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Complete spontaneous crystalline lens dislocation into anterior chamber with severe corneal endothelial cell loss

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Abstract

**Purpose:** To report an unusual case of a spontaneously dislocated crystalline lens into the anterior chamber that was successfully treated.

**Design:** Interventional case report

**Methods:** A generally healthy 49-year-old woman with no history of trauma presented with a spontaneously dislocated crystalline lens into the anterior chamber accompanied by corneal endothelial cell loss. The crystalline lens was extracted intracapsularly following gentle separation from the corneal endothelium using a viscoelastic material.

**Results:** Visual acuity remained at 20/16 from 1 day postoperatively, with little endothelial cell loss compared with preoperatively.

**Conclusion:** A spontaneously dislocated crystalline lens into the anterior chamber with severe corneal endothelial cell loss was treated successfully by intracapsular extraction.
Lens luxation into the anterior chamber is rare compared with luxation into the vitreous body. However, anterior dislocation of the crystalline lens can cause severe complications such as corneal edema and acute glaucoma. Therefore, unlike with a lens dislocated into the vitreous, an anteriorly dislocated crystalline lens should always be removed. We report a successful case of intracapsular lens extraction in a patient with a spontaneously dislocated crystalline lens into the anterior chamber where prevention of postoperative corneal endothelial cell loss was successful.

Case report

A 49-year-old Japanese woman was referred to the Department of Ophthalmology, Tokyo Dental College in July 2005 to treat complete lens dislocation into anterior chamber in the right eye. The patient had blurred vision but no pain for 2 weeks. She had a history of uncomplicated laser iridotomy (LI) for narrow angle in the right eye at the age of 48 years, at which time endothelial cell density was $2421/mm^2$ at 7 days after treatment. A general physical examination revealed no other abnormalities. The patient’s medical history and familial history were unremarkable, with no other history of injury or treatment in
the right eye, other than the above-mentioned LI. Medical records prior to the LI in the right eye were not available for review.

At her initial examination, slit lamp microscopy revealed crystalline lens dislocation into the anterior chamber and corneal touch (Figure 1). Stromal edema and Descemet’s fold were seen in the superior cornea. No zonules were seen around the dislocated lens, except at 6 o’clock, and intraocular pressure (IOP) was 13mmHg. Despite an edematous cornea and the crystalline lens into the anterior chamber, the visual acuity was correctable to 20/20. She showed a best-corrected visual acuity (BCVA) of 20/20 in the right eye and 20/16 in the left eye. Manifest refraction was -4.75-1.25×130 in the right eye and -0.50-1.00×120 in the left eye. Central endothelial cell density was 496/mm² in the right eye and 2469/mm² in the left eye, and pachymetry of the right eye was 495 μm at presentation. The left eye was unremarkable, and showed no sign of damage to Zinn’s zonule, gonioscopy of the left eye revealed no abnormalities, and pseudoexfoliation of the lens capsule was not identified in either eye.

The patient underwent intracapsular lens extraction and anterior vitrectomy on the following day. First, a corneoscleral incision was made appropriate to the lens size. The anterior chamber was then penetrated at 10
o’clock, and viscoelastic material (Healon® AMO Inc.) was injected between the cornea and the crystalline lens to protect endothelial cells. Next, the incision was penetrated, and the whole lens was extracted without difficulty. Herniated vitreous in the anterior chamber was removed with a vitreous cutter probe. The incision was closed with a shoelace suture and astigmatism was adjusted with the aid of circular LED lights attached to a surgical microscope (Nevious light®, Varitronix Ltd.).

On the first postoperative day, moderate stromal edema was noted, and IOP was 12mmHg. On the second day, BCVA reached 20/16, remaining at that value after one month. This was accompanied by a stable IOP. Specular microscopy revealed that corneal central endothelial density was 423/mm² at one month postoperatively. The stroma, however, showed no edema. The patient’s condition has remained stable, with a clear cornea and an IOP in the low teens for more than 3 months (Figure 2).

**Discussion**

The etiologies of dislocation of the crystalline lens have been reported to include trauma, heredity, and spontaneous occurrence.¹ Among these, the most
common cause of lens luxation is trauma. Hereditary forms of lens dislocation have been associated with other systemic anomalies (e.g. Marfan’s syndrome, homocystinuria, Weill-Marchesani syndrome). This case was one of spontaneous dislocation of the lens with luxation into the anterior chamber, an example of the most unusual among these etiologies. Such anterior dislocation of the lens can cause several complications such as pupillary block, which may lead to angle closure and a precipitous increase in intraocular pressure.\textsuperscript{1-3} Here, however, although the anteriorly dislocated crystalline lens was pressing down on the pupil margin, the IOP was within the normal range. It is rare for lens luxation into the anterior chamber to occur spontaneously with no increase in intraocular pressure. LI had been performed previously, so pupillary block was avoidable in this case. In anterior lens dislocations, prolonged contact of the lens with the corneal endothelium can result in permanent decompensation of the cornea. Although acute glaucoma was not seen in this case, spontaneous severe endothelial cell loss did occur; showing an endothelial cell density of 496/mm\textsuperscript{2} preoperatively.

Previous studies have recommended the immediate removal of a lens when dislocation is seen into the anterior chamber to prevent severe
Choi et al reported anterior vitrectomy and lensectomy using the closed chamber technique. This technique reduces risk of expulsive hemorrhage with sudden decrease in IOP. In our case, we chose intracapsular lens extraction to prevent both lens fragment dissemination in the vitreous cavity and further damage to the corneal endothelium by unavoidable lens rubbing. By using viscoelastic material, it is possible to protect endothelial cells during surgical invasion, resulting in minimum endothelial cell loss and maintenance of corneal clarity. In this case, we considered the risk of expulsive hemorrhage to be minimal as IOP was within the normal range. Use of this method allowed minimum surgical invasion and early recovery of BCVA. This suggests that intracapsular lens extraction is a robust approach when LI has already been performed in a patient or when there is no increase in IOP.

The low central endothelial cell counts, preoperatively and postoperatively, are consistent with imminent corneal edema. Chronic corneal endothelial cell decompensation typically occurs when the central endothelial cell density declines to 700 to 400 cells/mm$^2$. However paracentral and/or peripheral endothelial cell density may be higher than central location xx, which may mislead the exact endothelial cell count. From typical cell counts and
corneal edema onset of this patient, corneal decompensation is probably expected to develop during her lifetime, whereas annual cell loss rates following cataract surgery approach 2.5% \textsuperscript{7}. She will likely develop corneal edema due to the endothelial damage.

We report a successful case of intracapsular lens extraction in a patient with a crystalline lens that spontaneously dislocated into the anterior chamber causing severe corneal endothelial cell loss.
Figure legend

Figure 1

Photograph of a 49-year-old Japanese woman with crystalline lens dislocation into the anterior chamber and corneal touch in her right eye. Intraocular pressure (IOP) = 13 mmHg, cell density (CD) = 496/mm²

Figure 2

After intracapsular extraction, visual acuity was 20/16, the cornea was clear, and IOP was 12 mmHg. CD = 423/mm²
References

Fig. 1
Fig. 2