CASE REPORT

Toxic anterior segment syndrome following phakic posterior chamber IOL: a rarity

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SUMMARY

Implantable collamer lenses (ICL) have gained popularity for correction of myopia where kerato-refractive procedures are not indicated as in cases of high myopic refractive errors. Toxic anterior segment syndrome (TASS) is a very uncommonly reported postoperative complication following ICL implantation. A young patient developed severe corneal oedema and anterior segment inflammation on the first day after ICL implantation. Analysing retrospectively, possible idiosyncratic response to intracameral pilocarpine was considered as a cause for TASS. Prompt and intensive therapy with oral and topical potent steroids was visually rewarding. TASS, though a sterile inflammation can have catastrophic sequelae such as corneal decompensation and secondary glaucoma. Hence, timely identification and management is important.

BACKGROUND

Implantable collamer lenses (ICL) have gained extensive popularity in the field of refractive surgery with improving technologies and better results. They are particularly useful in patients with high refractive errors and in those where kerato-refractive surgery is relatively contraindicated.[1] Although these lenses have proven benefits, they are known to be associated with certain postoperative adverse events. With the newer designs and better manufacturing protocols, complications and adverse events following insertion of phakic intraocular lenses (pIOLs) have impressively declined. Currently, the ICL (Staar company) is one of the preferred lenses, owing to the minimal complications like early cataractogenesis,[1] glaucoma,[2] endothelial cell loss and inflammation associated with its implantation.[3]

Toxic anterior segment syndrome (TASS) is one such postoperative event, which very rarely may follow insertion of an ICL.[4 5] Here in, we report a case of post-ICL implantation TASS in a young patient with high myopia.

CASE PRESENTATION

A 25-year-old woman presented to our outpatient department for the purpose of refractive surgery. She was the mother of a 3 year-old with no significant systemic or drug history. She was regularly using spectacles for refractive correction since the age of 14 years with occasional use of soft contact lenses during the last 4 years. She gave a history of significant change in refractive correction 5 years ago which had been stable henceforth. On evaluation her uncorrected distance visual acuity (UCVA) was right eye (OD) 1/60 and left eye (OS) 2/60, which improved to 6/6 both eyes (OU) with a refractive correction of OD −8.00 dioptre sphere (DS)/−0.50 dioptre cylinder (DC)×90° and OS −7.25DS/−0.50 DC×180°. The anterior segment examination was within normal limits. The intraocular pressure (IOP) on applanation tonometry was 14 mm Hg OU. The central corneal thickness was 467 µm OD and 478 µm OS. The specular count was 3165 cells/mm^2 OD and 3169 cells/mm^2 OS. Corneal topography (Pentacam HR, Oculus) OU revealed steep corneas with a symmetrical bow tie. The posterior elevation maps were within normal range. Anterior chamber depth measured from the endothelium was 3.52 mm OD and 3.42 mm OS. The white-to-white diameters were OD 12.01 mm and OS 12.04 mm on three consecutive measurements using digital callipers when measured by two independent observers. The fundus examination revealed changes of myopia OU with no peripheral treatable lesions. In view of thin corneas with high refractive error, a decision for bilateral ICL-V4c (Staar company) implantation was made. The patient underwent uneventful implantation of ICL-V4c (−9.50 diopter power (overall diameter:13.2)) OD under topical anaesthesia. Intraoperatively, a viscodispersive type of ophthalmic viscosurgical device (OVD) that is, 2% hydroxylpropyl methylcellulose was used. At the end of the surgery, 0.2 mL of 0.5% pilocarpine (preservative-free, Carpinol, Sunways, India) was injected in the anterior chamber.

On the first postoperative day, the UCVA was counting fingers. There was minimal ciliary congestion, mild corneal oedema and a 3 mm whitish mobile hypopyon in the anterior chamber. The pupil was mid-dilated to 6 mm with poor response to photopic stimulation (figure 1). The patient had mild photophobia with no evidence of pain. The IOP as measured by non-contact tonometry was noted to be 18 mm Hg. Fundus examination revealed no evidence of posterior segment inflammation. A diagnosis of most likely TASS rather than endophthalmitis was made.

INVESTIGATIONS

Posterior segment ultrasound of the right eye was anechoic.

DIFFERENTIAL DIAGNOSIS

Post-ICL endophthalmitis.

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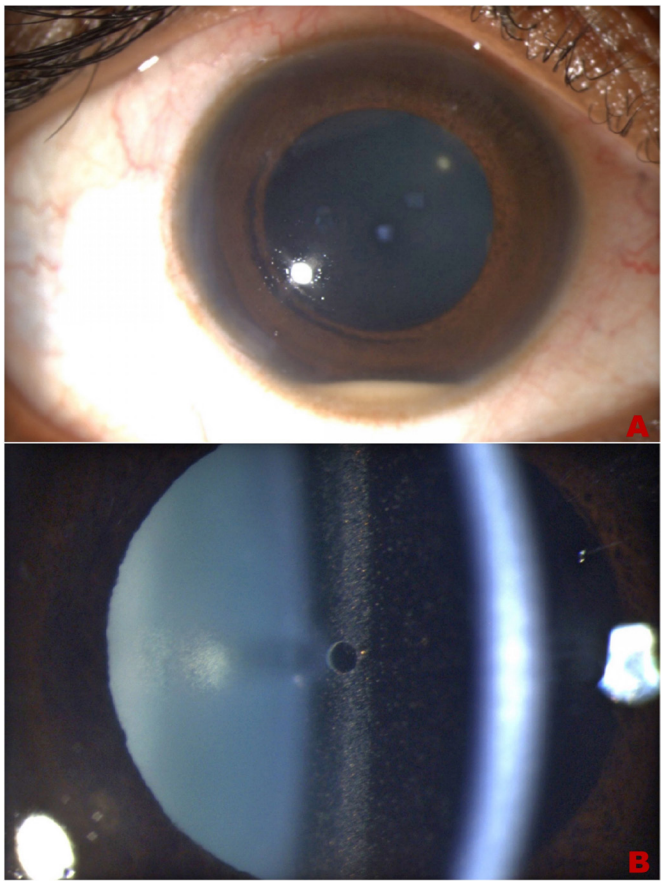

Figure 1 (A) Slitlamp biomicroscopic clinical photograph of the right eye showing under diffuse illumination the hypopyon. (B) Slit photograph under x16 magnification revealing anterior chamber reaction (B).

TREATMENT

The patient was treated with oral steroid therapy (prednisolone, 1 mg/kg) OD, tablet ciprofloxacin 750 mg twice a day (BD) and tablet pantoprazole 40 mg OD along with topical moxifloxacin 0.5% 4 hourly, topical prednisolone acetate 1% 1 hourly, topical tropicamide 1% four times in a day (QID) and topical carboxymethyl cellulose 0.5% 4 hourly. The patient was monitored at 8 hourly intervals for improvement or worsening of symptoms, visual acuity, corneal oedema, anterior and posterior chamber inflammation and IOP. Though there was no change in the initial few hours of the therapy, the IOP was noted to be raised to 24 mm Hg the next day. As clinically there still was nothing to suggest infectious aetiology, topical prednisolone was replaced with a higher potency steroid, 0.05% difluprednate 4 hourly along with addition of oral acetazolamide 250 mg 8 hourly. A decrease in the size of hypopyon was noted within the next 24 hours to 1 mm with a significant resolution of anterior chamber flare. The UCVA at day 3 was 6/60, IOP 13 mm Hg with relative improvement in signs. The topical difluprednate was then tapered slowly over the next 4 weeks. The oral steroids were continued for 10 days and stopped by which time the clinical signs had resolved, and the UCVA had improved to 6/12. Following cessation of the oral and topical steroids, the patient was maintained on topical nepafenac 0.1% three times per day for a period of 2 months.

OUTCOME AND FOLLOW-UP

The UCVA at the 15th day was noted to be 6/6. The patient was then planned for left eye ICL-V4c (−8.50 diopter power (overall diameter:13.2)) implantation under topical anaesthesia. The intraoperative and postoperative course for the left eye was uneventful. The same OVD was used intraoperatively for the left eye however, intracameral pilocarpine was not administered as following the previous experience with the fellow eye, pilocarpine was considered a possible cause of inflammation. The vaulting as measured on an anterior segment optical coherence tomography (AS-OCT) was 536 µm and 692 µm for the right and left eye, respectively.

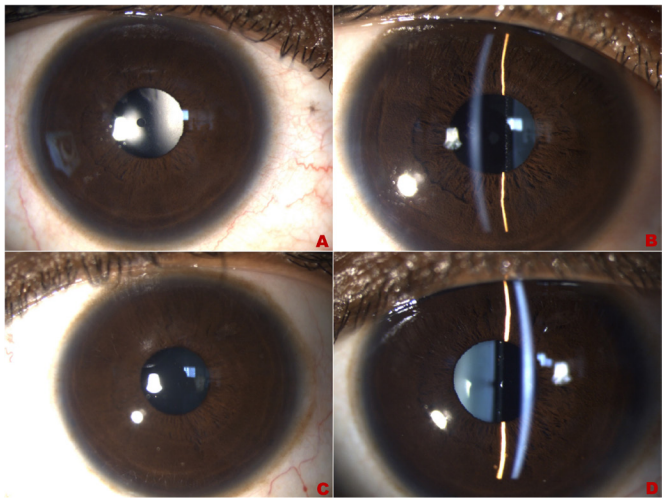

Figure 2 Slitlamp clinical photograph of the right (A, B) and left eye (C, D) under diffuse and slit illumination at 4 weeks postoperative period.

At 4 weeks, the UCVA OU was 6/6, cornea OU was clear, IOP was 12 and 14 mm Hg in the right and the left eye, respectively. The specular count was 3061 cells/mm^2 OD and 3109 cells/mm^2 OS. The photopic pupil diameter in both eyes was 3 mm (figure 2).

DISCUSSION

The incidence of postsurgical inflammation following posterior chamber pIOLs has been reported to be low when compared with anterior chamber pIOLs.[4–9] TASS is considered a very severe form of postsurgical inflammation and is defined as a unique entity. TASS following an anterior segment intraocular surgery has been reported most commonly following cataract extraction. It is a severe form of acute sterile inflammation in response to foreign toxic substances and characteristically involves only the anterior segment of the eye. It occurs due to the break down of the blood–aqueous barrier and is usually seen with in the initial 12–24 hours.[10–12] Although, it has been frequently reported following cataract surgery, TASS following ICL-V4c has been reported in very few cases previously.[4 5 8 9] Typically characterised by a decrease of vision, diffuse corneal oedema, anterior chamber reaction and hypopyon with a rise of IOP, it may have an atypical and variable clinical presentation. In comparison to endophthalmitis, the first postoperative day presentation of anterior segment hypopyon with no posterior segment inflammation and the absence of pain was in the favour of sterile inflammation. The close and grave differential diagnosis of TASS is postsurgical endophthalmitis. Hence, it is of prime importance to start steroid therapy under antibiotic cover in suspicious cases where chance of either is possible.

The common factors that have been implicated in aetiopathogenesis of TASS include inadequate removal of OVD, presence of preservatives and residues on cannulas which may not have been cleaned or sterilised properly. However, it is not always possible to trace and identify the actual causative agent.

In our case, we could not identify the exact cause, but the possibilities could be OVD residues or an idiosyncratic inflammatory response to intracameral pilocarpine.

It is important to identify and treat TASS with intensive steroid therapy as extensive anterior segment inflammation can result in grave sequelae such as toxic endothelial cell damage and corneal decompensation, trabecular meshwork damage and secondary glaucoma, synechiae formation, pupil irregularities and cystoid macular oedema. These sequelae lead to visual disturbances and loss of visual function and may require surgical intervention in rare cases. Further chances of early lenticular opacification increases following intense inflammation. This is of particular relevance in patients undergoing ICL implantation surgery, where obtaining good unaided vision is of prime importance. The management of TASS includes the use of oral and intensive topical potent steroids to decrease inflammation, cycloplegics to decrease the ciliary spasm and prevent synechiae formation, antiglaucoma agents to reduces IOP and antibiotics for prophylactic cover. TASS typically responds within 24–48 hours of starting steroid therapy and features of inflammation and corneal oedema typical resolve completely within few weeks. Failure to respond requires review of drug therapy and looking for a possible infective focus.

Patient's perspective

I was a little worried after my first eye surgery as I was able to see very less when compared to what I was expecting. But luckily with treatment my vision started to improve. And by the end of first week I was comfortable.

Learning points

- Toxic anterior segment syndrome (TASS) is a form of sterile inflammation of the anterior segment seen in post surgical cases.
- TASS when managed timely and effectively has an eventual good outcome.
- The most common differential diagnosis of TASS is endophthalmitis, which should always be kept in mind.

To conclude, TASS is a manageable postsurgical event which when diagnosed early and treated adequately with intensive steroid therapy can be controlled and is visually rewarding. It is preventable with proper and adequate surgical practice with use of intraocular agents and solutions as well as proper maintenance and sterilisation techniques for reusable instruments. Though rarely seen post-ICL implantation, it is a noteworthy complication, which an ophthalmic surgeon should be aware of as timely intervention is beneficial.

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