Research Square

Preprints are preliminary reports that have not undergone peer review.
They should not be considered conclusive, used to inform clinical practice,
or referenced by the media as validated information.

Spontaneous posterior capsular dehiscence during long-term follow-up of sporadic posterior lenticonus: a case report

Yan Zhang
Keio University School of Medicine
Yohei Tomita
y.tomita@keio.jp

Keio University School of Medicine
Tomonori Yuda
Keio University School of Medicine
Hidemasa Torii
Keio University School of Medicine
Kazuno Negishi
Keio University School of Medicine

Case Report

Keywords: Posterior lenticonus, Posterior capsular dehiscence, Anterior segment OCT, Hyperopic shift, Cataract surgery, Pars plana vitrectomy

Posted Date: October 6th, 2026

DOI: https://doi.org/10.21203/rs.3.rs-10747749/v1

License: This work is licensed under a Creative Commons Attribution 4.0 International License.
Read Full License

Additional Declarations: No competing interests reported.

Abstract

Background

Posterior lenticonus is a rare congenital lens anomaly characterized by localized conical protrusion of the posterior lens capsule and cortex. It is most commonly reported in children, and visual outcomes may be limited by amblyopia. Adult sporadic cases are uncommon, and longitudinal documentation of posterior capsular dehiscence before surgery has rarely been emphasized. We report a young adult with sporadic posterior lenticonus in whom serial anterior segment optical coherence tomography (AS-OCT) documented spontaneous posterior capsular dehiscence during long-term follow-up and guided surgical timing and planning.

Case presentation

A 25-year-old man had been diagnosed with posterior lenticonus of the right eye at age 3 at another hospital. He first visited our institution at age 13 with preserved visual function and re-presented at age 19. Although AS-OCT was performed at age 19, retrospective review could not definitively establish posterior capsular dehiscence. He subsequently underwent regular clinical follow-up, and AS-OCT first clearly demonstrated posterior capsular dehiscence at age 23. During subsequent follow-up, visual-axis opacity progressed and best-corrected visual acuity (BCVA)declined to 20/133, accompanied by a marked hyperopic shift from approximately + 5.50 D at age 13 to + 16.00 D at age 25. Preoperative AS-OCT demonstrated persistent posterior capsular dehiscence with cortical material prolapsing into the vitreous cavity. Planned cataract surgery combined with 27-gauge pars plana vitrectomy was performed, followed by in-the-bag implantation of a three-piece intraocular lens. Postoperatively, BCVA improved to 20/16 and remained stable through 8 months.

Conclusions

Serial AS-OCT may be valuable for detecting and monitoring posterior capsular dehiscence in posterior lenticonus, particularly when slit-lamp findings are insufficient. Longitudinal imaging can help anticipate capsular complications, individualize surgical timing, and plan combined anterior–posterior segment management.

Background

Posterior lenticonus is a rare congenital lens anomaly characterized by localized conical protrusion of the posterior lens capsule and cortex. It is usually detected in childhood because of unilateral cataract, anisometropia, irregular astigmatism, or amblyopia[1]. In a recent pediatric series, posterior lenticonus was associated with characteristic refractive abnormalities, including high lenticular myopia at the conical region and irregular astigmatism, and postoperative visual recovery could be limited by

amblyopia [1]. Surgical management may be challenging because posterior capsular thinning, weakness, or a pre-existing defect can increase the risk of lens material drop and vitreous traction.

Most reported cases focus on pediatric presentations, distinctive morphology, or intraoperative findings. Adult cases have been described, but many are associated with systemic disease, particularly Alport syndrome[2, 3], or are reported as single-time-point observations. Other reports have described associations with persistent fetal vasculature, atypical peripheral posterior lenticonus, and clinical signs such as the atoll sign or jellyfish sign [4–7]. However, longitudinal documentation of spontaneous posterior capsular dehiscence in a sporadic adult case before surgery remains uncommon.

We report a young adult with sporadic posterior lenticonus in whom AS-OCT clearly documented posterior capsular dehiscence at age 23, 2 years before surgical intervention. This case highlights the role of serial AS-OCT in detecting capsular pathology not apparent on slit-lamp examination alone, monitoring structural progression, and guiding the timing and strategy of surgery.

Case presentation

A 25-year-old man had been diagnosed with posterior lenticonus of the right eye at age 3 at another hospital. He first visited our institution at age 13 with preserved visual function and no subjective visual decline. Because visual function was stable, he subsequently discontinued follow-up.

He re-presented at age 19. Slit-lamp examination demonstrated posterior lenticonus with central lens opacity. Although AS-OCT was performed, retrospective review of the image could not definitively establish the presence of posterior capsular dehiscence. BCVA was 20/40. Because his visual function remained acceptable and he had recently completed driver's license renewal, surgery was deferred.

The patient subsequently attended annual follow-up examinations, except at age 22. AS-OCT images adequately documenting the posterior capsule were not available during this interval. At age 23, AS-OCT clearly demonstrated posterior capsular dehiscence with cortical material protruding toward the vitreous cavity (Fig. 1). The capsular defect remained evident on subsequent AS-OCT examinations. During follow-up, visual-axis opacity progressed and autorefractometry demonstrated a marked hyperopic shift from approximately + 5.50 D at age 13 to + 16.00 D at age 25. At age 25, the patient reported increased visual difficulty and required driver's license renewal. BCVA had decreased to 20/133. Preoperative AS-OCT demonstrated persistent posterior capsular dehiscence with cortical material prolapsing into the vitreous cavity and visually significant opacity along the visual axis (Fig. 1).

There was no history of ocular trauma, hearing impairment, renal disease, or family history suggestive of Alport syndrome. Preoperative blood testing showed normal renal function, with serum creatinine of 0.89 mg/dL and estimated glomerular filtration rate of 89 mL/min/1.73 m². Ocular biometry showed axial length of 22.89 mm in the right eye and 23.89 mm in the left eye, and keratometry of K1 44.24 D at 179° ; K2 48.24 D at 89° in the right eye and K1 44.35 D at 170° ; K2 47.01 D at 80° in the left eye, measured using a swept-source OCT biometer (ARGOS, Alcon Laboratories, Fort Worth, TX, USA). The

left eye had a BCVA of 20/16 with a refraction of -1.75 -1.75 × 169° (TONOREF III; NIDEK Co., Ltd., Gamagori, Japan).

Given the high risk of lens material drop and vitreous traction, planned cataract surgery combined with pars plana vitrectomy was performed. A continuous curvilinear capsulorhexis was created, followed by phacoemulsification and aspiration. Because inspection of the fundus revealed cortical material within the vitreous cavity, a 27-gauge pars plana vitrectomy (PPV) was performed to remove the dropped cortical material and relieve vitreous traction. No tamponade was used. Because adequate peripheral capsular support was preserved despite the central posterior capsular defect, a three-piece intraocular lens (+ 24.0 D, NX70s, Santen Pharmaceutical Co., Japan) was implanted in the capsular bag. IOL power was calculated using the Barrett True AL formula, targeting − 2.0 D to minimize anisometropia with the fellow eye.

Postoperatively, BCVA improved to 20/16 with a manifest refraction of + 0.25–3.50 × 5° (TONOREF III; NIDEK Co., Ltd., Gamagori, Japan) and remained stable through 8 months of follow-up. Slit-lamp examination and AS-OCT demonstrated a clear visual axis and stable capsular–IOL complex without recurrent cortical prolapse or IOL instability (Fig. 2).

Discussion and Conclusions

This case provides several clinically relevant observations. First, posterior capsular dehiscence in posterior lenticonus may not be evident on slit-lamp examination alone. Although AS-OCT was performed at age 19, retrospective review could not definitively establish the presence of posterior capsular dehiscence at that time. The patient continued regular clinical follow-up thereafter, but AS-OCT images adequately documenting the posterior capsule were unavailable until age 23, when dehiscence was first clearly demonstrated. Therefore, the exact timing of spontaneous posterior capsular dehiscence cannot be determined. Subsequent serial AS-OCT enabled longitudinal visualization of the capsular defect and its relationship with progressive lens opacity, refractive change, and visual deterioration.

The decision to proceed with surgery in posterior lenticonus should be individualized according to visual function, structural progression, and patient-specific functional needs, as discussed in previous case-based literature [8]. At age 19, surgery was deferred because BCVA remained 20/40 and the patient had recently completed driver's license renewal. After posterior capsular dehiscence was clearly documented at age 23, continued observation was selected while functional vision remained acceptable. Progressive visual-axis opacity, BCVA decline to 20/133, and renewed functional requirements related to driver's license renewal eventually supported surgical intervention at age 25.

Second, the case adds information to the limited literature on sporadic adult posterior lenticonus. Posterior lenticonus is predominantly reported in children [1]. Adult cases have been described in syndromic settings, particularly Alport syndrome [2, 3]. In contrast, our patient had no history of renal disease, hearing impairment, or family history suggestive of Alport syndrome, and preoperative renal

function was normal, supporting a sporadic presentation. Unlike many pediatric cases in which amblyopia can limit postoperative recovery, this patient underwent surgery in young adulthood without evident amblyopia, which may have contributed to the excellent postoperative BCVA of 20/16.

Third, the marked hyperopic shift in this case may provide a clinical clue to progressive lens pathology. Posterior lenticonus is often associated with localized lenticular myopia and irregular astigmatism [1]. However, hyperopic refractive change may occur when posterior capsular disruption is accompanied by changes in lens contents, such as liquefaction, redistribution, or extrusion, resulting in reduced effective lenticular power. Progressive hyperopic shift after posterior capsule tear has been reported following blunt ocular trauma [9]. In our patient, serial autorefractometry demonstrated a marked hyperopic shift from approximately + 5.50 D at age 13 to + 16.00 D at age 25. Axial length was 22.89 mm in the affected eye, supporting a predominantly lenticular rather than axial origin of the hyperopic shift. Given the magnitude of the refractive change, together with progressive visual-axis opacity and AS-OCT–documented posterior capsular dehiscence, these findings suggest a clinically meaningful reduction in effective lenticular power.

The mechanism of spontaneous posterior capsular dehiscence in posterior lenticonus remains uncertain. A plausible explanation is congenital focal posterior capsular weakness combined with cumulative mechanical stress over time. Similar spontaneous posterior capsule rupture has been described in posterior polar cataract, supporting the concept that capsular fragility plus mechanical stress can result in rupture even without overt trauma [8]. In posterior lenticonus, progressive deformation of the posterior capsule and changes in lens volume or lens contents may further increase local stress at the conical lesion.

Preoperative recognition of posterior capsular dehiscence is actionable. In this case, AS-OCT findings allowed us to anticipate lens material drop and plan combined cataract surgery with pars plana vitrectomy. Intraoperatively, cortical material was confirmed within the vitreous cavity as anticipated, and 27-gauge vitrectomy allowed safe removal of dropped material and vitreous management. The excellent postoperative outcome supports the value of careful preoperative imaging and surgical planning.

This report has limitations. It describes a single patient, and the exact timing of spontaneous posterior capsular dehiscence cannot be determined because the finding could not be definitively established on the age-19 AS-OCT image and adequate AS-OCT documentation of the posterior capsule was unavailable until age 23. Nevertheless, subsequent serial AS-OCT demonstrated persistent capsular dehiscence and enabled longitudinal assessment of its association with refractive and visual changes.

In conclusion, spontaneous posterior capsular dehiscence may occur during the long-term course of sporadic posterior lenticonus, although its exact timing may be difficult to determine. Serial AS-OCT is valuable for detecting posterior capsular pathology not clearly visible on slit-lamp examination and for monitoring subsequent structural changes. In this case, preoperative identification of the capsular defect informed surgical planning and was associated with a favorable postoperative outcome.

Abbreviations

AS-OCT
anterior segment optical coherence tomography
BCVA
best-corrected visual acuity
IOL
intraocular lens
PPV
pars plana vitrectomy

Declarations

Ethics approval and consent to participate

Ethics approval was waived by Ethics Committee of Keio University Hospital because this study is a single case report. The study adhered to the tenets of the Declaration of Helsinki.

Consent for publication

Written informed consent was obtained from the patient for publication of the de-identified clinical details and accompanying images. No personally identifiable information or identifying images are included in this case report.

Competing interests

The authors declare that they have no competing interests.

Authors' information

Not applicable.

Funding

This study received support from Grants-in-Aid for Scientific Research (KAKENHI) from the Ministry of Education, Culture, Sports, Science and Technology to Dr Tomita (23K15938, 25K12855).

Author Contribution

YZ and YT conceived the report and drafted the manuscript. YZ, YT, TY, HT and KN acquired, analysed and interpreted the clinical data. TY, HT and KN critically revised the manuscript. KN supervised the work. All authors read and approved the final manuscript.

Acknowledgements

Not applicable.

Data Availability

All data generated or analyzed during this study are included in this published article. Additional clinical data are available from the corresponding author on reasonable request, subject to patient privacy protection.

References

1. Kekunnaya R, Deshmukh AV, Kulkarni S. Newer insights into the clinical profile of posterior lenticonus in children and its surgical, visual, refractive outcomes. Eye (Lond). 2022;36(5):985–93. https://doi.org/10.1038/s41433-021-01564-4.
2. Halawani LM, Abdulaal MF, Alotaibi HA, Alsaati AF, Bin Dakhil TA. Development of Posterior Lenticonus Following the Diagnosis of Isolated Anterior Lenticonus in Alport Syndrome. Cureus. 2021;13(1):e12970. https://doi.org/10.7759/cureus.12970.
3. Al-Mahmood A, Al-Swailem S, Al-Khalaf A, Al-Binali G. Progressive posterior lenticonus in a patient with Alport syndrome. Middle East Afr J Ophthalmol. 2010;17:379–81. https://doi.org/10.4103/0974-9233.71591.
4. Khokhar S, Dhull C, Mahalingam K, Agarwal P. Posterior lenticonus with persistent fetal vasculature. Indian J Ophthalmol. 2018;66(9):1335–6. https://doi.org/10.4103/ijo.IJO_276_18.
5. Ranjan P, Mishra D, Bhadauria M. Atoll sign in posterior lenticonus: A case report of bilateral posterior lenticonus with review of literature. J Clin Ophthalmol Res. 2014;2(3):152. https://doi.org/10.4103/2320-3897.138861.
6. Ganesh S, Brar S, Chopra K. Jellyfish sign for intraoperative identification of posterior lenticonus. Int Ophthalmol. 2017;37(5):1239–41. https://doi.org/10.1007/s10792-016-0386-1.
7. Agarwal R, Jain V, Kaur M, Titiyal JS. Atypical presentation of peripheral posterior lenticonus and role of multi-modal ocular imaging in its diagnosis and management. BMJ Case Rep. 2021;14(2):e238801. https://doi.org/10.1136/bcr-2020-238801.
8. Lu MZ, Cao WL, Xing YQ. Contemplating the necessity of surgical treatment for posterior lenticonus: a case report. BMC Ophthalmol. 2023;23(1):283. https://doi.org/10.1186/s12886-023-03042-9.

9. Shibata T, Seki Y, Seida Y, Yoshita T, Tsuchiya S, Sasaki H, et al. Progressive hyperopic refractive changes after posterior capsule tear following blunt ocular trauma. Am J Ophthalmol Case Rep. 2024;34:102032. https://doi.org/10.1016/j.ajoc.2024.102032.

Figures

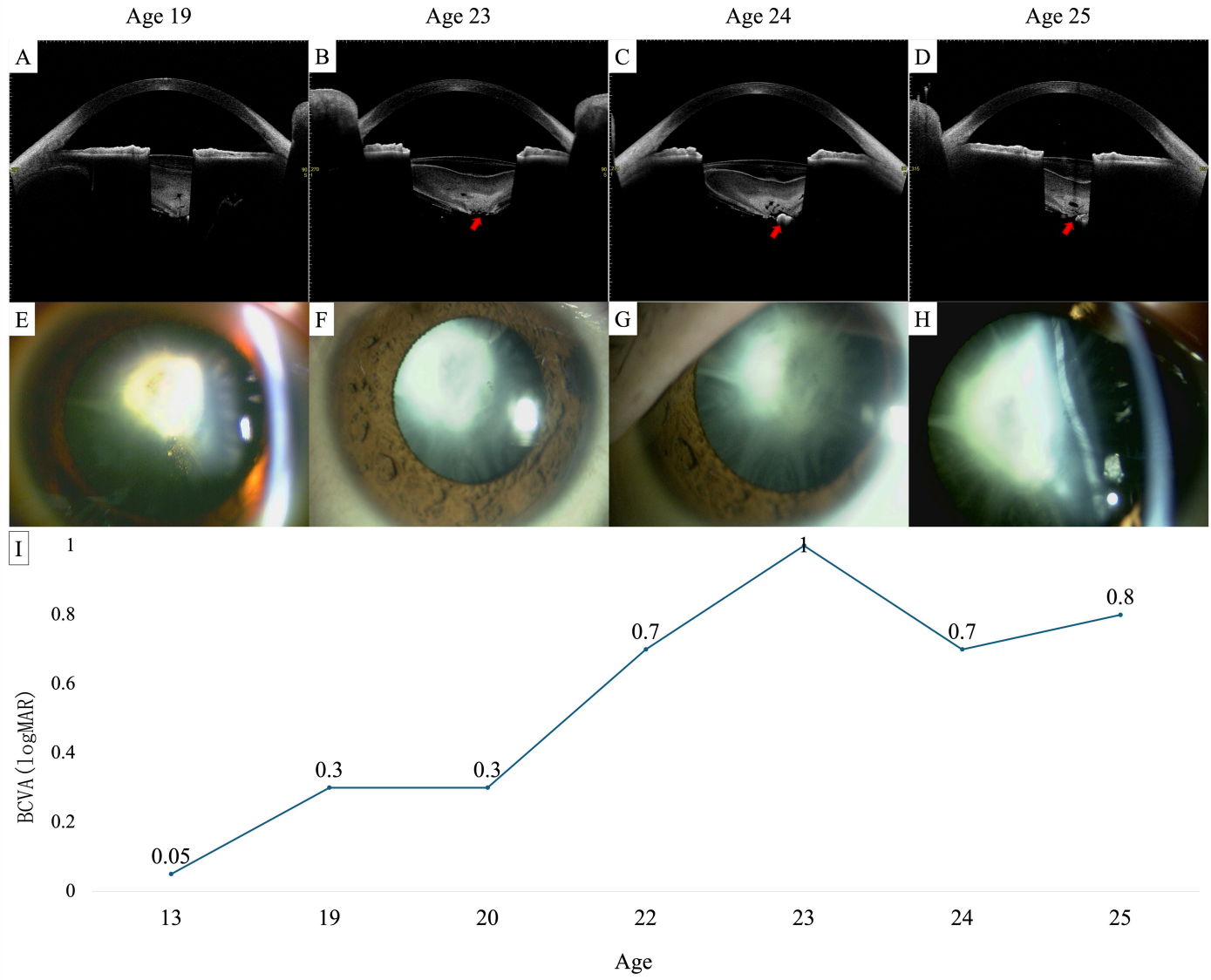


Figure 1

Longitudinal preoperative course of posterior lenticonus with spontaneous posterior capsular dehiscence

Top row (A–D), anterior segment optical coherence tomography (AS-OCT); bottom row (E–H), slit-lamp photographs. A and E, age 19 years; B and F, age 23 years; C and G, age 24 years; D and H, age 25 years. At age 19, posterior lenticonus was present, but posterior capsular dehiscence could not be definitively identified on AS-OCT (A). At age 23, posterior capsular dehiscence was clearly delineated for the first time (B, red arrow) and remained visible on subsequent AS-OCT examinations (C and D, red arrows), with

progressive cortical prolapse and visual-axis opacity. I, Longitudinal BCVA showing an overall decline with fluctuation during follow-up.

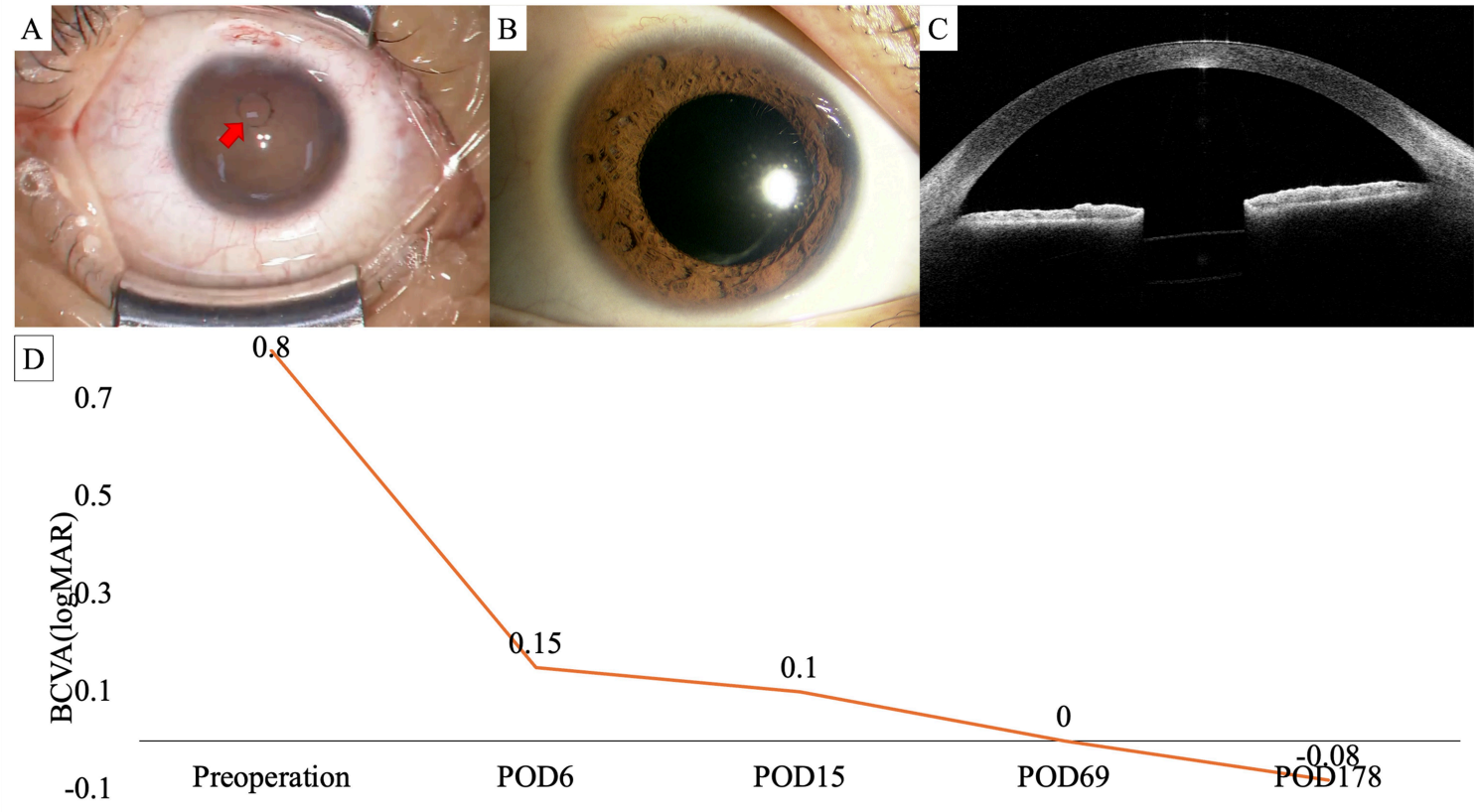


Figure 2

Intraoperative findings and postoperative visual recovery after planned phaco-vitrectomy

Intraoperative photograph showing the cataractous lens with a pre-existing posterior capsular defect (red arrow) noted during surgery (A). Slit-lamp photograph at 8 months postoperatively demonstrating a clear visual axis and stable anterior segment appearance (B). Anterior segment optical coherence tomography at 8 months postoperatively showing postoperative anterior segment configuration with a stable capsular–intraocular lens complex and no evidence of recurrent cortical prolapse (C). Postoperative BCVA course showing improvement from 20/133 preoperatively to 20/16 after surgery, with stable visual recovery through 8 months of follow-up (D).