed-globe injury [13]. UBM is a useful adjunctive modality for the evaluation of abnormalities in closed-globe injuries and may be superior to SL-OCT to image angle recession [14].

Differential diagnosis involves unilateral steroid use, surgical anterior segment trauma (e.g., an AC IOL, uveitis-glaucoma-hyphema syndrome, iridocorneal endothelial syndrome, and carotid-cavernous fistula). Pseudoexfoliation or pigmentary glaucoma must be considered in the differential diagnosis as well.

Medical management is dependent upon the severity of the initial injury and the variable clinical course. It is always indicated when is greater than an arbitrary range of 25–28 mm Hg and/or when the glaucomatous optic nerve or visual field changes are documented over time. Topical aqueous suppressants are effective, including beta-blockers, carbonic anhydrase inhibitors, and alpha agonists. Prostaglandin analogs are pro-inflammatory and there should be avoided in the initial acute phase. However, prostaglandin analogs can be used after the acute phase is over because they have a theoretical benefit of bypassing the dysfunctional TM by increasing the uveoscleral outflow. Pilocarpine exacerbates angle recessions and therefore should be avoided. If the pupillary block is present from lens dislocation, cycloplegics may be helpful until the surgery is performed.

Topical medical treatment may be effective in cases of mild-to-moderate angle recession, while elevated IOP of eyes with extensive-angle injury eventually may become refractory to medications. Severe early cases may fail to show an initial response to aggressive medical treatment, indicating a poorer overall prognosis [15]. Surgical management of angle recession is indicated in cases where the maximally tolerated medical treatment fails to control the IOP; and risk of progressive visual loss outweighs the estimated risk of the planned surgical management. In general, outcomes of surgical treatment are less favorable than cases of POAG [16]. These modalities of treatment include Nd:YAG laser trabeculopuncture, laser trabeculoplasty [17], filtration surgery [18], and glaucoma drainage devices.

In the management of severe blunt trauma cases involving angle recession with dense vitreous hemorrhage and/or retinal detachment, combined trabeculectomy and pars plana vitrectomy have been reported with some successful outcomes [19]. Cyclodestructive procedures may be an alternative option for eyes with limited visual potential. Angle recession glaucoma is generally more difficult to control medically and surgically than other types of glaucoma.

The patients should be adequately counseled and follow-up examinations should be performed regularly. The follow-up depends on the degree of IOP control and the risk of progressive visual loss of the visual the patients with an early increase in IOP after blunt trauma should be re-examined every 4–6 weeks during the 1st year to monitor their condition. The patients with angle recessions of >180° without evidence of glaucoma should be advised of the need for lifelong follow-up care.

CONCLUSION

The bimodal distribution of post-traumatic glaucoma shows two peaks of incidence, one at <1 year and other at 10 years after the trauma. It is important to diagnose the risk factors for traumatic glaucoma so that ocular morbidity is prevented by appropriate therapy as early as possible.

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