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, intralamelar 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, tare 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). The observation and demonstration of deep retinal involvement in toxoplasmosis was first introduced by Gass in 1968. Once Gass introduced this concept, other variations on nonclassic retinal toxoplasmosis were described. Later that year, Friedmann and Knox 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 Gass 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
pathophysiology of ocular toxoplasmosis.

Report of Cases. Case 1. A 17-year-old healthy girl was seen initially with blurred vision in the left eye (visual acuity [VA], 20/25). The right eye’s VA was normal (20/20). Results of biomicroscopy showed a mild vitreous reaction associated with an active punctate retinitis in the macula of the left eye. An optical coherence tomographic (OCT) line scan through the active lesion showed thickening and elevation of the retinal pigment epithelium (RPE) associated with minimal subretinal fluid in the macular area. Clinical suspicion of toxoplasmosis was confirmed by elevated serum levels of IgG (438 IU/mL; reference value, <2 IU/mL) and IgM (2.7 IU/mL; reference value, <0.5 IU/mL) to toxoplasma. Two weeks after treatment with an oral combination of trimethoprim (160 mg) and sulfamethoxazole (800 mg) (Bactrim F; Roche Pharmaceuticals, São Paulo, Brazil), 2 tablets per day for up to 4 weeks, and prednisone (20-40 mg/d) added 24 hours later, the lesion regressed and VA improved (20/20) (Figure 1). 

Case 2. A 11-year-old boy was seen initially with central blurred vision in the left eye (VA, 20/30). The right eye had normal VA (20/20). Results of biomicroscopy showed a mild vitreous reaction associated with active punctate retinitis in the macula of that eye. An OCT line scan through the active lesion showed attachment of the RPE-choriocapillary layer and the neurosensory retina over the lesion with inflammatory retinal infiltrate in the deeper retinal layers and minimal intraretinal fluid in the macular area. Clinical suspicion of toxoplasmosis was confirmed by elevated serum levels of IgG (626 IU/mL; reference value, <2 IU/mL) and IgM (1.7 IU/mL; reference value, <0.5 IU/mL) to toxoplasma. Two weeks after treatment with an oral combination of trimethoprim (160 mg) and sulfamethoxazole (800 mg) (Bactrim F), 2 tablets per day for up to 4 weeks, and prednisone (20-40 mg/d) added 24 hours later, the lesion regressed and VA improved (20/20) (Figure 2).

Case 3. A 10-year-old boy presented with blurred central vision in the right eye (VA, 20/50). The left eye had normal VA (20/20). Results of biomicroscopy showed a mild vitreous reaction associated with active recurrent punctate retinal lesions caused by presumed toxoplasmosis in the macula of that eye. Optical coherence tomography showed different patterns of retinal involvement of the lesions. Such patterns consisted of small punctate lesions showing attachment of the RPE-choriocapillary layer and the neurosensory retina and a large punctate lesion with evidence of inner retinal involvement. Clinical suspicion of toxoplasmosis was confirmed by elevated serum levels of IgG (513 IU/mL; reference value, <2 IU/mL) and IgM (2.7 IU/mL; reference value, <0.5 IU/mL) to toxoplasma. Two weeks after treatment with an oral combination of trimethoprim (160 mg) and sulfamethoxazole (800 mg) (Bactrim F), 2 tablets a day for up to 4 weeks, and prednisone (20–40 mg/d) added 24 hours later, the lesions regressed and VA improved (20/30). Six months after presentation, the lesions have re-
solved. Resolution of the previous active large lesion under the RPE was noted with OCT (VA, 20/20) (Figure 3).

**Case 4.** Recurrent vitreitis associated with active multifocal deep gray-white lesions caused by presumed toxoplasmosis was observed in a 10-year-old boy with blurred central vision in the right eye (VA, 20/400). The left eye had normal VA (20/20). Results of OCT through 1 active lesion showed thickening and elevation of the RPE. A partial posterior vitreous detachment with a thickened hyaloid was also observed in that eye. The clinical suspicion of toxoplasmosis was confirmed by elevated serum levels of IgG (1769 IU/mL; reference value, <2 IU/mL) and IgM (1.7 IU/mL; reference value, <0.5 IU/mL) to toxoplasma. Two weeks after treatment with oral combination of trimethoprim (160 mg) and sulfamethoxazole (800 mg) (Bactrim F), 2 tablets a day for up to 4 weeks, and prednisone (20-40 mg/d) added 24 hours later, resolution of the previous active lesions with formation of newly punctate lesions associated with mild vitreitis was noted in the left eye. Four months after initial presentation, a new site of retinitis was present in the parafoveal region of the eye. The patient returned only in the year 2005, presenting again with multiple recurring deep punctate lesions in the left macula. An OCT scan through the fovea showed a focal hyperreflectivity limited to beneath the RPE. A few months later, during that year, an area of classic toxoplasmic retinitis associated with a shallow posterior vitreous detachment was observed in the same eye. An OCT3 scan showed increased reflectivity in the inner retinal layers corresponding to the focal retinitis. Decreased reflectivity was observed in the RPE and photoreceptor layer secondary to the shadowing effect (Figure 5).

**Comment.** Ocular toxoplasmosis is a leading cause of posterior uveitis in humans. The typical ocular toxoplasmosis lesions are accompanied by an overlying vitritis, leading to the assumption that the inner retinal layers or full-thickness retina are involved in this disease.4 This article describes the clinical and tomographic findings of 5 patients with characteristic features of macular punctate outer retinal toxoplasmosis, a subset of ocular toxoplasmosis characterized by the presence of multifocal, punctate outer retinal lesions associated with little vitreous reaction.2,3 In all 5 patients, the diagnosis of toxoplasmosis was supported by elevated serum levels of immunoglobulin to toxoplasma (IgG and IgM) and a favorable response to therapy. Our findings suggest that OCT3 provides new insights into the pathophysiology of this subset of acquired ocular toxoplasmosis presentation. We wonder if the lesions we found were the anatomic representations of granuloma-like lesions (chorioretinitis) as a first line of immunologic defense against choroidal circulating infecting toxoplasm-
mic organisms (acquired infection). This was suggested in our cases 1 and 2 with OCT. The resultant deep punctate lesions may exudate into the retina and regress slowly or encyst in the subretinal pigment epithelial space. Following this proposed mechanism of disease, we believe that failure to control the infection can give the parasites access to inner portions of the retina (as seen in cases 3 and 4), causing temporary focal white inflammatory lesions (punctate inner retinal toxoplasmosis) or the more familiar and classic manifestation of toxoplasmic retinitis with adjacent vitreous involvement (case 5).

In 1985, Doft and Gass, Without the advantage of OCT, believed that the punctate changes in macu-
lar PORT represented focal outer retinal gliotic scars or encysted tissue forms of *Toxoplasma gondii*. Unless histopathology is obtained from these eyes in the active stages of the disease, OCT will be of great clinical use in investigation of the pathogenesis of macular PORT. Our experience with consecutive Brazilian cases with macular PORT indicates that they typically present with acute unifocal or multifocal gray-white lesions, largely confined to the macular outer retinal layers, with mild vitreous reaction. Such lesions resolve slowly, but frequently recur in adjacent areas of the macula. Macular PORT is commonly underreported and underdiagnosed as an inflammatory macular disease. The white dots that characterize macular PORT must be differentiated from similar deep gray-white changes that may occur in children and young patients with *Toxocara* infection, punctate inner choroiditis, multifocal choroiditis, multiple evanescent white dot syndrome, diffuse unilateral subacute neuroretinitis, and acute retinal pigment epithelitis. Unlike patients with these inflammatory diseases, those with macular PORT present serologic evidence of exposure to *T. gondii* and recurring focal white active lesions that may resolve or cause focal chorioretinal scars in the macula with little vitreous reaction. Recognition of the different presentations of toxoplasmosis is important because there is evidence that treatment of macular PORT may be effective.

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