Pigmentary glaucoma due to Soemmerring ring: A histopathologic study

Zia S. Pradhan, FRCOphth, John T. Lonsdale, PhD, Federico Gonzalez-Fernandez, MD, PhD

We report a case of secondary pigmentary glaucoma due to Soemmerring rings in a patient with retinitis pigmentosa and describe the histopathologic findings. Unlike other cases of pseudo-phakic pigmentary glaucoma that are usually due to malpositioned intraocular lenses (IOLs) or bent haptics, this case had a well-positioned IOL in the capsular bag. The prominent Soemmerring ring appears to be the cause of the pigment release. Although Soemmerring rings have been associated with angle-closure glaucoma, they have not previously been shown to cause any form of open-angle glaucoma.

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CASE REPORT

A histopathologic study was performed of both eyes of a 76-year-old Caucasian man who died of respiratory failure secondary to myocardial infarction. The ophthalmic history consisted of bilateral cataract surgery with intraocular lens (IOL) implantation, age-related macular degeneration, and pigmentary retinal dystrophy. Consent for procurement of the globes was obtained by the National Research Disease Interchange and the Lions Eye Bank. The research adhered to the tenets of the Declaration of Helsinki.

The main gross pathologic findings are shown in Figure 1. The anteroposterior diameter of each eye was 23.0 mm. In Figure 1, A, the opened eye was tilted back to view the ciliary body ring from the vitreous cavity. The anterior segment showed a 3-piece IOL in the bag with a dense Soemmerring ring. The anteroposterior thickness of the ring varied and was greater than 2.2 mm in several locations. On close inspection of the angle structures, the trabecular meshwork appeared pigmented (Figure 1, C, white arrow).

The main histologic findings in the anterior structures are shown in Figure 2. At low magnification (A), the angle was open with no evidence of synchiae. Higher magnification (B) showed extensive deposition of melanin pigment along the trabecular meshwork beams. Melanin pigment...
was also internalized by the corneal endothelium and was clearly visible in the cytoplasm of those cells (C). The apparent adhesions between the iris-pigmented epithelium and the lens capsule of Soemmerring ring (D) may be a processing artifact; however, it points to the close relative positioning of these structures. Focal areas showing evidence of iris-pigmented epithelium denudation were noted (not shown). The Soemmerring ring demonstrated scattered collections of melanin (D, E). Prussian-blue staining did not show hemosiderin in the trabecular meshwork, Soemmerring ring, or any of the above locations. These findings suggest that dispersion of melanin from the iris pigment epithelium may have been the result of mechanical rubbing between the expanding Soemmerring ring and the iris.

Representative Bielschowsky silver stains of optic nerve cross-sections are shown in Figure 3. The spatial distribution of the degeneration in both the right and left optic nerve followed a sectorial or hourglass pattern of axonal loss (curved dashed lines in A and B). The transition was often sharp between degenerated areas and those showing viable nerve bundles with discernible axons (C).

Macroscopic examination of the posterior segment in both eyes showed disc pallor, attenuated blood vessels, and irregularly stellate black-pigmented material scattered throughout the fundus with relative sparing of the macula consistent with retinitis pigmentosa. Light microscopy revealed diffuse retinal degeneration, particularly of the outer nuclear layer, and migration of pigment from the retinal pigment epithelium to the inner retinal layers. Focal nodular drusen was associated with Bruch membrane.

**DISCUSSION**

Primary OAG is known to be associated with retinitis pigmentosa. In contrast, pigmentary glaucoma in retinitis pigmentosa is rare, with only one case reported in a patient with Usher syndrome. Our histopathology report describes a case of retinitis pigmentosa with pseudophakia and pigment dispersion in which anteriorly protruding Soemmerring ring cataracts were noted to correspond to areas of pigment loss on the posterior iris. Additionally, the anteroposterior thickness of the Soemmerring ring was approximately the same as, and in some areas greater than, the thickness of the natural peripheral lens. Therefore, the cause was possibly secondary pigmentary glaucoma in which enlargement of the Soemmerring rings contributed to the mechanism of pigment dispersion. Additional clinical information
would have been useful to confirm the chronology of events but was unavailable.

Pseudophakic pigmentary glaucoma is often caused by malpositioned IOLs in the ciliary sulcus or bent IOL haptics in the bag. This report describes pigment dispersion secondary to a prominent Soemmering ring with the IOL inside the capsular bag. There is one other case of pigment dispersion thought to be secondary to opacification. However, that case differs from ours as the authors hypothesized that the proliferating LECs resulted in Elschnig pearls that displaced the optic edge out of the bag, leading to mechanical rubbing of the IOL against the iris, which was diagnosed using anterior segment imaging.

The optic nerve in both eyes in our case showed an hourglass pattern of axonal loss characteristic of glaucomatous degeneration. The diagnosis of glaucoma in our patient may have been missed on clinical examination because of the severe retinal dystrophy. Furthermore, the intraocular pressure (IOP) may not have been high when recorded in the clinic since it is known to fluctuate in pigmentary glaucoma. If this condition were identified ante mortem and the IOP were high, it would warrant explanation of the IOL with the surrounding Soemmerring ring and capsular bag to prevent further pigment dispersion.

To our knowledge, this is the first report of secondary pigmentary dispersion with glaucoma as a complication of Soemmerring ring formation. Although opacifications have been associated with ACG ante mortem, they have not previously been shown to cause any form of OAG.

**Figure 2.** Histopathologic findings (hematoxylin & eosin stain). A: Low-magnification overview of the angle structures. B: Higher magnification of the trabecular meshwork showing extensive melanin pigment deposition. C: Posterior cornea showing pigment within the corneal endothelial cytoplasm (arrows). D: Low magnification showing points of apposition (arrows) between the iris and Soemmerring ring (asterisk). E: Higher magnification showing melanin pigment deposited in the Soemmerring ring (arrows) (original magnification ×100 [A, D], ×400 [B], ×630 [C, E]). A Prussian-blue histochemical stain for hemosiderin was negative.
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First author:
Zia S. Pradhan, FRCOphth
SUNY Eye Institute and Department of Ophthalmology (Ross Eye Institute), Pathology & Anatomic Sciences, the State University of New York, Buffalo, New York, USA

Figure 3. Bielschowsky silver stain of optic nerve cross-sections (A, right optic nerve; B and C, left optic nerves). Note the regional hourglass pattern of degeneration (asterisks). Higher magnification shows contrasting areas of axonal loss (asterisk) with nerve bundles still containing axons (arrows) (original magnification ×100 [A, B] and ×630 [C]).