A small amount of postoperative inflammation is always associated with cataract surgery. Toxic anterior segment syndrome (TASS) represents an inordinate amount of inflammation, usually with hypopyon. The complication was called sterile endophthalmitis and sterile hypopyon1,2 before being more appropriately named TASS by Monson et al3 in 1992. Lens-induced TASS has likely occurred since Harold Ridley implanted the first IOLs in 1949, but only in the past 15 years has the problem been well recognized as a toxic syndrome.

DIFFERENTIAL DIAGNOSIS

It is difficult to distinguish TASS from infectious endophthalmitis, but the distinction is important, because the treatment of each complication is so different. Several clinical findings help to separate the two. The onset of TASS is generally earlier, 1 versus 4 to 7 days. With TASS, the vitreous is usually clear, and the heavy administration of topical steroids yields improvement. If performed, a vitreous tap and culture show no organisms in TASS. A confounder, unfortunately, is that the results of Gram staining and culturing may also not be positive with infectious endophthalmitis, depending on the care taken and method used.

A variant of TASS involves severe endothelial damage with limbus-to-limbus edema. This problem is usually not induced by the IOL but by a toxin such as detergent or sterile water that has entered the anterior chamber. Mamalis et al4 published a review of TASS that is an excellent resource for ophthalmologists.

If the clinical appearance of the eye and the characteristics of the infection are suspicious for TASS rather than an infectious etiology, it seems reasonable to dose the patient heavily with topical steroids for 8 to 12 hours and then reevaluate the eye before proceeding to vitrectomy and intraocular antibiotics.

ANECDOCTAL CASES

The 1970s

Early uveitis-glaucoma-hyphema (UGH) syndrome with poorly manufactured ACIOLs was not strictly TASS, but the problem plagued ophthalmologists in the 1970s. The original Choyce Mark VIII anterior chamber lens (Rayner Intraocular Lenses Ltd., East Sussex, UK) was never associated with UGH syndrome. Unfortunately, the company's production capacity was limited and the lens in short supply. US manufacturers began to produce copies of the lens, but they were poorly made with rough edges. Also, the IOLs were injection molded rather than lathe cut. The injection-molded PMMA tended to warp over time in the eye. This warpage and the lens' rough edges led to iris chafing and UGH syndrome. Fortunately, the Choyce VIII lenses were easily exchanged. Closed-loop lenses were more difficult to remove and frequently required surgeons to cut the loops and leave portions in the angle.

When used with intracapsular surgery, metal looped, Binkhorst-style lenses were too heavy and caused fraying of the iris sphincter, UGH syndrome, and dislocation or subluxation of the IOL.

The initial Binkhorst and Worst Medallion IOLs (both manufactured by Dutch Medical Workshop) came in a glass tube filled with NaOH solution. The other side of the tube was filled with a neutralizing solution. After neutralizing the lenses, ophthalmologists washed them with 2L of normal saline solution to eliminate residual NaOH. Although I had no related complications, there were reports of severe damage to the anterior segment in cases where the NaOH was not fully neutralized and removed.

Interestingly, the lenses stored in this solution had a reputation for the least postoperative inflammation. The reason was probably that the NaOH dissolved and washed off residual contaminants, particularly polishing compound.

Morcher GmbH (Stuttgart, Germany) had a problem with TASS involving gamma-radiation-sterilized IOLs around 1970. Its exact cause was not determined.
COVER STORY

Orcolon

The IOP is frequently high with TASS, due either to a clogging of the trabecular meshwork or a direct, toxic effect. One unusual variant occurred with Orcolon (polyacrylamide gel; Optical Radiation Corporation) around 1991. This viscoelastic performed well in clinical trials. After its FDA approval, however, there were a number of cases in which the eye, after doing well initially, suddenly developed an IOP in the 50s along with pain but little inflammation. The increased pressure was minimally affected by aqueous suppressants and responded only to paracentesis and filtering surgery. It was as if the trabecular meshwork suddenly and totally shut down 1 week after surgery.

In my first case of a shutdown trabecular meshwork, I had no idea what had happened. I made some calls, and Randy Craven, MD, a glaucoma specialist in Denver, advised me of the Orcolon disaster. At that time, Optical Radiation Corporation had a particularly aggressive and effective sales representative covering the Denver area, and a number of the doctors there were using both Orcolon and the Memorylens (CIBA Vision, Duluth, GA). In retrospect, it was a terrible combination. As one of the few glaucoma specialists in Denver at that time, Dr. Craven rapidly accumulated a lot of experience with Orcolon-associated glaucoma.

Only a few eyes in which Orcolon was used developed this form of glaucoma. At that time, the pre-rolled Memorylens had just been approved. With the initial lenses, I waited for the IOL to unroll partially, which took a few minutes, and then aspirated the viscoelastic. Those eyes are the ones that developed the glaucoma. Apparently, the prolonged contact time with the viscoelastic allowed it to penetrate the trabecular meshwork and cause the problem.

It was a mystery why the complication was never encountered in the clinical trials. It turned out that, when Orcolon’s production went from small to large lots, the glass bottles in which it was made were rinsed and autoclaved after each batch. Residual Orcolon on the glass containers’ walls was polymerized by the autoclaving, and then microscopic particles flaked into the next batch. They were invisible to conventional microscopy and only observable by phase contrast microscopy. These particles apparently embedded in the trabecular meshwork and set off some type of inflammatory reaction that culminated in a complete shutdown of the meshwork. After the reaction was understood, ophthalmologists could see small, inflammatory precipitates on the surface of the trabecular meshwork by careful gonioscopy. Early, heavy dosing of topical steroids could rescue eyes developing Orcolon-related glaucoma.

Unfortunately, the rash of subsequent lawsuits derailed a full investigation of the problem’s etiology, and few reported the condition in the literature. A useful animal model of glaucoma might have been found.

The Memorylens

My personal experience is fortunately fairly limited, but I think it might be instructive to share some of it. In 1999, a small number of eyes in which I had implanted a Memorylens developed what appeared to be infectious endophthalmitis postoperatively. The first few I referred for vitrectomy and intraocular antibiotics. All did well, and none had any identifiable organism. I thought the results were due to inadequate culturing and Gram staining techniques by the vitreoretinal surgeons. After hearing about cases of sterile postoperative inflammation across the country, I realized that TASS was likely the culprit. A couple of cases thereafter were successfully treated with heavily dosed topical steroids alone. An investigation by CIBA Vision identified residual polishing compound as the apparent problem.

CONCLUSION

Although IOL-related TASS still occurs, it is fortunately increasingly rare. Manufacturers’ awareness of the importance of removing polishing and cleaning compounds from the IOLs has vastly reduced the problem. Most cases now appear to derive from endotoxins in sterilizers’ reservoirs, detergent in instruments’ lumens, and other local issues. Vigilance continues to be necessary.

Luther L. Fry, MD, is Clinical Assistant Professor of Ophthalmology at the University of Kansas Medical Center in Kansas City, and he is in private practice in Garden City, Kansas. He acknowledged no financial interest in the products or companies mentioned herein. Dr. Fry may be reached at (620) 275-6302; lufry@fryeye.com.