We describe a patient with systemic graft-versus-host disease who developed a nonhealing epithelial defect after cataract surgery that healed on cessation of a topical nonsteroidal antiinflammatory drug (NSAID) (ketorolac). The patient developed a central corneal perforation in the fellow eye while on a new NSAID formulation (nepafenac) after routine cataract surgery. Our case suggests that new topical NSAIDs may be similar to older NSAID formulations in promoting corneal melting in patients predisposed to poor epithelialization and corneal wound healing.

**CASE REPORT**

A 56-year-old woman with a history of systemic graft-versus-host disease following allogenic stem-cell transplantation for acute myelogenous leukemia had phacoemulsification with posterior chamber intraocular lens (IOL) implantation in the left eye. Postoperatively, she was placed on topical prednisolone 8 times a day, moxifloxacin 4 times a day, and ketorolac 4 times a day. She developed a persistent paracentral epithelial defect that healed with cessation of the ketorolac and tapering of the prednisolone. The patient then had uneventful phacoemulsification with posterior chamber IOL implantation in the right eye. Postoperatively, she was placed on topical prednisolone 4 times a day, moxifloxacin 4 times a day, and nepafenac 3 times a day. She presented 2 weeks after surgery with a central corneal melt and perforation in the right eye. Attempts to seal the defect with cyanoacrylate glue were unsuccessful so emergency corneal transplantation was performed.

**DISCUSSION**

Nonsteroidal antiinflammatory drugs exert their effect by inhibiting the formation of prostaglandins from arachidonic acid, a conversion mediated by the enzyme cyclooxygenase. By selectively blocking cyclooxygenase activity, NSAIDs shunt arachidonic acid to the lipoxygenase pathway, which results in the formation of leukotrienes in higher than normal levels. Leukotrienes are potent neutrophil chemoattractants as well as stimulators of neutrophil degranulation. The neutrophil granules contain powerful collagenases that may play a crucial role in the development of NSAID-related corneal melts and perforations. Our case suggests that new topical NSAIDs may be similar to older NSAID formulations in promoting corneal melting in patients predisposed to poor epithelialization and corneal wound healing.

**REFERENCES**