



Occult lens subluxation related to laser peripheral iridotomy

A case report and literature review

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Abstract

Rationale: Laser peripheral iridotomy (LPI) is commonly performed as a primary treatment for acute primary angle closure glaucoma after administration of anti-glaucoma medications or for prevention of this condition. Minor complications may occur following LPI and most of them do not have deleterious consequences. We report a rare case of lens subluxation that has a possible relationship with LPI treatment.

Patient concerns: A 54-year-old female patient was initially referred for surgical treatment of medication-uncontrollable angle closure glaucoma in her left eye. The patient had undergone Neodymium:YAG LPI at an outside hospital 2 months prior to the presentation due to an episode of elevated intraocular pressure (IOP). About 5 days after the LPI, she had spontaneous blurred vision, redness, and pain in the left eye. Her IOP was found to re-rise and was not controlled well even with maximum tolerated antiglaucoma medications during the following 2 months. On slit-lamp examination, the significant shallowing of both peripheral and central anterior chamber was noted in the left eye. Ultrasound biomicroscopy examination revealed the lens tilting towards the iris and the inferior zonular dehiscence corresponding to the iridotomy site.

Diagnoses: Lens subluxation secondary to LPI treatment in the left eye.

Interventions: Phacoemulsification combined with in-the-bag intraocular lens implantation was performed in the left eye. The zonular weakness corresponding to the iridotomy site was further confirmed during surgery.

Outcomes: The patient's IOP remained stable in the first postoperative 3 months without additional anti-glaucoma medications.

Lessons: Laser peripheral iridotomy may cause structural zonular damage, and ophthalmologists should be aware of this potential complication and proceed with caution.

Abbreviations: IOP = intraocular pressure, LPI = laser peripheral iridotomy, Nd:YAG = neodymium:YAG, UBM = ultrasound biomicroscopy.

Keywords: complication, laser peripheral iridotomy, lens subluxation, zonular weakness

1. Introduction

Laser peripheral iridotomy (LPI) has been the standard 1st-line treatment for primary angle closure and primary angle closure glaucoma in many parts of the world.^[1-3] LPI is typically

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performed to prevent or relieve pupillary block, which is a critical factor in the development of acute angle closure. Minor complications may occur following LPI such as intraocular pressure (IOP) spikes, corneal endothelial burns, anterior chamber inflammation, and hyphema.^[4] Most of them are transient and do not have deleterious consequences. A few serious complications, such as cataract progression^[5] and bullous keratopathy,^[6] have been reported in the literature. We describe a case of lens subluxation which occurred shortly after an LPI treatment for primary angle closure attack in an otherwise healthy individual.

2. Consent

This study adhered to the tenets of the Declaration of Helsinki and was approved by the ethics committee of the First Affiliated Hospital, College of Medicine, Zhejiang University. Informed consent was signed by the patient for the publication of this report and its related images.

3. Case presentation

A 54-year-old Chinese woman was referred for the surgical treatment of uncontrollable angle closure glaucoma in the left eye. She had undergone neodymium:YAG (Nd:YAG) LPI in both eyes at an outside hospital 2 months prior to the presentation due to an episode of elevated IOP in the left eye. Prior to the LPI, her uncorrected vision was 20/25 in both eyes, with IOP of 16 mm Hg in the right eye and 18 mm Hg in the left eye, respectively. About 5

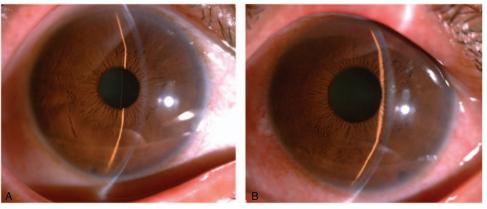


Figure 1. Slit-lamp photographs of the patient on presentation. (A) Slit-lamp photograph of the right eye shows slightly shallow anterior chamber and patent peripheral iridotomy. (B) Slit-lamp photograph of the left eye shows conjunctival hyperemia, significantly shallow anterior chamber, mildly dilated pupil, and patent peripheral iridotomy.

days after the LPI, the patient had spontaneous blurred vision, redness, and pain in her left eye. Her IOP was found to rerise in the left eye and multiple antiglaucoma medications were prescribed including Pilocarpine, Timolol, Brimonidine, Latanoprost, and Methazolamide at the outside hospital. However, her IOP was not controlled well. The patient's medical history and familial history were unremarkable, without any other history of trauma or surgical interventions in both eyes. The patient reported no history of myopia in either eye. A general physical examination did not reveal any systemic abnormalities.

On presentation, the best corrected visual acuity in the right eye was 20/25 (+0.75 +0.50 × 150) and in the left eye was 20/32 (-2.25 +0.75 × 60). IOP was 14 mmHg in the right eye and 31 mmHg in the left eye. Corneal endothelial cell density in the right eye was 2310 cells/mm^2 and in the left eye was 2560 cells/mm^2 . Axial length in the right eye was 21.36 mm and in the left eye was 21.45 mm. On slit-lamp examination, the significant shallowing

of both peripheral and central anterior chamber was noted in the left eye (Fig. 1B). The LPI was patent in the inferior quadrant in both eyes (Fig. 1A, B). Mild lens opacity with no signs of capsular pseudoexfoliation was observed in both eyes. The cup-to-disc ratio was 0.5 to 0.6 in both eyes. The optical coherence tomography scan revealed the thinning of mean retina nerve fiber layer thickness in the inferior quadrant and the standard automated perimetry (Humphrey Field Analyzer, 30-2 pattern) showed the superonasal paracentral scotoma in the left eye; no abnormality was detected in the right eye.

Ciliary block glaucoma was initially suspected. A cycloplegic provocative test was planned for diagnostic treatment with the patient's informed consent. Before the cycloplegic provocation, ultrasound biomicroscopy (UBM) examination was taken to examine the ciliary body and the lens-iris diaphragm position. On UBM examination, the significantly shallow anterior chamber, a large scale of extremely narrow angles with superior angle closure,

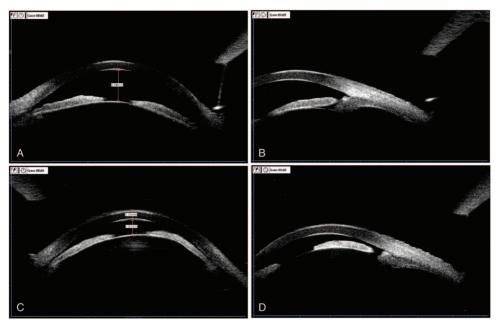


Figure 2. Ultrasound biomicroscopy (UBM) images of the patient on presentation. (A) UBM image of the right eye shows shallow anterior chamber, superior narrow angle, and superior ciliary body anteposition and forward rotation. (B) UBM image of the right eye shows the inferior open angle, iridotomy, slight ciliary body anteposition and forward rotation. (B) UBM image of the right eye shows the inferior open angle, iridotomy, slight ciliary body anteposition and forward rotation, and a few zonular fibers. (C) UBM image of the left eye shows significantly shallow anterior chamber, extremely narrow angles, and slight lens tilting toward the iris. (D) UBM image of the left eye shows the iridotomy at the 5 o'clock position, ciliary body anteposition, and forward rotation, while zonular fibers were invisible.



Figure 3. Intraoperative photograph of the left eye. An iris hook was inserted to prevent further lens zonular dehiscence at the 5 o'clock position and stabilize the capsule at the stage of cortical aspiration.

ciliary body anteposition and forward rotation, and slight lens tilting toward the iris were observed in the left eye (Fig. 2C, D). The central anterior chamber depth was 1.01 mm in the left eye, while it was 2.09 mm in the right eye. Further examination revealed the possible dehiscence of lens zonula in the inferior quadrant, which corresponded to the LPI position (Fig. 1D). After the cycloplegic provocation, IOP rose to 44 mm Hg, thus the pathogenetic factor related to ciliary block was excluded.

Phacoemulsification was then performed in the left eye. During the continuous curvilinear capsulorhexis, capsule wrinkling was noted around 5 o'clock position. At the stage of cortical aspiration, the ophthalmic viscosurgical device was refilled to maintain the anterior chamber, and a small paracentesis was made at the 5 o'clock position. An iris hook was inserted to prevent further lens zonular dehiscence and to stabilize the capsule (Fig. 3).^[7] After lens removal, a 3-piece intraocular lens was gently implanted into the capsule bag (ZA9003, Abbott Medical Optics).

On the first 2 postoperative days, IOP was 15 to 17 mmHg without any additional antiglaucoma medication. A week later, best corrected visual acuity reached 20/32 and IOP remained stable. The patient's condition remained stable in the first 3 months postoperatively.

4. Discussion and literature review

In the present case, lens subluxation occurred 5 days after the LPI. UBM examination revealed the close positional relationship between the lens zonular dehiscence and the LPI site, both of which were located in the inferior quadrant. The lens zonular dehiscence was further confirmed as the sign of corresponding inferior capsule wrinkling noted during capsulorhexis.^[8] Common etiologies of lens dislocation include a history of trauma or previous intraocular surgery, comorbidities of eye diseases associated with zonular weakness (eg, pseudoexfoliation syndrome, high myopia, retinitis pigmentosa, uveitis, endophthalmitis, and intraocular tumor), and hereditary disorders with zonular weakness (eg, microspherophakia, Marfan syndrome, homocystinuria, and Weill-Marchesani syndrome).^[9-14] Nevertheless, there was no relevant evidence concerning these etiologies for this patient other than the above-mentioned LPI. Hence, we speculate that the lens subluxation may have a possible relationship with the LPI for this case.

Nd:YAG laser delivers intense amounts of energy into a wellfocused spot in a period of picoseconds to nanoseconds^[4] and has been commonly used for LPI treatment. Although focal lens damage is one of the concerns in performing Nd:YAG LPI, major lensrelated complications are rare in clinical practice. The main reasons for the limited impact on the lens are 2-fold. First, iridotomy sites are routinely picked in the far iris periphery, even if not the farthest, to distance the lens and avoid iatrogenic damage to lens. Second, the iris itself absorbs most of the laser energy during iridotomy.

We further searched literatures reporting lens dislocation with a history of LPI. Melamed et al^[9] first reported a case of further lens inferior dislocation following a supratemporal LPI treatment that was initially caused by trauma. Kwon et al^[12] reported bilateral complete lens dislocation occurring (8 months in the right eye and 2 years in the left eye) after LPI in a patient with retinitis pigmentosa and phacodonesis. Seong et al^[11] reported complete lens dislocation 10 months after LPI in a high myopia eye. In these cases, the predisposed conditions may have already weakened the zonular fibers: the shock-wave effect from LPI was considered to bring further zonular damage and result in lens dislocation. Nevertheless, a few cases of spontaneous lens dislocation without any relevant predisposed condition besides a history of LPI treatment were also reported (1 eve with posterior dislocation and 4 eyes with complete dislocation; intervals ranging from 1 month to 1 year),^[15,16] as in our case. Except for the traumatic lens dislocation reported by Melamed et al,^[9] clear evidence of causality between the LPI and lens dislocation is inadequate in these cases.

Our case is unique as the close sequential and positional relationships between the LPI and the lens zonular dehiscence provide clear etiological evidence. In the present case, 2 likely reasons may contribute to the unexpected structural lens zonular damage with LPI treatment in a synergistic manner. First, the ciliary body anteposition and forward rotation, as observed on the UBM examination, may result in the uncommonly short distance between lens zonula and iris, which might be unsafe for LPI shots. As reported by Melamed et al,^[9] LPI may further damage the zonular fibers of the lens that were already tilted forward toward the iris due to trauma. Second, the episode of pupillary block and angle closure may result in zonular laxity due to the weakened iris and ciliary body, as suggested by previous authors.^[11,17] In addition, laser retrofocus setting is commonly selected for the sake of perforation efficiency during iridotomy; however, it may increase the risk of throughput of laser energy, especially in the above-mentioned circumstances.

In the present case, the medication-uncontrollable IOP elevation was likely caused by mixed reasons. First, the large scale of appositional angle closure resulting from the pressure of posterior chamber (the extremely narrow angles with superior angle closure observed on the supine UBM examination) may have led to the IOP elevation. The significant pupillary block caused by the lens subluxation and crowding posterior chamber may be the pathogenetic factor. The further elevation of IOP after the cycloplegic provocation test was possibly due to the aggravated appositional angle closure. Second, the possible inflammation of aqueous outflow pathway due to the sustained IOP elevation may have also contributed to the uncontrollable IOP elevation. Therefore, we performed the phacoemulsification as the 1st surgical step for this patient to tackle the lens-associated factor. A further glaucoma surgery might be needed if irreversible peripheral angle synechiae had been formed. An alternative surgical option might be combined phacoemulsification and trabeculectomy. The extreme narrowing of angle, however, not completely closure, was observed on UBM examination, hence we did not perform the 1stage antiglaucoma surgery. In the present case, a careless diagnosis missing the lens subluxation may result in surgical overtreatment and unexpected trouble during surgery.

5. Conclusions

We report a rare case of lens subluxation that has close sequential and positional relationships with LPI. Future studies would be worthwhile to explore if LPI is an independent risk factor for lens zonular dehiscence. Care should be taken in performing LPI in eyes with crowding posterior chamber, especially those with predisposed conditions associated with zonular weakness, and a careful examination of posterior chamber structure before LPI would be helpful. Ophthalmologists should be aware of this potential complication and appropriately counsel their patients.

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