disease, although 10% of hospitalized patients require intensive care because of respiratory distress, renal failure, or severe neurologic involvement.[5] Ophthalmic involvement is rare and only a few reports on ophthalmic murine typhus can be found in the literature.[6–9,11]

The largest cases series of murine typhus–associated ophthalmic findings was reported by Khairallah et al.[8] The most common ophthalmic findings, present in 50% of their patients, were white retinal lesions located in the inner retina associated with mild vitritis. These lesions were thought to represent areas of retinitis from either rickettsial multiplication or inflammatory cell accumulation. Similar retinal lesions have also been described by Hudson et al.[6] and by Lu et al.[7] Angiographically confirmed retinal vasculitis (38.9%) and retinal hemorrhages (22.2%) were also common in Khairallah et al.'s series.[8]

Optic neuropathy has also been described in murine typhus. The exact mechanism of this typhus-associated optic neuropathy is unknown, but it probably represents an inflammatory reaction. In Khairallah et al.'s series,[8] 66.6% of eyes had staining on FA, 11.1% had disc edema, and 1 had optic neuritis. Our patient's case is unique as, to our knowledge, there are no previous reports of bilateral simultaneous retrobulbar optic neuropathy associated with endemic typhus. Although optic atrophy may appear after disc edema has resolved, our patient did not demonstrate disc edema on funduscopy, nor was there leakage on FA. Unlike Khairallah et al.'s series[8] in which most patients were asymptomatic, visual acuity was significantly decreased in our case.

In conclusion, endemic typhus can present as a retrobulbar optic neuropathy with severe and irreversible vision loss. The differential diagnosis of a patient with vitritis-associated optic neuropathy should include infectious and inflammatory causes. Infectious disease merits stronger consideration when these ocular findings occur in the setting of a recent exanthematic illness. Although more common infectious agents should be investigated first, *R. typhi* infection should also be considered in endemic areas, such as the State of Texas.

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Late spontaneous dislocation of a silicone iris-claw phakic intraocular lens

A 43-year-old female presented with sudden monocular diplopia and blurred vision OD for 4 days. Three years prior in Peru she had undergone successful implantation of bilateral Artiflex (Ophtec, Groningen, The Netherlands) phakic intraocular lenses (pIOL) for high myopia. The patient denied any recent or previous trauma. On examination, her Snellen uncorrected distance visual acuity (UDVA) was 20/30 OD and 20/50 OS. Intraocular pressures (IOPs) were normal in both eyes (13 mm Hg OD, 12 mm Hg OS). Slit-lamp examination showed clear cornea with no anterior chamber reaction evident. The nasal clip of the OD Artiflex pIOL was loose, causing the pIOL to hang inferiorly (Fig. 1). The temporal haptic remained fixed at the 9-o'clock position. Signs of iris atrophy were evident at the previous site of enclavation, and a peripheral iridotomy was patent superiorly.

Three days after presentation, the patient underwent successful repositioning of the dislocated pIOL. A 2.75-mm keratome was used to make a 12-o'clock corneal incision. Side ports were created using a 15-degree blade at 10 o'clock and 2 o'clock. Miochol (Iolab Pharmaceuticals, Claremont, Calif.) was injected to minimize pupil size followed by a cohesive viscoelastic to form the anterior chamber. A Sinskey hook was used to reposition the dislocated pIOL. The pIOL was held in place using a pIOL implantation forceps through the 12-o'clock incision. The dislocated haptic was re-enclavated using the

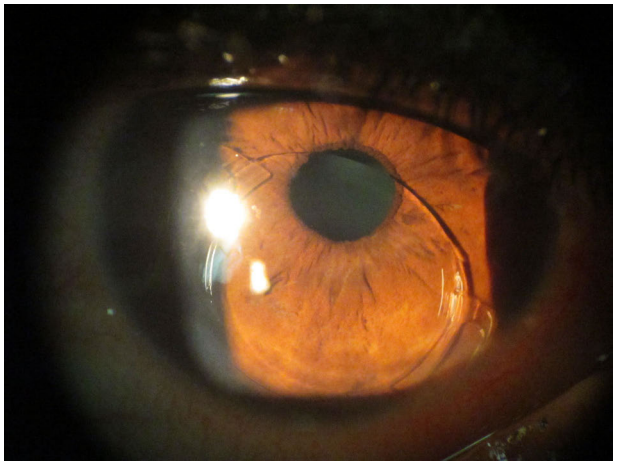

Fig. 1—Slit-lamp photo of patient's right eye at presentation, 3 years after original phakic intraocular lens (pIOL) implantation. Note the loose nasal clip of the Artiflex pIOL causing the lens to be dislocated inferiorly.

enclavation needle through the side ports. The 12-o'clock corneal incision was closed using 3 interrupted 10–0 nylon sutures. Irrigation of the anterior chamber through the main incision using a 3-mL syringe with balanced salt solution was performed to clear the viscoelastic. Postoperatively, the patient used topical moxifloxacin 0.5% and dexamethasone 0.1% qid and combination 2% dorzolamide HCl and 0.5% timolol maleate ophthalmic solution drops bid for 1 week.

The patient was followed at 1 day, 1 week, 1 month, 2 months, and 9 months postoperatively. At day 1 postoperatively, her diplopia had resolved. At 9 months, UDVA was 20/40 OD, manifest refraction of −1.00 −0.50 × 17 yielded best corrected vision of 20/25, and IOP was normal. Slit-lamp examination showed a stable and centred Artiflex lens and clear cornea (Fig. 2).

Phakic IOL implantation has become an effective and safe procedure for refractive correction, particularly in patients not suitable for LASIK and phototherapeutic keratectomy. The Artisan lens (Ophtec, Groningen, The Netherlands) was the original lens in the iris-claw pIOL family. It has a 5.0- to 6.0-mm polymethylmethacrylate lens (PMMA) optic with PMMA haptics. However, a corneal incision up to 6.2 mm in length is required for insertion. The Artiflex pIOL is a foldable version capable of being inserted through a 3.2-mm sutureless incision and uses a 6-mm silicone optic with PMMA haptics.

Given the longer history of the Artisan pIOL, the majority of literature concerning the complications associated with iris-claw pIOLs is derived from this older model. These studies often do not make the distinction between early and late spontaneous dislocation, traumatic and spontaneous dislocation, or the type of iris-claw pIOL in mixed cohorts. Titiyal et al.[1] reported the incidence rate of spontaneous dislocation at 7.3% in Artisan pIOL eyes in their series. Akcay et al.[2] showed dislocation (unspecified type) in 3.2% of Artiflex eyes, whereas Kwitko and Stolz[3] found spontaneous dislocation (mixed Artisan and Artiflex cohort; unspecified model) in 2.4% of iris-claw pIOL eyes. Van Philips[4] reported 1 Artiflex lens (2.4%) dislocation twice nasally (unspecified time) without evidence of trauma.

The precise causes of spontaneous dislocation of iris-fixed pIOLs are unclear. In early spontaneous dislocation, surgical factors are likely to play a significant role. The surgeon must ensure adequate amounts of iris tissue are securing the haptics, whereas avoiding endothelium and crystalline lens contact. Stulting et al.[5] noted that in their 662 cases of Artisan implantation, half of the adverse events and cases needing repositioning (31/61) involved the first 10 cases performed by each surgeon.

Late spontaneous dislocation of an iris-claw pIOL likely involves atrophy of the enclavated iris tissue caused by pressure exerted by the implant's haptic. In their report on Artisan pIOLs, Titiyal et al.[1] found evidence of iris tissue depigmentation and atrophy at the enclavation sites in 29.4% of eyes, and all cases of spontaneous dislocation showed signs of atrophy. Several other reports have also detected atrophy by the Artisan iris-claw haptics, including El Danasoury et al.[6] (27.9%), Benedetti et al.[7] (16.3%), and Saxena et al.[8] (11.8%), whereas others have found no evidence of iris atrophy.[9–11] Excessive manipulation of the iris tissue during enclavation may also predispose the iris tissue to atrophy. Rai et al.[12] reported a case where unrecognized damage to a haptic in a challenging enclavation procedure led to disenclavation in an Artisan iris-claw pIOL 4 weeks later. This was successfully repaired by suturing the damaged haptic to the iris. In exceptional circumstances where no replacement lenses are available and leaving the patient aphakic for a later surgery is contraindicated, this alternative procedure may be appropriate.[12]

The presence of a dislocated pIOL is easily spotted by slit-lamp examination. The importance in recognizing its occurrence lies in educating the patient about identification of symptoms to prompt early medical examination

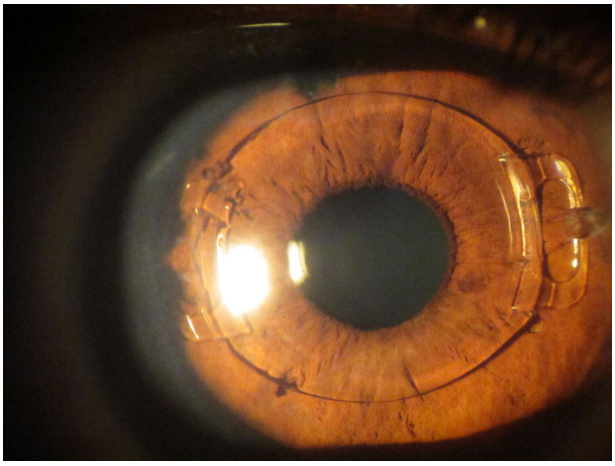

Fig. 2—Slit-lamp photo of patient's right eye 9 months after repositioning of the dislocated Artiflex silicone phakic intraocular lens.

before more serious complications occur. This includes corneal endothelial damage and cataract formation from rubbing of the implant against the cornea and lens, respectively. Harsum et al.[13] reported a case of an Artisan late spontaneous dislocation with delayed seeking of medical attention resulting in corneal decompensation requiring an endothelial graft. Symptoms may be mild with no significant loss in visual acuity, as in our patient whose primary complaint was monocular-diplopia and as reported by Singhal and Sridhar[14] in 2 cases of Artisan late spontaneous dislocation. No studies have examined the recurrence of these complications.

In conclusion, this case highlights the possibility of late spontaneous dislocation of an Artiflex lens as a rare complication in iris-claw pIOL implantation, as well as the importance of educating patients to identify symptoms and seek medical treatment even in the absence of obvious trauma to prevent further complications.

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A cystic epithelial downgrowth mimics an intraocular tumour following penetrating eye trauma

Epithelial ingrowth, also known as epithelial downgrowth (ED), is a potentially blinding complication of intraocular surgery or penetrating eye trauma.[1] It is characterized by the proliferation of corneal or conjunctival epithelium in the anterior chamber.[1] ED can be cystic or diffuse.[2] The cystic type has a more benign course and is defined by its well-circumscribed margins.[2] The diffuse type has a sheet-like morphology and tends to follow an aggressive, recurring course.[3]

In the literature, approximately 82% of affected individuals will present for care within 1 year after a penetrating ocular event.[4] These patients typically complain of decreasing visual acuity, ocular injection, and pain.[5] Clinical signs such as a visible iris cyst or a retrocorneal membrane may be observed.[6] Pupil irregularities, an abnormal iris surface, and corneal edema could also indicate a potential ED.[5] Early identification of an ED is important, because the proliferation of epithelial cells in the anterior chamber can lead to secondary glaucoma and phthisis.[4]

In this article, we report a case of a cystic ED that was diagnosed on an eye enucleated 43 years after penetrating eye trauma. This case highlights the potential of mucin-secreting goblet cells, incorporated in the ED, to complicate the diagnosis. This case also represents one of the longest reported time intervals between eye trauma and enucleation.

A 61-year-old female with a blind left eye presented with a pearly-white lesion in the anterior chamber. At age 20 years, she had been involved in a car accident in which shards of glass penetrated her left eye, resulting in blindness. Apart from a history of recurrent intraocular inflammation within recent years, this patient had minimal to no ocular complaints in the left eye after the