

BULLOUS KERATOPATHY



Bruh, your cornea is "swole."

BY JACOB LANG, OD, FFAO, DIPL ABO

In a healthy eye, the corneal endothelium prevents the stromal layers from swelling with its tight junctions between endothelial cells. The metabolic processes it performs to maintain proper dehydration and optical clarity of the cornea also aid in the prevention of swelling.¹⁻⁴

Bullous keratopathy is characterized by stromal edema and anterior corneal bullae (Figure) rooted in the loss of corneal endothelial cell integrity and function. Clinically, patients with this corneal pathology present with decreased vision and ocular pain caused by the epithelial manifestation of the disease, specifically the ruptured and intact epithelial bullae. As for the patient shown, endothelial damage was presumed to have been caused by the multiple surgeries she underwent to control her glaucoma, including a Baerveldt shunt (Johnson & Johnson Vision).

In children, normal endothelial cell density is greater than 3,500 cells/mm². This density naturally declines with age to approximately 2,400 cells/mm² in adulthood. Edema occurs when the cell density drops below 1,000 cells/mm².⁵

Conditions or events that give rise to bullous keratopathy include Fuchs corneal endothelial dystrophy, viral endothelialitis, exfoliation syndrome, traumatic injury, and endothelial injury caused by intraocular procedures such as cataract or glaucoma surgery.^{2,5,6} Bullous keratopathy may occur in around 1% to 2% of patients undergoing cataract surgery, a nonsignificant number when you consider that approximately 10 million patients undergo cataract surgery worldwide each year.⁵

With the increase in intraocular implants such as those used in microinvasive glaucoma surgery, and with the anterior chamber now being used as a depot for medication, it is more important than ever that optometrists stay vigilant in monitoring the corneal endothelium for abnormal or expedited cell loss.⁶

It is notable that patients with dry eye disease also have an accelerated rate of corneal endothelial cell loss. Those with lower subbasal nerve density, in particular, are at higher risk for accelerated endothelial cell loss.⁷ This observation stresses the importance and role of the neurosensory system in corneal disease, including but not exclusive to dry eye disease.

Keratoplasty is the primary treatment option for most patients with bullous keratopathy. The surgical strategy has

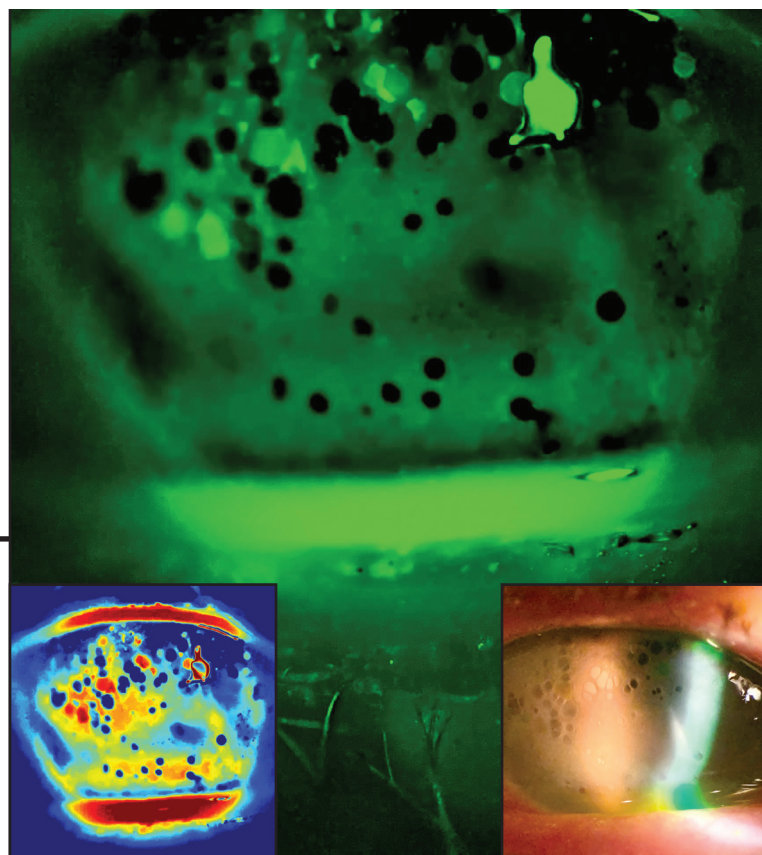


Figure. Elevated corneal bullae demonstrated with fluorescein image. Advanced Ophthalmic System's fluorescein thickness analysis allows further investigation (left inset). Corneal edema and bullae in bullous keratopathy (right inset).

evolved from full thickness penetrating keratoplasty to lamellar, endothelium-only strategies such as Descemet-stripping automated endothelial keratoplasty and Descemet membrane endothelial keratoplasty.⁵ Further evolution now includes non-transplant options such as Descemet stripping only, or DSO.⁴ The use of a rho-kinase inhibitor as an endothelial "encourager," in conjunction with surgical endothelial cell implantation procedures or as an independent therapy, has also shown promise.^{4,8,9} ■

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