Spontaneous Anterior Capsular Rupture Associated With Anterior Polar Cataract

Lens capsular rupture has been reported to be traumatic or spontaneous. Traumatic capsular ruptures can occur from penetrating,1 surgical,2 3 or blunt trauma.4 Spontaneous capsular rupture has been described after cataract extraction in the fellow eye5 and associated with hypermature cataracts.6

To our knowledge, there have been no reports of spontaneous anterior capsular rupture causing lens-induced uveitis in the setting of anterior polar cataracts. We report the case of a 59-year-old woman with long-standing anterior polar cataract who developed spontaneous capsular rupture and lens-induced uveitis.

Report of a Case. A 59-year-old white woman complained of recurrent episodes of pain and photophobia in the left eye. The last episode was 1 year prior to initial examination, during which time she was diagnosed as having anterior uveitis and treated with topical steroids. Her ocular history was notable for a congenital polar cataract diagnosed early in childhood and amblyopia in the affected left eye. She had no history of trauma or ocular surgery. Her medical history included controlled type 2 diabetes mellitus and hypertension but was otherwise unremarkable.

When seen at the University of Texas Health Science Center at San Antonio Cornea and Uveitis Service, her best-corrected visual acuity was 20/80 OS. Ophthalmic examination revealed a white mass protruding on the anterior lens capsule adjacent to the anterior polar cataract (Figures 1, 2, and 3). The remainder of the lens showed 1 to 2+ nuclear sclerosis. Flare and cells (2+) were present in the anterior chamber, but there was no fibrin or hypopyon. Guttata and pigment were present in the endothelium of both eyes. Uveitis evaluation was noncontributory.

Hourly 1% prednisolone acetate and once daily 1% atropine were prescribed. The anterior uveitis resolved in 26 days, and phacoemulsification without intraocular lens implantation was performed 5 weeks after initial examination. During surgery, the anterior capsule and anterior polar cataract were recovered. At the end of surgery, a sub-Tenon injection of 40 mg of triamcinolone acetonide was administered in the inferonasal quadrant.

The capsular specimen was fixed in 10% formaldehyde and stained with hematoxylin-eosin. The presence of a break in the anterior capsule, through which the cortical material was protruding, was confirmed histologically (Figure 4). Aside from a few isolated pigment-containing macrophages, no neutrophils, eosinophils, or other inflammatory cells were present at the site of the capsular break.

One month postoperatively, the eye continued to be quiet and topical steroids were tapered until administration was discontinued. Five months postoperatively, the patient obtained a visual acuity of 20/30 OS with a +8.50-diopter contact lens. At this time, both eyes were quiet during slitlamp examination.

Comment. Lens-induced uveitis has been recognized since 1919 by Straub7 and later by Verhoeff and Lemoine.8 The condition is traditionally explained by rupture of the lens capsule either by trauma or spontaneously; the latter is usually related to the presence of hyperma-

Figure 1. Anteroposterior slitlamp photograph of the anterior polar cataract.

Figure 2. Gonioscopic view of the anterior cortex protruding through the capsular opening.

Figure 3. Slitlamp photograph of the protruding cortex in the anterior chamber.

Figure 4. Hematoxylin-eosin low-power light microscopy of the anterior lens. The solid arrows point to the thinning edges of the capsule at the site of rupture. The central dashed arrow is pointing to the anterior subcapsular cataract. Cortical material is seen between the capsular edges.
ture cataracts. To our knowledge, spontaneous capsular rupture causing lens-induced uveitis in the setting of anterior polar cataract has not been described.

Definitive treatment for lens-induced uveitis is cataract extraction. Intracapsular cataract extraction has been the proposed method for treatment of phacogenic uveitis associated with hypermature cataracts to avoid the protein exposure resulting from extracapsular techniques. This technique, however, precludes capsular-based intraocular lens fixation. In patients with uveitis, surgery is considered safer when there has been control of the inflammation for at least 3 months. In our patient with lens-induced uveitis, however, we performed phacoemulsification promptly once the uveitis was quiet. Although primary posterior chamber intraocular lens insertion may have been reasonable, we decided against it, knowing that some patients can develop chronic intractable intraocular inflammation requiring lens removal to quiet the eye and prevent irreversible damage.

Our diagnosis of lens-induced uveitis was confirmed histopathologically by the presence of a break in the anterior capsule. The absence of inflammatory cells within the substance of the specimen can be explained by the topical use of hourly prednisolone acetate prior to surgery.

We believe our case is the first report of spontaneous capsular rupture in the setting of anterior polar cataract, causing moderate lens-induced uveitis. This patient’s uveitis was successfully treated by cataract extraction and local steroids. Visual rehabilitation was achieved with a contact lens.

Juan G. Gaviria, MD
Daniel A. Johnson, MD
Frank W. Scribbick III, MD
Mark J. Gallardo, MD

Correspondence: Dr Gaviria, Department of Ophthalmology, University of Texas Health Science Center at San Antonio, 7703 Floyd Curl Dr, MSC 6230, San Antonio, TX 78229 (juangaviria@lycos.com).

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