Calcification in Hydrophilic Lenses

Recent cases suggest environmental factors can cause calcification in the pupillary region.

BY ANISH DHITAL, MRCOPTH; AND DAVID J. SPALTON, FRCS, FRCP, FRCOPTH

Hydrophilic IOLs have many appealing characteristics, such as good optical clarity, good biocompatibility, a low incidence of dysphotopias, and good compressibility allowing implantation through small incisions. Many of these characteristics are due to the water content of hydrophilic lenses, which can vary from 18% to 30%. In contrast, hydrophobic lenses contain less than 1% water.

Calcification has been reported with some hydrophilic IOLs, and subsequently many have had to be explanted. As a result, the usage and reputation of these lens materials have suffered. However, newer hydrophilic IOLs appear to be largely free of this problem and are widely used today.

BACKGROUND

Varying patterns of calcification were seen with different hydrophilic IOLs in the past. For example, the Hydrioview IOL (Bausch + Lomb, Rochester, New York) developed a uniform superficial calcification on the anterior and posterior surfaces of the lens. The Aquasense IOL (Ophthalmic Innovations International Inc.; now Aaren Scientific, Ontario, Canada) developed a deep nuclear pattern of calcification. Some hydrophilic lenses have developed widespread opacification over the haptics and the optic, which was thought to be due to dystrophic calcification.

Recently, there have been a small number of case reports of an unusual pattern of calcification seen with some hydrophilic lenses. But the sources of the development of calcification in these recent cases are due to environmental or patient-related factors rather than the lens material.

Compared with earlier incidents of calcification in hydrophilic IOLs, these cases are characterized by a pattern of calcification confined to the pupillary area. On slit-lamp examination, the calcification appears to be superficial, either on or in the IOL surface, and consists of myriad tiny vesicular lesions 10 to 20 µm in size.

These tiny lesions coalesce in some regions almost to confluence, causing visual loss.

These observations were first reported at the Annual Congress of the Royal College of Ophthalmologists this year. In five cases of IOL opacification, intracameral tissue-plasminogen activator (tPA) had been used to remove a fibrinous membrane within a few weeks of routine cataract surgery. Opacification of the hydrophilic IOLs developed 6 months to several years later. Imaging with light microscopy of the explanted lenses confirmed granular IOL opacification on the anterior surface of the IOL limited to the pupillary region, and calcium stains confirmed the presence of calcium within these areas, suggesting the lesions were due to calcification. The authors hypothesized that tPA elevated calcium levels in the anterior chamber by causing lysis of the fibrinous membrane, thus predisposing the hydrophilic IOL to these changes. The Medicines and Healthcare products Regulatory Agency in the United Kingdom is warning surgeons of this association.

Lee et al recently described two cases of focal opacification confined to the pupillary area of C-flex IOLs (model 570C; Rayner Intraocular Lenses, Ltd., East Sussex, United Kingdom) in patients who had undergone combined phacoemulsification and vitrectomy. One patient had proliferative diabetic retinopathy and cataract; the other had a combined procedure due to a retinal tear and cataract. The opacification developed...

TAKE-HOME MESSAGE

- Recent cases have been characterized by a small pattern of calcification confined to the pupillary area.
- Intraocular gas is a suspected culprit in IOL opacification.
- A linking factor among these cases is multiple surgeries with surgical trauma and damaged blood aqueous barriers, which possibly allow protein penetration into the IOL surface followed by calcification.
approximately 6 months after cataract surgery in both cases. The opacified IOLs were explanted and examined with scanning electron microscopy and x-ray energy dispersive spectroscopy, which confirmed focal calcium deposits on the anterior surface of the IOL limited to the pupillary zone. The IOLs also had quadrangular cracks on their surfaces, which could be caused by pathologic changes or may be artifacts due to the hydrophilic lens drying out in the environment of the scanning electron microscope (SEM).

CASE REPORTS
We have seen an identical pattern of opacification in a 45-year-old patient who presented with glare and reduced visual acuity to our eye emergency service. The patient had previously suffered blunt trauma to the same eye and had undergone uneventful cataract surgery with implantation of a hydrophilic IOL 4 years previously at a different unit. He had also undergone vitrectomy with cryotherapy and SF₆ gas injection 1 year earlier for retinal detachment. Visual acuity was 0.2 logMAR and 0.8 logMAR at 9% contrast at presentation. Upon slit-lamp examination, there was central IOL opacification comprising granular areas confined to the pupillary zone of the anterior IOL surface (Figure 1). The patient subsequently underwent IOL exchange, during which the preexisting damage to the zonules in the inferonasal sector was seen, but surgery was otherwise unremarkable. The explanted IOL was imaged with environmental SEM to examine the surface opacification (Figures 2 and 3). The opacities were approximately 15 µm across, homogeneous, and resided on the surface of the lens with extension into the substance of the lens. X-ray energy dispersive spectrometry confirmed the presence of calcium and phosphorus within these areas.

We have observed similar changes in two patients with hydrophilic IOLs (data submitted for publication in the Journal of Cataract & Refractive Surgery). We, like Saeed et al, identified intraocular gas as a culprit in these IOL opacifications. In our two cases, intraocular gas (SF₆ and C₃F₈) was injected into the anterior chamber for tamponade to treat hypotony following trabeculectomy or to reposition a Descemet’s membrane detachment. In the first case, we hypothesize that the SF₆ injected into the vitreous cavity escaped to the anterior chamber via
the area of deficient zonules sustained from previous blunt trauma and came into contact with the anterior IOL surface in the pupil, initiating the calcification. The iris protected the peripheral IOL from this reaction.

CONCLUSION

There are obvious clinical similarities among these cases, the most significant being localized central superficial anterior opacification of the hydrophilic IOL in the pupillary area due to calcification. This well demarcated and localized appearance suggests an environmental cause. These cases are rare, with no common denominators among the intracameral drugs or ophthalmic viscosurgical devices used. A linking factor appears to be multiple surgeries with surgical trauma and damaged blood aqueous barriers, which possibly allow protein penetration into the IOL surface followed by calcification. Visual loss is severe, and the only treatment is IOL exchange.

Anish Dhital, MRCOphth, is a research fellow at St. Thomas’ Hospital, London. Mr. Dhital states that his research is funded by Fight for Sight. He may be reached at e-mail: anishdhital@gmail.com.

David J. Spalton, FRCS, FRCPh, FRCOphth, is a Consultant Ophthalmic Surgeon at St. Thomas’ Hospital, London. Professor Spalton is a member of the CRST Europe Editorial Board. He states that he is a consultant to Bausch + Lomb and Santen Pharmaceutical, Co., Ltd. He may be reached at e-mail: spalton@eyepractice.fsnet.co.uk.


CONTACT US

Send us your thoughts via e-mail to letters@bmctoday.com.