

Lens Expulsion into Sub-conjunctival Space Following Peribulbar Anaesthesia

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Various studies have shown that there is an incidence of ocular penetration during peribulbar and retrobulbar anaesthesia of <0.1% for eyes having axial length <26mm,^{1,2} and an incidence of <1% for eyes of axial length >26 mm.³ Damage to the globe after penetration can result from either toxic effects of anaesthetic drugs⁴ or from mechanical injury caused by the needle itself. Mechanical injury from the needle can be from a single penetration or a double perforation of the globe or from direct optic nerve penetration.

Case Report

We would like to report the occurrence of an ocular explosion after peribulbar anaesthesia. A 65-year-old male was referred to us with a report suggesting a subconjunctival cyst in the supero-temporal quadrant of his right eye (Figure). This swelling had been noticed by the referring ophthalmologist immediately after giving the patient a peribulbar block 2 months earlier for a planned cataract surgery. About 5 cc of anaesthetic (combination of 2% xylocaine and 0.5% sensorcaine) had been injected in each of the superonasal and inferotemporal peribulbar sites. No digital massage was given since the swelling appeared immediately after the injection. Surgery was abandoned and the patient was referred to our centre.

On examination we found that the lens of the patient had been expelled into the subconjunctival space. The overlying and surrounding conjunctiva was mildly hyperemic. We thoroughly examined the sclera of the same eye as well as the other eye to look for any predisposing thin areas or signs of previous scleritis but could find none. Pupil was drawn superotemporally towards the site of the displaced lens. The eye was aphakic and there were no capsular remnants seen. Vitreous face was broken and a few strands were seen in the anterior chamber. A minimal resolving vitreous haemorrhage was noticed in the inferior part. Rest of the fundus looked normal and in spite of thorough peripheral examination with scleral indentation no needle entry wound was found. Ultrasonography confirmed the clinical findings of inferior vitreous haemorrhage, and axial length was found to be 24.5 mm. The best corrected visual acuity was counting fingers at 1.5 ft with +11 D which could not be explained as the media were clear and there were no obvious posterior pole changes. The intraocular pressure (IOP) in the right eye was 7.1 mm Hg and in the left eye 14.6 mm Hg with Schiotz tonometer.

We scheduled the patient for lens removal from the subconjunctival space and carefully dissected the lens from the overlying conjunctiva and underlying sclera. On removal of the lens the underlying sclera showed a brownish coloured sealed defect. The lens tissue measured 9 mm in diameter and the sealed defect was 7.5 mm. The uveal tissue had sealed the gap. We did not try to dissect it further lest the wound give way. The conjunctiva was resutured.

Three months after the surgery, the vision was still unchanged. The IOP was 12.2 mm Hg and pupil was updrawn and not reactive to light. The disc and the macula appeared normal but there was still a resolving vitreous haemorrhage in the inferior part.

Discussion

We could not explain the poor visual acuity since the posterior pole was seemingly normal and despite a non-reactive pupil there was no obvious optic atrophy. Probably it might develop in due course. Transient visual loss has been described due to intraocular xylocaine toxicity⁵ but none that would last so long. Hypotony in the immediate post lens expulsion period could be explained due to the sudden rupture of the globe leading to decompartmentalisation of the eye and possibly a ciliary shock which recovered in due course. The site of perforation was possibly in the region of the resolving vitreous haemorrhage and thus could not be seen.

A similar case has been reported recently, in which the lens prolapsed into the subconjunctival space after

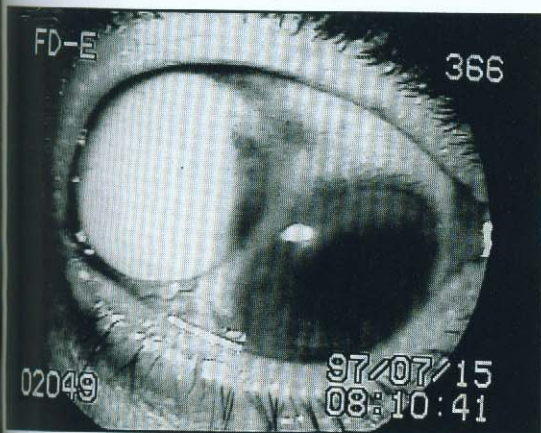


Figure. Lens in subconjunctival space after peribulbar injection

an accidental perforation of the globe while giving peribulbar anaesthesia.⁶ The authors of this report conducted an experimental study where they ruptured 21 cadaver eyes with intraocular saline injections. Of the 21 eyes, 11 ruptured in the perilimbal region while 10 ruptured in the equatorial area. Of the 11 perilimbal ruptures, 3 had lens extrusion into the subconjunctival space. The extremely high IOP achieved just before globe rupture was preceded by corneal whitening and a marked resistance to further advancement of the injecting syringe plunger. These signs should be considered to strongly suggest an impending disaster. Also, a sudden localised swelling coming up in the subconjunctival space immediately after a peribulbar injection could quite possibly be a lens expelled due to the raised volume in the posterior chamber.

References

1. Ramasay RC, Knobloch WH. Ocular perforation following retrobulbar anaesthesia for retinal detachment surgery. *Am J Ophthalmol* 1978;86:61-64.
2. Schechter RJ. Management of inadvertent intraocular injection. *Ann Ophthalmol* 1985;17:771-75.
3. Hay A, Flynn Jr HW, Hoffman JL, Rivera AH. Needle penetration of the globe during retrobulbar and peribulbar injections. *Ophthalmology* 1991;98:1017-24.
4. Kimble JA, Morris RE, Witherspoon CD, Feist RM. Globe perforation from peribulbar injection. *Arch Ophthalmol* 1987;105:749.
5. Lincoff H, Kreissig I. Local anaesthesia with accidental perforation of the eye: an acute emergency. *Klin Monatsbl Augenheilkd* 1986;188:128-32.
6. Magnante DO, Bullock JD, Green WR. Ocular explosion after peribulbar anaesthesia. *Ophthalmology* 1997;104:608-15.

Bilateral Accessory Iris Membrane

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Accessory iris membrane is a rare congenital anomaly resulting from hyperlasia of the superficial mesodermal layer of the iris.¹ It presents as an additional layer of iris tissue giving the impression that the iris is duplicated. We present a unique case of bilateral accessory iris membrane associated with microcornea.

Case Report

A 24-year-old male presented with history of low vision in the right eye since childhood. There was no family history of any eye problem and his parents were not consanguineous. On examination, unaided visual acuity

in the right eye was 1/60 with no improvement after refraction. Visual acuity in the left eye was 6/18, which improved to 6/6 after refraction (+1.0 D, +2.75 D x 180). Right eye had esotropia (16 prism dioptres) and was amblyopic. The horizontal corneal diameter in both eyes was 9.5 mm. In the right eye, from the anterior aspect the iris bulged forward a normal coloured additional layer with multiple full-thickness irregular defects and a pseudopupil in the centre (Figure 1). The accessory layer of iris tissue commenced from the prominent collarette at approximately 2 mm from the pupillary margin. Superiorly, the pupillary end of the accessory layer was adherent to the lens which had localised cataractous changes at the site of attachment. The fundus was not clearly visible in this eye; the

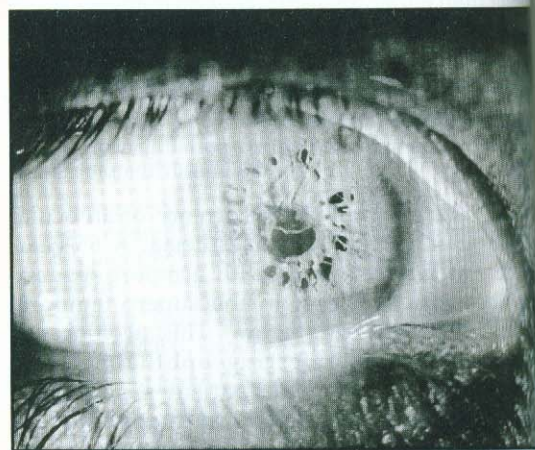


Figure 1. Right eye after mydriasis showing the accessory iris layer with multiple full thickness irregular defects around the spurious pupil

The lens is visible through the defects.

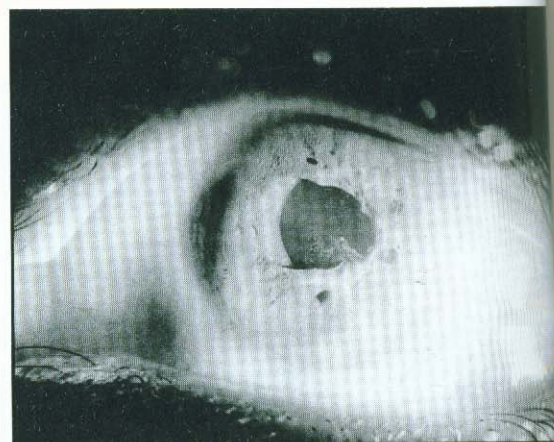


Figure 2. Left eye after mydriasis showing the accessory iris layer with multiple small defects and a large defect nasally through which normal pupil can be seen

however appeared to be normal. The left eye also had a similar well developed accessory iris layer commencing at the collarette, with a spurious pupillary aperture and multiple defects (Figure 2). In this eye one strand of the accessory layer adherent to the lens at 5 o'clock position and there were a few pigment deposits on the anterior lens surface. The lens was clear, however, and the fundus was normal. In both eyes the normal pupil was seen through the defects and was reacting to light unlike the spurious pupil. The intraocular pressure was 14 mm Hg in both eyes, and the angle of anterior chamber was open (30°, regular, scleral spur: Spaeth grading system) in both eyes. On general physical examination the patient had no other systemic disorders.

Discussion

In the embryonic life, the superficial mesodermal layer of the iris which terminates at the lesser circle normally undergoes atrophy and persists only as a thin tissue interspersed with crypts.^{1,2} Rarely this stromal layer of iris may be hypertrophied resulting in an anterior accessory layer of iris tissue giving the appearance that the iris is duplicated. The hyperplasia may present in various forms, it may affect a segment of iris or may occur around the entire circumference of the pupil. In extreme cases it may extend across the pupil with multiple full-thickness defects and a virtual second pupillary aperture which however has no muscular activity. This condition is usually sporadic but occasionally it may have an autosomal dominant mode of inheritance. We examined all members of our patient's family covering three generations, but none of the family members had similar finding or any other ocular anomaly.

Accessory iris membrane is a rare congenital disorder. Few reports of this anomaly were published in the past,¹ but to the best of our knowledge this condition has not been reported in recent literature. Its association

with persistent pupillary membrane, coloboma and anterior polar cataract has been previously reported.^{1,3} This case report is of a unique case of bilateral accessory iris membrane associated with microcornea in both eyes, and cataract in one eye. In 1957, Levy reported a case of bilateral accessory iris membrane with persistent pupillary membrane but no other associated ocular anomalies.³ There are conflicting reports in literature about the overlapping nature of accessory iris membrane and extreme forms of persistent pupillary membrane.⁴ Although these two conditions have similar origin, the clinical appearance of the two entities is quite different. Persistent pupillary membrane even in its extreme forms presents as a translucent or opaque membranous structure and extends across the pupil. Whereas in accessory membrane, also called iris duplication, the accessory layer closely resembles the normal iris tissue in colour and thickness. Also, the anterior accessory iris layer may be differentiated to form a virtual second pupillary aperture unlike the former condition. Microscopically, it has been demonstrated in the past that in case of accessory iris membrane the accessory tissue shows an extensive anomalous hyperplasia even when compared with pupillary membrane at the stage in fetal life when it has reached its maximum development.¹ Hence this condition is separate entity and should be classified a hyperplasia of the stromal layer of iris and rightly called accessory iris membrane.

References

1. Duke-Elder S. *System of Ophthalmology*. London: Henry Kimpton; 1964. Vol 3, part 2. p 587-89.
2. Sorsby A. *Modern Ophthalmology*. 2nd ed. London: Butterworths; 1972. Vol 3. p 267.
3. Levy WJ. Congenital iris lesion. *Br J Ophthalmology* 1957;41:120-23.
4. Cassady JR, Light A. Familial persistent pupillary membranes. *Arch Ophthalmol* 1957;58:438-48.