Published in final edited form as:

J Cataract Refract Surg. 2010 March ; 36(3): 512–516. doi:10.1016/j.jcrs.2009.08.040.

Long-term pathological follow-up of obsolete design: Pannu universal intraocular lens

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Abstract

We studied an enucleated postmortem eye from an 82-year-old white donor who had been implanted with a Pannu “universal” intraocular lens (IOL) in the anterior chamber approximately 20 years earlier. This IOL has design features characteristic of a 1-piece, C-loop posterior chamber IOL. Magnetic resonance imaging showed a relatively well-centered IOL in the anterior chamber with haptics impinging on the iris. Gross and light microscopic analyses of the eye and the IOL showed peripheral anterior synechiae enclaving one haptic, areas of angle widening, significant attenuation of the corneal endothelium, multiple areas of iris trauma secondary to optic and haptic iris abrasion, large areas of pigment dispersion in the angle, diffuse pigment accumulation within the anterior chamber, and attenuation of the ganglion cell layer. The histopathological findings were consistent with glaucoma and chronic inflammation.

In the early 1980s, the concept of a “universal” intraocular lens (IOL) design appeared. These IOLs were designed to be fixated in the anterior or posterior chamber. The haptics of the universal IOL would accommodate to the size of the anterior chamber, capsular bag, or ciliary sulcus, keeping the IOL centered. The universal IOL could theoretically markedly decrease the overhead of surgical centers by allowing them to stock a single IOL design. One such IOL was invented by Jaswant Pannu.¹

The Pannu IOL consists of a 1-piece poly(methyl methacrylate) (PMMA) design with an optic measuring 6.0 mm. Two haptics radiate tangentially from the optic in a C shape, similar to posterior chamber looped IOLs. The haptics terminate in an enclosed ring structure measuring 2.5 mm in diameter; the overall IOL diameter is 13.0 mm. The IOL design also incorporates 2 positioning holes in the optic. Because of the C-shaped flexible PMMA haptics, the IOL was reported to “bend” to fit chamber sizes from 11.0 mm to 13.0 mm with minimal vaulting. Also, the terminal haptic rings facilitated ciliary, iris, or scleral suturing when the IOL was implanted in the anterior chamber or the ciliary sulcus or resided within a dislocated or a phacodonectic capsular bag.^{2,3}

The Pannu IOL was removed from the market in the late 1980s because of adverse outcomes related to IOL implantation and design. We recently had the opportunity to perform pathological analyses of an enucleated eye obtained postmortem that was implanted with this IOL in the anterior chamber. To our knowledge, this is the only report describing the

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Financial Disclosure: No author has a financial or proprietary interest in any material or method mentioned.

Design details of the magnetic resonance imaging instrumentation used are proprietary to MRI Research Inc.

long-term pathological features of a Pannu IOL in the human eye. The findings are similar to those from cases of implantation of a posterior chamber IOL (PC IOL) in the anterior chamber.

CASE REPORT

The pseudophakic (left) postmortem eye of an 82-year-old white man was enucleated and donated to the MRI Research Laboratory for research purposes in 2007. The IOL model/manufacturer and IOL implantation date along with other antemortem patient information could not be obtained. The whole eye was initially evaluated with a high-resolution anterior-segment magnetic resonance imaging (MRI) system. This revealed the presence of an IOL with features characteristic of a 1-piece, open-loop PC IOL implanted in the anterior chamber. The haptics were fixated to the angle. A longitudinal MRI view revealed the optic to be in a coronal plane slightly anterior to the plane of the haptics. This slight vaulting of the optic appeared to push one of the haptics onto the surface of the iris, causing distortion (Figure 1, *A*). The frontal MRI view (Figure 1, *B*) showed the haptics in a counterclockwise configuration. By MRI inspection, the IOL was fairly centered with respect to the pupil. The IOL was identified as a Pannu universal IOL. As this design was withdrawn from the market in the late 1980s, the IOL implantation date was estimated as approximately 20 years earlier.

Laboratory Analysis

The enucleated eye containing the Pannu IOL was immersed in 10% neutral buffered formalin and sent to the John A. Moran Eye Center for pathological examination. The eye was sectioned at the equator, and gross examination of the anterior segment was performed from a posterior view and an anterior view (after excision of the cornea). The IOL was then carefully explanted and underwent gross and light microscopic analyses. Gross photographs were taken using a digital camera (model D1x with an ED 28–70 mm AF lens, Nikon). The eye (including the removed cornea) was prepared for histopathological examination using standard tissue preparation and processing. Sections were cut and stained with hematoxylin–eosin, Masson trichrome, and periodic acid-Schiff (PAS). Light microscopy was performed using a light microscope (model BX40, Olympus), and photomicrographs were obtained via a microscope-mounted camera (model DP20, Olympus).

Gross analysis of the anterior segment revealed the presence of significant pigmentary dispersion and peripheral anterior synechial formation. The pupil margin was slightly ovalized with the pupil long axis oriented along the axis of loop contact with the angle. Two surgical peripheral iridectomies had been performed. The IOL appeared to be relatively well centered, and there were various iris transillumination defects surrounding one haptic, especially in relation to its peripheral ring. No capsular bag was present (Figure 2). Explantation of the IOL was difficult as one haptic was incarcerated in the iris tissue. Iris pigments were found throughout the IOL surfaces (Figure 3).

Histopathology showed that the cornea was thickened and there was significant attenuation of corneal endothelial cells (Figure 4, *A* and *B*). The anterior chamber angle was widened in areas corresponding to the haptic footprints. In other areas, peripheral anterior synechiae obliterated the angle architecture. There was pigment within the trabecular meshwork and significant pigment dispersion throughout the anterior chamber. The specimen also showed significant iris pigment epithelium vacuolization with multiple areas of iridoschisis, probably corresponding to gross transillumination defects (Figure 4, *C* and *D*). Histopathology of the posterior pole yielded attenuation of the ganglion cell layer, consistent with glaucoma, with other retinal changes seen in normal aging. Finally, the basement membrane in the ciliary body epithelium was moderately thickened, consistent with diabetes mellitus.

DISCUSSION

The Pannu IOL was designed and marketed to function as a universal IOL and has design features that are overall similar to those of a modern PC IOL.¹⁻³ In our specimen, the IOL was relatively well centered in the anterior chamber and on the pupil of the specimen.⁴ It induced only marginal pupillary ovalization, perhaps indicating that the area of haptic contact with the angle did not exert enough pressure to compromise blood flow and induce iris stromal and muscular atrophy over time. However, pathological findings, possibly related to the presence of the IOL in the anterior chamber, included significant pigmentary dispersion with iris transillumination defects, peripheral anterior synechial formation, loop incarceration in iris tissue, and attenuation of corneal endothelial cells. Although unknown in this donor, development of complications such as pigmentary glaucoma remains a possibility considering the pigmentary dispersion and attenuation of the ganglion cell layer. Corneal damage in relation to an IOL implanted in the anterior chamber might have been caused by contact between the IOL and the corneal endothelium and also by chronic subclinical inflammation. Irritation of the angle by the IOL loops releases inflammatory mediators in the anterior chamber. Iris chafing by an IOL not only leads to inflammation; it can also liberate pigments from damaged cells and induce pigment dispersion syndrome.⁵

Clinical studies have described the complications associated with implanting posterior chamber IOLs in the anterior chamber. Hara and Hara⁶ report the outcomes in 21 eyes with a Sinsky-type conventional PC IOL. High corneal endothelial cell loss was observed after a mean 10-year follow-up. Liu et al.⁷ and Koch and Wang⁸ describe loop encapsulation by peripheral anterior synechiae, recurrent iritis, central loop displacement with corneal endothelial touch, progressive endothelial cell loss, corneal edema, glaucoma, and cystoid macular edema as some of the possible complications with 3-piece C-loop PC IOLs in the anterior chamber.

Surgeons worldwide still implant PC IOLs in the anterior chamber in eyes that lack capsule support. We postulate that this is a dangerous practice given the various clinical studies showing the possible complications in these cases. This study also shows deleterious long-term biological consequences of implanting the Pannu universal IOL in the anterior chamber because of its overall design and mechanism of fixation.

Acknowledgments

Supported in part by a Seed Grant of the University of Utah to Liliana Werner, MD, PhD, and by Alcon Laboratories, Inc. and the National Eye Institute (grant # R43EY018518) to MRI Research.

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Biography

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Figure 1. High-resolution MRI photographs of the enucleated eye implanted with the Pannu IOL. *A*: Longitudinal view; the *arrow* shows one loop pushing onto the iris. *B*: Frontal view; the IOL is fairly centered and the haptics are in a counterclockwise configuration. A small degree of pupil ovalization can be seen.

Figure 2.

Gross photographs of the anterior segment of the eye implanted with the Pannu IOL. *A*: Anterior view after removal of the cornea. Note the pupil ovalization approximately along the axis of the rings at the extremities of the loops of the lens. *B* and *C*: Posterior view. No capsular bag was found. There were multiple transillumination defects and 2 iridectomies. The *arrow* shows one of the loop rings through an iris defect. *D*: Oblique view showing the ring at one loop extremity contacting the angle and pushing onto the iris.

Figure 3. Gross (A) and light (B to D) photomicrographs of the explanted Pannu IOL. The *arrow* shows iris tissue attached to one of the loops. Iris pigments can be seen throughout the surface of the loops (B and D) and optic (C) (B to D, original magnification $\times 20$).

Figure 4. Photomicrographs from histopathological sections cut through the cornea (*A* and *B*) and anterior segment (*C* and *D*) of the eye implanted with the Pannu IOL. *A* and *B*: Cornea is thickened and the *arrow* shows an area of attenuation of the corneal endothelium (PAS stain; original magnification $\times 200$). *C* and *D*: The iris exhibited vacuolization of the iris epithelium and areas of schisis (PAS stain; original magnification $\times 200$ and $\times 400$, respectively).