Interface Wavelike Deposits After Descemet Stripping Automated Endothelial Keratoplasty

Descemet stripping automated endothelial keratoplasty (DSAEK) is a new treatment option for corneal endothelial dysfunction. Compared with penetrating keratoplasty, DSAEK is less invasive and leads to more rapid visual recovery.

The most common complications after DSAEK described in the literature are graft detachment and/or dislocation, rejection, epithelial ingrowth, and pupillary block, all of which can lead to graft failure.1-3

Report of Cases. Patients were asked to sign an informed consent form prior to treatment. Institutional review board approval was obtained before medical record review. The operations were successful and without complications in both cases. The precut lamellar corneal DSAEK grafts were provided by eye banks. The DSAEK donor tissues were prepared using a Moria (Antony, France) automated microkeratome and a Moria artificial chamber. Optisol GS solution (Bausch & Lomb Surgical, Irvine, California) was used as storage medium. The folded donor tissues were grasped gently using angled nonappositional forceps (Charlie forcep; Moria) and inserted into the eye. Venting incisions were made to remove interface fluid.

Interface corneal deposits and/or debris have been described after laser in situ keratomileusis but have not been associated with DSAEK.4,5 In this case series, we describe 2 patients after DSAEK with the appearance of characteristic wavelike deposit accumulation at the donor-recipient interface in the immediate postoperative period.

Case 1. A 73-year-old female patient with Fuchs endothelial dystrophy and pseudophakia had DSAEK for bullous keratopathy. Preoperatively, the best spectacle-corrected visual acuity (BSCVA) in this eye was 20/70. On postoperative day 1, characteristic interface wavelike deposit accumulation was visible on slitlamp examination despite corneal edema. The deposits were limited to the interface between the recipient cornea and lenticular lamellar corneal graft, without anterior or posterior extension. There was no anterior chamber reaction noted. As the corneal edema resolved, the deposits became more prominent on slitlamp examination (Figure 1). Subjectively, the patient denied any associated symptoms. A short trial of intensive steroids (from 4 times initially to 8 times per day) was tried without any evidence of deposit appearance.

At the 3-month postoperative examination, the interface deposits remained stable. Despite complete resolution of the corneal edema, the patient's uncorrected and BSCVAs did not improve (20/70). One year after DSAEK, the interface deposits remained unchanged and there was no improvement in the patient's visual acuity.

Figure 1. Case 1. Slitlamp biomicroscopy revealed significant characteristic interface wavelike deposit accumulation.

Figure 2. Case 2. Interface wavelike deposits accumulate immediately (A) and 1 month after (B) Descemet stripping endothelial keratoplasty.
Case 2. An 88-year-old man with Fuchs endothelial dystrophy had DSAEK in the left eye for bullous keratopathy after cataract surgery and trabeculectomy in 1997. Preoperatively, the BSCVA in this eye was 20/200. On the first postoperative day, characteristic interface wave-like deposit accumulation was visualized on slitlamp examination despite corneal edema (Figure 2). A short trial of intensive steroids (from 4 times initially to 8 times per day) was tried without any evidence of deposit appearance. No evidence of interface fluid was found using anterior segment optical coherence tomography (Visante; Carl Zeiss Meditec, Dublin, California).

At the 1-year follow-up examination, interface deposits remained stable. Despite complete resolution of the corneal edema, the patient’s uncorrected and best spectacle-corrected visual acuity did not improve (20/200).

Comment. In this case series, we present a post-DSAEK complication that has not previously been described in the literature. Characteristic interface accumulation of wave-like deposits without anterior or posterior extension were present in both patients immediately after DSAEK. The patients’ uncorrected and best spectacle-corrected visual acuities remained unchanged (compared with preoperative measurements), even after corneal edema had resolved.

The morphologic features, distribution, and density of these particles remained unaltered throughout the 1-year postoperative observation period. The etiology of these deposits is uncertain. Initially, the possibility of an infectious etiology, intralamel keratitis, or the development of epithelial downgrowth at the interface space were considered. However, the immediate appearance of the deposits on postoperative day 1, wide extension of the deposits, absence of anterior chamber reaction, lack of improvement after an intensive course of steroids, and unaltered appearance at follow-up visits supported an noninfectious etiology and excluded the possibility of epithelial downgrowth.

Precipitates from the storage media may be a possible source of these deposits/debris. In addition, talc from surgical gloves has been associated with particle deposition. Another possible source is microkeratome or blade debris. Previous studies during laser in situ keratomileusis have demonstrated that the oscillating microkeratome and blade could produce debris that remains at the flap interface. However, these interface deposits did not significantly affect corneal wound healing and remained unreactive and stable during the follow-up periods. The uncorrected and best-corrected visual acuities of both patients were significantly affected, showing no postoperative improvement. In these cases, meticulous rinsing of the stromal interface after flap creation in the eye banks or before injection into the anterior chamber may minimize deposit accumulation.

A major limitation of this study is the lack of confocal or tissue microscopy analysis of the deposits. Future studies including these analyses are needed to elucidate the origin of these deposits.

In conclusion, interface wave-like deposits are an infrequent post-DSAEK complication that could affect patients’ final visual outcome. Surgeons and eye banks should be aware of the possibility of this complication after DSAEK.

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Clinical and Tomographic Features of Macular Punctate Outer Retinal Toxoplasmosis

Classic active ocular toxoplasmosis affects the full-thickness retina with associated vitreous reaction (“light in the fog” appearance).1,4 The observation and demonstration of deep retinal involvement in toxoplasmosis was first introduced by Gass2 in 1968. Once Gass introduced this concept, other variations on nonclassic retinal toxoplasmosis were described.3,5 Later that year, Friedman and Knox6 described new morphologic presentations of ocular toxoplasmosis affecting the retina. Punctate lesions localized in the outer (punctate outer toxoplasmosis) or inner (punctate inner toxoplasmosis) portions of the retina were first described by these authors. It was only in 1985 that Doft and Gass7 elucidated the outer variation of punctate toxoplasmosis and introduced the term punctate outer retinal toxoplasmosis (PORT). According to these authors, PORT is a subset of ocular toxoplasmosis that initially and primarily affects the outer retinal layers of the macular area, with only mild vitreous reaction. Typically, recurrent lesions occurred in this variant of ocular toxoplasmosis that affected adjacent areas of the macula, resolving spontaneously or forming fine granular gray-white dots. In their small cases series, symptomatic patients seemed to respond satisfactorily early in the course of the disease to antimicrobials and steroids. In this article, we describe 5 patients who developed punctate retinal lesions in the macular area in association with toxoplasmosis. In all 5 patients, diagnosis of ocular toxoplasmosis was supported by elevated serum levels of immunoglobulin to toxoplasma (IgG and IgM) and a favorable response to therapy. Ocular clinical and tomographic findings in these cases provided new insights into the