Abstract: Hypotonous maculopathy is a condition characterized by hypotony associated with fundus abnormalities, including disc edema, vascular tortuosity, and chorioretinal folds. Incidence of hypotonous maculopathy is high following glaucoma filtering surgeries but can also occur following trauma. Once the cause is known, patients should be treated as soon as possible because delayed normalization of the intraocular pressure (IOP) may result in permanent macular chorioretinal changes and poor vision. Very few cases of traumatic hypotonous maculopathy has been described in literature.

Keyword: cyclodialysis cleft, hypotonous maculopathy, trauma

INTRODUCTION:
“Statistical hypotony” can be defined as an intraocular pressure (IOP) less than 6.5 mmHg, which corresponds to more than three standard deviations below the mean. “Clinically significant” hypotony represents the condition where the IOP is low enough to result in visual loss (4). Hypotony may result in maculopathy. The diagnosis and treatment are particularly difficult. The treatment aims at normalizing intra-ocular pressure (IOP), which usually brings about an improvement in visual acuity. There are many causes of post traumatic hypotonous maculopathy which include cyclodialysis cleft, ciliochoroidal detachment, retinal detachment or scleral rupture.

Cyclodialysis clefts are a rare ocular pathology that result from a disruption of the circumferential insertion of the meridional ciliary muscle fibers from the scleral spur, usually of traumatic etiology. Cyclodialysis may be caused accidentally by trauma, iatrogenically during intraocular surgery, or intentionally for the treatment of glaucoma. An abnormal route of drainage of aqueous humor is created into suprachoroidal space, as well as decreased aqueous production due to diminished blood supply to the ciliary body which can lead to chronic hypotony. Treatment is initially conservative. When the conservative medical treatment fails a surgical approach may be required to close the cleft.

CASE SUMMARY:
A 42 year old male patient presented with complaints of defective vision in the right eye following injury with a plastic ball 1 week ago. He was diagnosed as a case of traumatic anterior uveitis elsewhere and was treated with prednisolone 60 mg once daily, topical steroids hourly and atropine eye drops three times a day for 7 days when he presented to us.

On examination, visual acuity in the right eye was 2/60 not improving with pin hole and in the left eye it was 6/6. Anterior segment examination of the right eye revealed that the lids were normal. Extraocular movements were full. Conjunctiva was clear. Cornea showed few pigments over the back of the endothelium. Anterior chamber had 1+ cells and flare. Iris colour and pattern was found to be normal. Pupil was 5 mm pharmacologically dilated and not reacting to light and the lens was found to be clear with no evidence of phacodonesis. Anterior segment examination of the left eye was normal.

A dilated fundus examination of the right eye showed that the media was clear with disc margins being blurred and choroidal folds seen around the macula. The left eye fundus examination was found to be normal.

Intraocular pressure measurement by Goldmann applanation tonometry was found be 6mmHg in the right eye and 18mmHg in the left eye. Gonioscopy was possible as media was clear and was done using Ziesi four mirror which showed presence of probably a cyclodialysis cleft. In order to confirm the gonioscopic finding ultrasound biomicroscopy was done.
which showed a classic cyclodialysis cleft as an echolucent gap between the low-reflective ciliary body and the high-reflective sclera, extending from 9 – 12 o clock.

Figure 2 Gonioscopic examination of the right eye showing a cyclodialysis cleft
Considering the diagnosis of hypotonus maculopathy accounting for the defective vision secondary to cyclodialysis cleft the patient was advised to continue 1% topical atropine eye drops twice daily in the right eye. On follow up after 1 week IOP increased to 8 mm of Hg and visual acuity was 6/36. At one month follow up patient’s visual acuity in the right eye improved to 6/12 with -1.5D sphere and +1.00D cylinder at 110O. The intraocular pressure as measured using Goldmann applanation tonometry showed that the intraocular pressure increased to 12mmHg.

Figure 3 Slit lamp fundus photograph of the right eye showing hyperaemia of the disc as indicated by black arrow and presence of chorioretinal folds as indicated by red arrow

Figure 4 UBM picture of the right eye showing cyclodialysis cleft

DISCUSSION:
Hypotony maculopathy was first described by Dellaporta in 1954 and it occurs usually after antiglaucoma surgery or after trauma. Later Gass (1972) coined the term ‘hypotonus maculopathy’, to emphasize that alterations in the macular region were primarily responsible for the loss of central vision. It is characterised by presence of fundus changes which include disc edema, vascular tortuosity and chorioretinal folds. These changes are possibly due to hypotony causing collapse of scleral wall throwing the retina and choroid into folds. The chorioretinal folds produce visual dysfunction due to distortions of overlying retinal photoreceptors. Disc edema occurs possibly due to anterior bowing of lamina cribrosa affecting the orthograde and retrograde transport in the axons leading to stasis of axoplasm and edema of the disc. This condition is usually reported following antiglaucoma surgeries especially more so with the use of adjunctive antimetabolites like Mitomycin C and 5-fluorouracil. But can also occur following trauma due to variety of reasons namely iridocyclitis, cyclodialysis, ciliochoroidal detachment, retinal detachment or scleral rupture. Prompt identification and restoration of normal IOP is necessary in order to prevent prolonged hypotony causing irreversible fibrosis within the retina, choroid or sclera, maintaining the choroid in a folded position thereby affecting the vision of the patient which cannot be restored again.

Cyclodialysis establishes an abnormal pathway for the aqueous humor to reach the suprachoroidal space and causes secondary hypotony. However, the magnitude of the hypotony is not proportional to the size of the cyclodialysis cleft. Although the diagnosis can often be made by gonioscopy, clefts may be difficult to detect in recently traumatised or operated eyes because of hazy media, hypotony, shallow anterior chamber, or abnormal anterior segment architecture. Treatment of cyclodialysis induced hypotony should start once the cyclodialysis cleft is identified. Due to its low prevalence, it is difficult to determine the best treatment for cyclodialysis.

For small clefts, typically less than 4 clock hours, the first intervention is medical treatment with a topical cycloplegic agent such as atropine 1% for 6 to 8 weeks. 1% atropine eye drops displaces the ciliary muscle backwards and outwards, bringing it close to the scleral spur and closing the gap from the posterior area. If medical treatment is ineffective, noninvasive management with laser photocoagulation applied to the cleft should be attempted. The mechanism by which laser treatment causes reversal of hypotony is unknown. It is assumed that swelling of the choroid following laser treatment closes the cleft and blocks aqueous flow into the suprachoroidal space, or, perhaps, the iritis that is caused by the treatment plays a role in altering the aqueous humor composition, thereby obstructing drainage. If medical/noninvasive laser methods do not work then surgical methods become the next option. Surgical options include direct/indirect cyclopexy by ciliochoroidal diathermy or anterior scleral buckling. Large chronic clefts are reported to benefit from parsplana vitrectomy, cryotherapy and gas tamponade. Also transscleral diathermy, transscleral cyclophotocoagulation using Nd:Yag laser and transconjunctival cryotherapy have been reported to induce an inflammatory reaction to enhance cleft closure.

The loss of vision secondary to macular edema can be permanent if not treated in time. However, the period after which irreversible alterations occur in the eyesight is not clear.

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Alternate surgical techniques employ a capsular tension ring or an IOL. A capsular tension ring in the sulcus as well as the large haptics of a PMMA lens take advantage of internal cerclage by compressing the cleft against the sclera to close it. These novel techniques are currently described only as case reports and have not been studied extensively.

Conclusion: It is important to identify a cyclodialysis cleft in secondary hypotony maculopathy after blunt trauma and patients should be treated as soon as possible because delayed normalization of the intraocular pressure (IOP) may result in permanent macular chorioretinal changes and poor vision. Management of cyclodialysis clefts requires a careful and logical approach. Conservative and noninvasive management is the best approach initially. When it fails, more invasive surgical interventions are required.

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