What is diabetic keratopathy and its treatment?

Diabetes also affects corneal morphology, metabolism and physiology that result in clinical abnormalities that are referred to as diabetic keratopathy. These include epithelial defects, recurrent epithelial erosions, delayed reepithelialization, slower wound repair, increased epithelial fragility, reduced sensitivity, increased auto-fluorescence, altered epithelial and endothelial barrier functions, ulcers, edema, and increased susceptibility to injury due to the popularity of corneal refractive surgery and increase in cataract surgery, including LASIK among diabetics. Loss of corneal sensitivity is linked to the development of DED because the corneal nerves regulate tear volume through their ability to detect osmotic changes in the tear film. More than 70% of diabetics develop diabetic keratopathy and demonstrate some morphological change of their cornea and they have an increase in DED.

Tight blood sugar control can reduce the severity of diabetic keratopathy. Specific medical treatments have focused on the use of various growth factors, cytokines, opioid growth factor antagonists, and immunosuppressives -- all without significant clinical success. Corneal epithelial and endothelial cells contain aldose reductase and both laboratory and pilot clinical studies have shown that aldose reductase inhibitors can beneficially treat the adverse diabetic changes observed in the corneal epithelium, endothelium, and nerves. Small clinical studies have shown that these include reduced Superficial Punctate Keratitis (SPK), surface defects, changes in epithelial cell morphology, improvement of corneal sensitivity, reduced corneal endothelial cell death and persistent corneal edema. These changes significantly increase in diabetics following cataract surgery.