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## Correspondence

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### Long-standing Asymptomatic Intralenticular Foreign Body

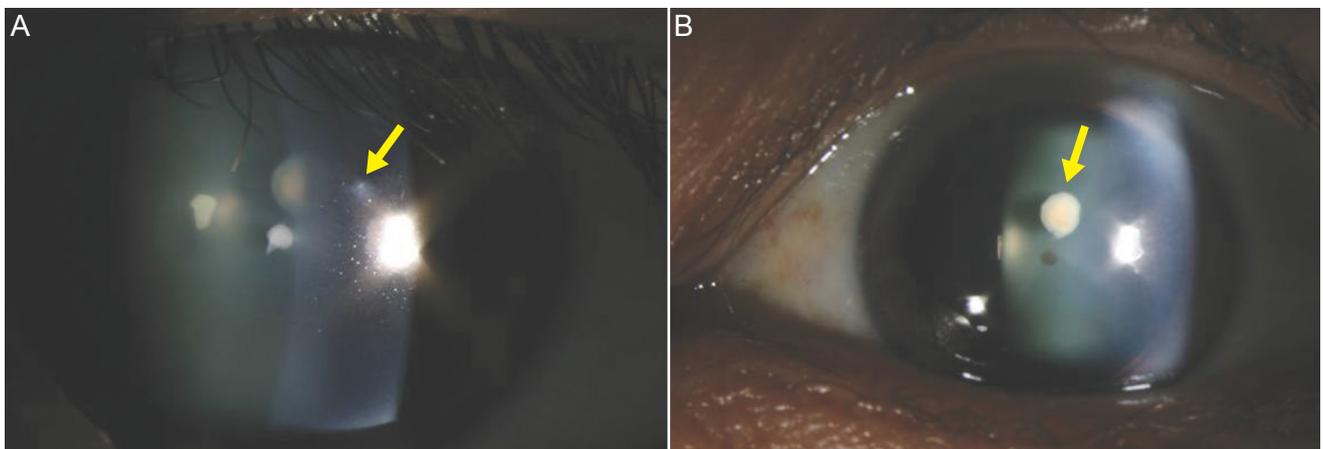
Dear Editor,

Intralenticular foreign bodies are uncommon after penetrating eye injuries, occurring in approximately 5% to 10% of cases [1]. They generally present with a decrease in visual acuity resulting from aggravation of a traumatic cataract. In Korea, there has been only one previous report of an intralenticular foreign body, which was retained for 6 months [2]. Several previous reports documented the occurrence of intralenticular foreign body worldwide, but only two cases described asymptomatic intralenticular foreign body [3,4]. We recently experienced a case of asymptomatic intralenticular foreign body retained for 30 years, and report the case herein.

A 43-year-old man presented to our institution with discomfort in his left eye. He lost his right eye in a landmine blast in 1983. On examination, visual acuity was 20 / 20

and intraocular pressure was 9 mmHg in the remaining eye. Corneal opacity without involvement of the visual axis was observed, but there was no inflammation in the cornea or anterior chamber. After pupillary dilatation, slit-lamp examination revealed an intact posterior lens capsule and an intralenticular foreign body in the form of a round, yellow-whitish lesion (Fig. 1). We assumed that the foreign body was metallic in nature. It was encapsulated by a membrane and had an estimated diameter of approximately 1.2 mm. Fundus examination revealed no abnormalities in the left eye. Since visual acuity was not significantly affected, close follow-up observation was the chosen course of management.

In small anterior lens capsule defects, epithelial proliferation rapidly restores its continuity, limiting the free passage of ions and fluid that may result in progressive cataract formation [5]. In our case, the size of the intralenticular foreign body was 1.2 mm and the capsular break was small enough to heal spontaneously. We believe that the foreign body remained stable because of encapsulation, although there was no pathologic confirmation of this. The visual axis was not involved. We believe that the reasons stated above explain why the patient did not experience any visu-



**Fig. 1.** Slit-lamp photography shows (A) corneal opacity (arrow) and (B) an intralenticular foreign body (arrow).

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al disturbance for 30 years despite the presence of an intralenticular foreign body.

In conclusion, we report a case of long-standing asymptomatic intralenticular foreign body. Conservative management of intralenticular foreign body is an acceptable course unless ocular complications, such as intraocular inflammation and cataract formation, occur.

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### Conflict of Interest

No potential conflict of interest relevant to this article was reported.

### References

1. Arora R, Sanga L, Kumar M, Taneja M. Intralenticular foreign bodies: report of eight cases and review of management. *Indian J Ophthalmol* 2000;48:119-22.
2. Chang YS, Jeong YC, Ko BY. A case of an asymptomatic intralenticular foreign body. *Korean J Ophthalmol* 2008;22:272-5.
3. Richards WW, Arrington JM. Asymptomatic intralenticular glass fragment. *Am J Ophthalmol* 1969;67:375-7.
4. Shiraishi S, Ueyama A, Okazaki T, et al. A case of intralenticular foreign iron body without any symptoms for 20 years. *Nihon Ganka Gakkai Zasshi* 2008;112:882-6.
5. Fagerholm PP, Philipson BT. Human traumatic cataract: a quantitative microradiographic and electron microscopic study. *Acta Ophthalmol (Copenh)* 1979;57:20-32.

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## Treatment of Serous Retinal Detachment Associated with Choroidal Ischemia with Intravitreal Bevacizumab Following Brain Surgery

Dear Editor,

We report a unique case of visual recovery where intravitreal bevacizumab injection (IVB) completely resolved serous retinal detachment (SRD) secondary to posterior ciliary artery (PCA) occlusion after brain surgery.

A 20-year-old male was referred to our clinic complaining of a sudden decrease in visual acuity in his left eye following neurosurgery. The patient had no other medical history except for meningioma in the left sphenoid ridge. According to the neurosurgeon, the removal of meningioma was uneventful and there were no complications during the entire surgical procedure. However, during the surgery, his scalp was compressed against the surgical bed for eight hours. His best-corrected visual acuity (BCVA) was 20 /

20 in the right eye and counting fingers at 30 cm in the left eye. Light reflex was intact and there was no relative afferent pupillary defect in either eye. Fundus examination revealed multiple patchy whitenings of the outer retina across the fundus. SRD in the distribution of a cilioretinal artery (CA) was noted (Fig. 1A and 1B). The right eye showed a completely normal fundus. Fluorescein angiography (FA) revealed delayed filling of the choroidal watershed zone and CA which persisted throughout the early phase, simultaneously with normal filling of arterial branches from the central retinal artery (Fig. 1C). Indocyanine green angiography (ICGA) revealed multiple patchy hypofluorescence and non-perfusion areas (Fig. 1D). The patient was observed during the following week, but because there were no signs of improvement in the degree of SRD (Fig. 1E and 1F), 1.25 mg of IVB was administered after patient consent was given despite the risk of aggravating the already ischemic retina. Three days later, his BCVA improved to 10 / 20 and SRD was markedly improved (Fig. 1G). Ten days later, BCVA improved to 20 / 20 and FA revealed improvement of leakage, but areas of hypofluorescence persisted (Fig. 1H). ICGA revealed that multiple patchy choroidal filling defects remained (Fig. 1I