Retained intraocular sand resulting in corneal endothelial cell dysfunction: A Case Report

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Abstract

Introduction: Intraocular foreign bodies (IOFB) are common in trauma settings[1]. Certain IOFB such as sand may lay inert and not cause an immediate immunologic response to the eye[2]. We present a case of an inert intraocular foreign body which slowly contributed to endothelial cell dysfunction and corneal decompensation for 5 months before initial patient presentation.

Patient: A 43-year-old male presented with a 5-month history of progressive blurry vision in the left eye. Initial presentation showed decreased visual acuity, slit lamp exam showed ascending corneal edema and a scar in the inferonasal cornea. On gonioscopy a small crystalline mass was noted to be resting over the iris near the inferior angle. The patient denied any previous history of trauma to either eye.

Case: The patient was initially treated conservatively with a course of valacyclovir and sodium chloride ointment without improvement. He subsequently underwent surgical removal of the crystalline fragment which was sent to pathology for examination. After removal of the foreign body, the corneal edema did not improve and the patient eventually underwent Descemet Membrane Endothelial Keratoplasty (DMEK) for the management of endothelial cell dysfunction. Following DMEK, the patient developed Urrets-Zavalia syndrome, however, his visual acuity was improved and his corneal edema resolved.

Discussion: Patients presenting with sectoral corneal edema and bullous keratopathy should raise suspicion for IOFB. Detailed history is often not enough, as objects can remain asymptomatic for years following initial insult[3,4]. The common complaint of decreased visual acuity is often accompanied by localized corneal edema due mechanical corneal contact or siderosis[5]. Further examination of gonioscopy and immediate action is indicated to locate the foreign body to prevent further damage and corneal decompensation[4]. This case was further complicated by Urrets-Zavalia (UZS) following DMEK. While UZS is a well-recognized, yet rare, postoperative complication that was first described following penetrating keratoplasty surgery but has not been reported following foreign body removal with subsequent DMEK.

Introduction:

Migratory foreign bodies resulting in endothelial cell dysfunction and DMEK have yet to be reported in the literature. In cases described by Liabson, corneal edema would reverse, and visual acuity was restored when the object was removed given adequate time (1965). The case presented includes an instance of a migratory foreign body from unknown trauma requiring foreign body removal and subsequent DMEK in order to meet the patient's visual acuity goals.

Patient:

A 43-year-old healthy male with no previous ocular history presented with a 5-month history of left sided progressive blurry vision. Best corrected vision was 20/20 in the right eye and 20/50 in the left eye, pinhole improvement only to 20/30. Intraocular pressure measured by Tono-pen was 20mm Hg in the right eye and 22mm Hg in the left eye. Screening revealed no afferent pupillary defect in either eye. The cornea was clear in the right eye. Examination of the patient's left cornea revealed ascending microcystic corneal edema to his visual axis. A few small bullae were noted inferiorly. A zig-zagging full thickness 2mm linear stromal scar without thinning located nasally, is shown in figure 1. Gonioscopy revealed open angles in the right eye and a sub- 1mm crystalline mass resting in iris near the inferior angle of the left eye; there were no peripheral anterior synechiae. The crystalline mass was difficult to characterize under high magnification however, based on appearance resembling a small piece of glass. This is illustrated in figures 2-3. Gonioscopy on the right eye was within normal limits, there was no anterior chamber cell bilaterally and posterior exam was within normal limits bilaterally.

Case:

The patient denied any previous history ocular trauma or previous episodes of blurry vision. Conservative management was initiated to treat the corneal edema with a course of muro ointment and oral valacyclovir due to concern for herpes simplex virus keratitis. The patient's follow-up was delayed due to the COVID-19 pandemic, however, the patient was evaluated two months after initial presentation at which time he remained symptomatic with unchanged visual acuity and no improvement in the corneal edema. Repeat gonioscopy showed a stable mass resting on the iris near the inferior angle, without apparent corneal touch. On further questioning the patient recalled an event five months prior to presentation where he had been caught in a sandstorm without eye protection. He endorsed a slight irritation for several days after the event and describes progressive blurriness after the incident. As the IOFB was believed to be the cause of the patient's corneal edema, the patient underwent IOFB extraction. The foreign body was removed through a superior paracentesis using healon, a Swan-Jacob gonio prism for direct visualization, and Grieshaber grasping forceps. Analysis via spectroscopy found the foreign body to be non-metallic, composed of carbon, oxygen, silicon, aluminum and potassium. The

composition was consistent with silica like material, most likely sand. It is believed the grain of sand was embedded into the corneal stroma during the sandstorm, possibly at high velocity. Then due to manipulation through rubbing, it worked its way through the reminder of the corneal stroma to rest near his inferior iridocorneal angle.

Following removal, the patient experienced minimal subjective improvement in his visual acuity with unchanged corneal edema for the following 3 months. At this point concern for endothelial cell failure secondary to mechanical trauma or trace metallic poisoning was increased. After consideration of the patient's visual acuity goals, a DMEK was completed 3 months after foreign body removal. The patient's early post-operative course was complicated with high postoperative day (POD) 1 intraocular pressures that were promptly decreased with use of Cosopt and Alphagan. On POD7 the patient was found to have a persistently mid-dilated pupil to 5mm on his operative eye. A course of pilocarpine was started. On POD 10 the patient's left pupil remained mid-dilated and unresponsive to pilocarpine therapy. A diagnosis of Uretts Zavalia was made. By POM 2 the patient had shown some improvement in pupil size. By POM6 the dilated pupil returned to 1mm larger than the right pupil and the visual acuity improved resulting in 20/30 vision with pinhole improvement to 20/20.

The removed endothelial host tissue was sent for histological analysis which revealed a dramatically thickened Descement's without significant endothelial cell loss. Iron stain returned negative however was limited given small sample size. Fungal and bacterial cultures returned without growth. See figure 4 for the histologic sample.

Discussion:

Presented is a case of a migratory foreign body, which was ultimately removed due to corneal decompensation. While the patient's account of his symptom onset was not for several weeks, further discussion revealed that he had been in a sandstorm five months earlier without eye protection. At that time, he admitted to an irritated and itchy eye which eventually resolved without ophthalmologic intervention. While the mechanism of migration cannot be confirmed, it is proposed to be due to high velocity trauma from the sand storm with additional mechanical manipulation from aggressive rubbing of the eye after the initial insult. This may have forced the foreign body through the cornea into his anterior chamber. The particle subsequently came to rest in the iris near the inferior iridocorneal angle leading to the presenting finding of endothelial decompensation and inferior bullous keratopathy.

With the additional history and mass spectrometry results, the substance identified in the presented patient is presumed to be sand. Traditionally, inert substances are not thought to migrate due to their lack of immunogenic response, however, the presented case as well as cases presented by Mitchell et al. demonstrate that this is not always true[2,4]. While the foreign body was discovered five months after the insult, it is important to examine inferior bullous keratopathy with gonioscopy, as foreign objects can take years to travel[5]. Further examination of the finding of inferior bullous keratopathy is always indicated with gonioscopy being the gold

standard [4,5]. Imaging can also help identify if there is a IOFB, with CT being the modality of choice [3]. In the presented case, CT was unrevealing. Once identified, prompt removal should ensue to prevent further mechanical damage, chemical damage, or the most common complication, infection[4]. Furthermore, as highlighted by the presented case, constant friction versus metallic toxicity can cause injury to endothelial cells and even result in complete corneal decompensation requiring more intensive management [4].

The thickened Descement's membrane seen in this case is unusual. It is hypothesized this may be secondary to stimulation from the retained intraocular forgien body versus a rare, familial form of early onset Fuch's dystrophy as described by Zhang et al[6]. The *COL8A2* mutation causes increased growth of the extracellular matrix and progressive deposition of extracellular material by endothelial cells resulting in a thickened and abnormal Descement's membrane[6]. Endothelial cell loss is seen later in the disease course as with traditional Fuch's Corneal Dystrophy[6]. This condition may explain why the patient was unable to clear his corneal edema following removal of the intraocular foreign body. The patient will continue to be monitored for signs of corneal edema and endothelial cell loss/failure in his contralateral eye.

This case was further complicated by Urrets-Zavalia (UZS) following DMEK. While UZS is a well-recognized, yet rare, postoperative complication that was first described following penetrating keratoplasty but has not been reported following foreign body removal with subsequent DMEK.

First described in 1963, UZS presented with a fixed dilated pupil, ocular hypertension, and often secondary glaucoma[7] . While the pathophysiology is not well understood, UZS is thought to be related to the atrophy and death of the vessels supplying the iris[8]. Ocular hypertension intraoperatively and postoperatively has been a suggested cause of UZS[8]. To the authors knowledge, UZS following foreign body removal and subsequent DMEK has not been reported in the United States. Immediately following completion of DMEK, the air bubble is reduced to prevent ocular hypertension and thus preventing iris atrophy perhaps acting as a safeguard against the development of UZS[9]. While this preventative measure was exercised within the reported case, the patient experienced high POD 1 intraocular pressures and experienced direct mechanical trauma and perhaps toxicity from the retained foreign body for close to 7 months prior to IOFB removal.

Conclusion:

This case shows a unique presentation of endothelial failure following presumed corneal embedded foreign body migration into the anterior chamber eventually requiring a DMEK likely influenced by a grossly abnormal Descement's membrane and probable early onset hereditary FCD. This case was further complicated by Urrets Zavalia syndrome. It should also be stressed that retained intraocular foreign bodies should always be considered when inferior bullous keratopathy is seen despite a lack of traumatic history.

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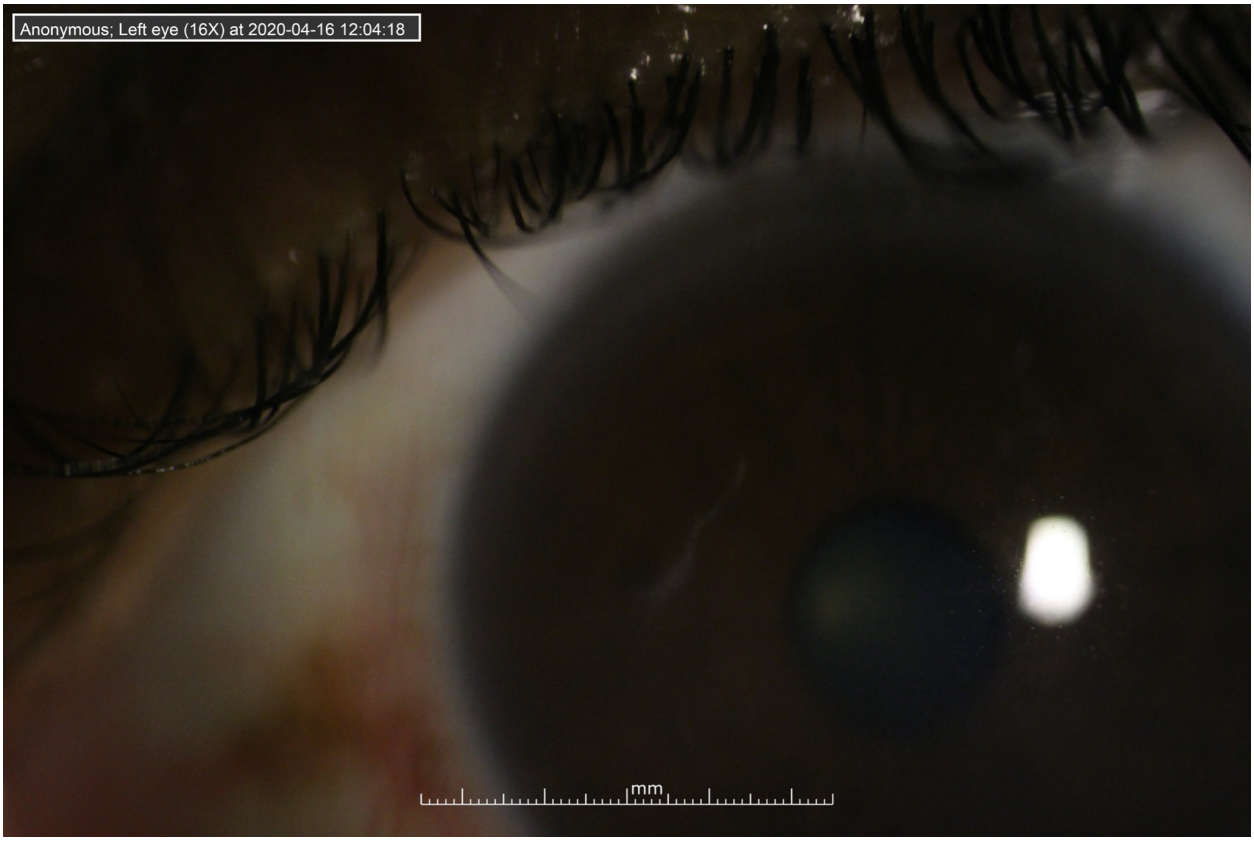
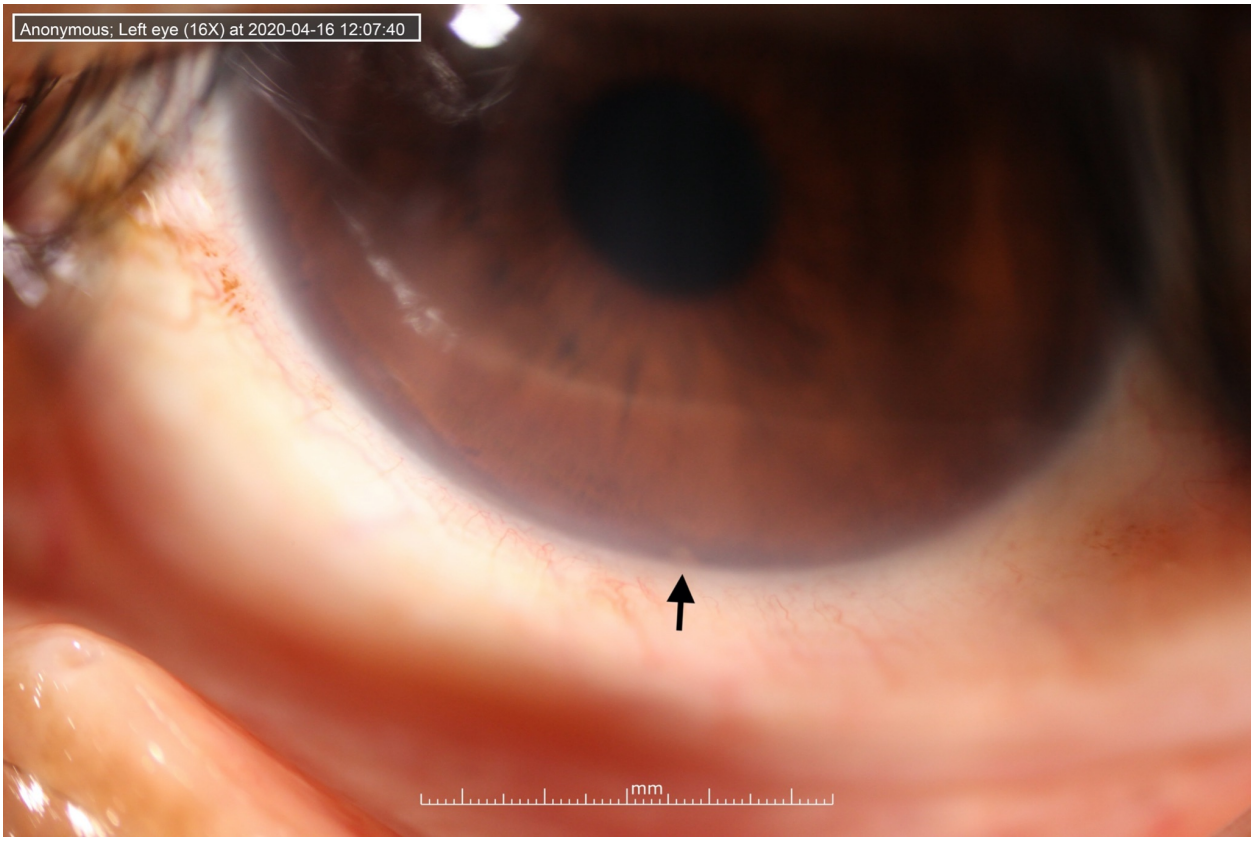
Figure 1.



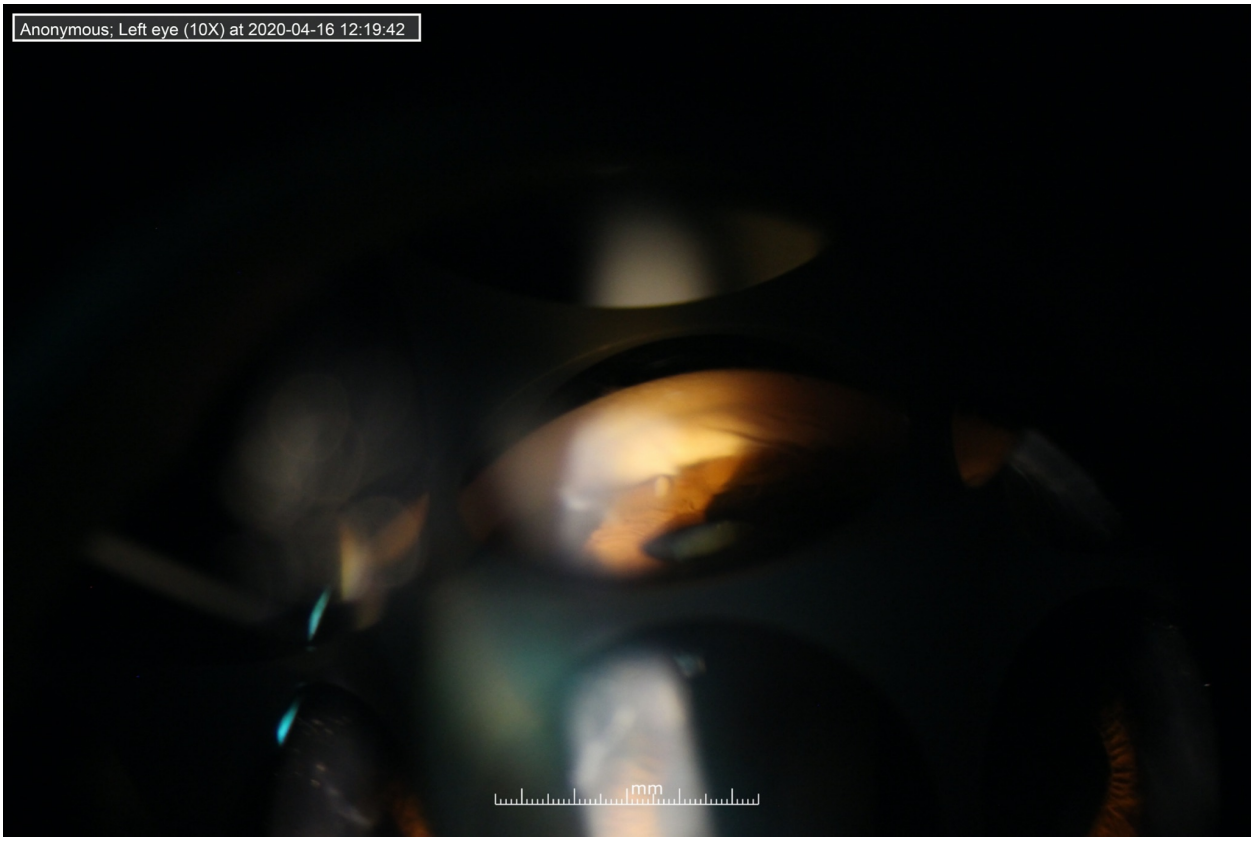
A zig-zagging full thickness 2mm linear stromal scar without thinning located nasally on left eye.

Figure 2.



Slit lamp photo of retained intraocular foregien body in left eye.

Figure 3.



Gonioscopic image showing the retained foreign body within the inferior corneal-irido angle.

Figure 4.

Histologic slide from DMEK sample showing significantly thickened descemet's with only mild loss of endothelial cells.