Anterior segment ischemia following Hummelsheim procedure in a case of sixth nerve palsy

Bansal Rakesh K, Bamotra Ravi K

A, 46-year-old Indian male, known hypertensive presented with left esotropia of 25 prism diopters (PD) after head injury in a roadside accident 9 months back. The deviation was constant in nature and was associated with complaints of diplopia in left lateral gaze. Traumatic sixth nerve palsy was diagnosed. The patient underwent left medial rectus recession of 5 mm and a split-tendon transposition of the left superior and inferior recti to the lateral rectus insertion (Hummelsheim procedure). On the first postoperative day, the patient developed corneal edema and anterior chamber reaction of flare 2+ and cells 2+. The pupil was semi-dilated and was sluggishly reacting to light. Anterior segment ischemia was diagnosed, which was managed with topical and systemic steroids.

Key words: Anterior segment ischemia, Hummelsheim procedure, sixth nerve palsy, steroids

Anterior segment ischemia (ASI) after extraocular muscle surgery is reported to occur once more than two extraocular muscles are operated. It results from inadequate perfusion of the iris and ciliary body owing to disruption of blood supply to iris and ciliary body which mainly comes through anterior ciliary blood vessels, which are seven in number with each rectus muscle contributing two anterior ciliary vessels except lateral rectus muscle which contributes only one ciliary vessel. Primate studies have shown that 70–80% of the blood supply to the anterior segment of the eye is contributed by the anterior ciliary system in addition to collateral blood supply from the anterior episcleral arterial circle.[1]

Incidence of severe ASI following strabismus surgery has been reported to be 30/400,000 surgeries.[2] It mostly occurs in adults and after transposition of the vertical recti. Symptoms usually occur after 1–2 days of surgery, which include conjunctival chemosis, corneal edema, anterior chamber reaction, lens opacities, hypotony and sector iris perfusion defects.[3] Late sequelae include correctopia, iris atrophy, cataract and on rare occasions phthisis bulbi.[2]

Access this article online

Quick Response Code:

Website: www.ijo.in

DOI: 10.4103/0301-4738.162619

Department of Ophthalmology, Government Medical College and Hospital, Chandigarh, India

Correspondence to: Dr. Bansal Rakesh K, Department of Ophthalmology, GMCH-32, Chandigarh, India. E-mail: bansalrk@hotmail.com

Manuscript received: 25.09.14; Revision accepted: 13.02.15

There are number of risk factors which contribute towards ASI such as; micro-vascular disease, sickle cell disease, leukemia, thyroid eye disease, high myopia, previous scleral buckling, previous strabismus surgery and radiation therapy.[3,4]

We report a case of traumatic left sixth nerve palsy in a 46-year-old man, who developed ASI following medial rectus recession and split thickness vertical muscle transposition (Hummelsheim) on the left eye.

Case Report

A, 46-year-old Indian male presented to our hospital with complaints of inward deviation of his left eye for the last 9 months, which he developed after sustaining a head injury. The deviation was associated with horizontal diplopia, which was more in the left lateral gaze. The patient was a known hypertensive and was on medication for the past 7 years.

Examination showed best-corrected visual acuity (BCVA) of 20/20 in both eyes. The alternate cover test showed left esotropia of 25 PD in primary gaze [Fig. 1]. Ocular motility examination showed absent abduction in the left eye. Examination of the anterior segment was normal. Pupils were normal in size and were reacting to light. Fundus examination was normal in both eyes. Intraocular pressures (IOP) were 14 mmHg in each eye. Forced duction and force generation tests were negative.

Patient underwent left medial rectus recession of 5 mm from the insertion and a split-tendon transposition of the left superior and inferior rectus muscles to the lateral rectus insertion (Hummelsheim procedure) through limbal conjunctival peritomy.

On the first postoperative day, the visual axis was parallel. BCVA of the left eye was 20/60. There was moderate corneal edema with anterior chamber reaction of flare 2+ and cells 2+ on slit lamp biomicroscopy. Left eye pupil was semi-dilated and was sluggishly reacting to light. The IOP was 18 mmHg in the left eye. The posterior segment was normal. Diagnosis of ASI was made. Patient was started on topical and systemic steroids. After 1-week of steroids, visual acuity was 20/40. Corneal edema and anterior chamber reaction had reduced. The pupil was still mid dilated and was nonreacting to light [Fig. 2]. IOP was 4 mmHg in the left eye.

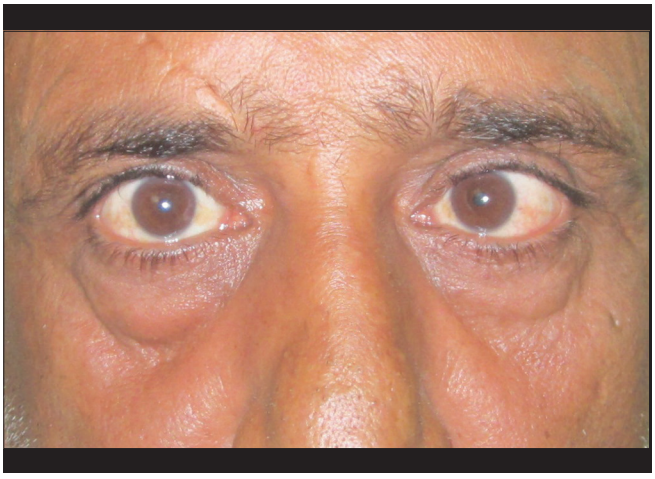

Figure 1: Preoperative photograph shows left esotropia

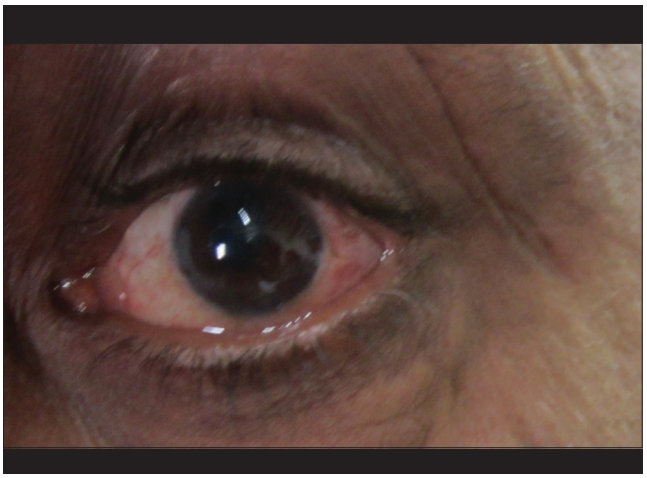

Figure 2: One week postoperative photograph shows mild conjunctival congestion, no corneal edema and a mid-dilated pupil

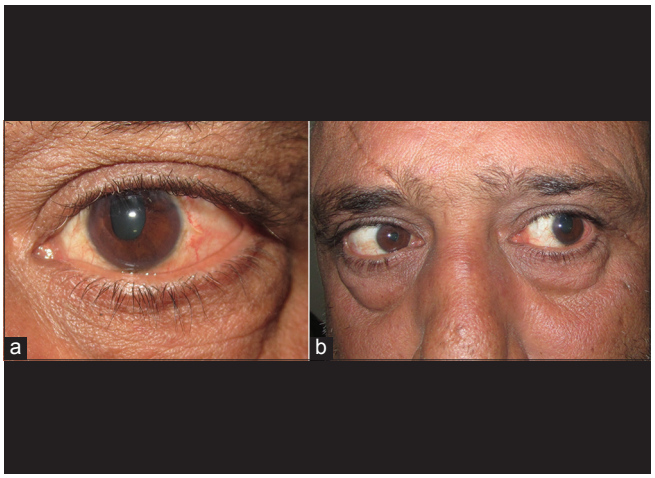

Figure 3: (a) One year postoperative photograph shows quite eye with mid dilated pupil, (b) 1-year postoperative photograph shows normal abduction range

Patient was followed up regularly. Oral and topical steroids were tapered and stopped after 1 month. One year after the surgery, visual acuity had improved to 20/30. On anterior segment examination, the cornea was clear, the anterior chamber was quiet, the pupil was mid-dilated and nonreacting to light [Fig. 3a]. Visual axis was parallel and abduction of the left eye was in near normal [Fig. 3b]. IOP and posterior segment were within normal limits.

Discussion

Our patient developed ASI following one muscle recession, and two muscle split muscle transposition, which is equivalent to two muscle surgery as the second halves of these vertical recti muscles still continue to contribute one ciliary vessel each. However, our patient was a known hypertensive, which could have been a risk factor for the development of ASI. Al Enezi and Al Wayel[4] reported ASI in a 35 years old myopic patient following a similar procedure. They proposed that high myopia could also be a risk factor for ASI with vertical muscle surgery. A two-staged procedure with an interval of 4–6 months could have prevented ASI in our patient with hypertension as a risk factor as this would have allowed time for remodeling of the collateral circulation.[5]

Both fornix and limbal incisions are associated with ASI. However, limbal incisions are said to predispose to more severe ischemia compared to fornix incisions as these compromise the anterior episcleral arterial circle.[6] Fornix incision also could have been a better option for this patient.

Microscopic surgery on recti muscle for carefully dissecting anterior ciliary vessels before disinsersion can also be considered for high-risk patients as this helps to keep the anterior ciliary vessels intact and prevent ASI.[7]

Conclusion

Anterior segment ischemia should be kept in mind during vertical muscle transposition in high risk patients during strabismus surgery.

References

1. Wilcox LM, Keough EM, Connolly RJ, Hotte CE. The contribution of blood flow by the anterior ciliary arteries to the anterior segment in the primate eye. Exp Eye Res 1980;30:167-74.
2. France TD, Simon JW. Anterior segment ischemia syndrome following muscle surgery: The AAPO and S experience. J Pediatr Ophthalmol Strabismus 1986;23:87-91.
3. Simon JW. Complications of strabismus surgery. Curr Opin Ophthalmol 2010;21:361-6.
4. Al Enezi MH, Al Wayel AH. Anterior segment ischemia in a young myopic following transposition surgery. Middle East Afr J Ophthalmol 2008;15:31-3.
5. Simon JW, Grajny A. Anterior segment ischemia following augmented 2-muscle transposition surgery. J AAPOS 2004;8:586-7.
6. Murdock TJ, Kushner BJ. Anterior segment ischemia after surgery on 2 vertical rectus muscles augmented with lateral fixation sutures. J AAPOS 2001;5:323-4.
7. McKeown CA. Techniques for preserving the anterior ciliary vessels in strabismus surgery. Ophthalmol Clin North Am 1992;5:143-56.

Cite this article as: Bansal RK, Bamotra RK. Anterior segment ischemia following Hummelsheim procedure in a case of sixth nerve palsy. Indian J Ophthalmol 2015;63:543-4.

Source of Support: Nil. **Conflict of Interest:** None declared.