Understanding the Subtleties of UGH Syndrome

Uveitis-glaucoma-hyphema (UGH) syndrome was first described by Ellingson in 1977 as a complication from the implantation of the Mark VIII anterior chamber IOL (Surgidev).[1] The condition was thought to be caused by mechanical chafing of the iris by the implant. UGH syndrome resolved, on average, by 2 months following removal of the IOL. The explanted lenses showed warpage of the footplates that was thought to have occurred in situ.

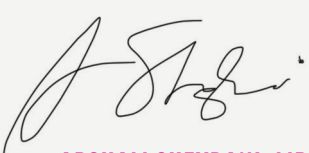
Replacement with the same model IOL in these same patients did not cause a recurrence of the syndrome, so the culprit was assumed to be the originally implanted IOL itself. Residual polishing compounds on the IOL, manufacturing defects, sharp edges, and imperfections of the IOL were also implicated.

UGH syndrome was subsequently reported to be caused by a variety of anterior chamber IOLs. The widely held definition of UGH syndrome implies the presence of a classic triad of anterior uveitis, glaucoma, and hyphema. However, in a not-yet-published case series, Iqbal Ike K. Ahmed, MD, FRCSC, and I found the following:

- Only about 5% of eyes presented with the classic triad;
- A total of 15% did not have anterior uveitis, glaucoma, or hyphema, but some of them had cystoid macular edema or pain;
- A delay in diagnosis sometimes resulted in the need for long-term steroid treatment, even after UGH syndrome had resolved; and
- Increased pigmentation in the angle viewed by gonioscopy was the most common sign of UGH syndrome (found in 68% of cases).

In this issue of *GT*, we hope to provide diagnostic and management pearls for a condition that can be subtle but is treatable when addressed properly. ■

1. Ellingson FT. Complications with the Choyce Mark VIII anterior chamber lens implant (uveitis-glaucoma-hyphema). *Am Intra-Ocular Implant Soc J.* 1977;3:199–201.



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