Nepafenac-associated corneal melt

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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.

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Nonsteroidal antiinflammatory drugs (NSAIDs) are commonly used perioperatively and postoperatively to prevent intraoperative miosis as well as postoperative pain and inflammation. However, NSAIDs have a side-effect profile that can be damaging to the eye. Previous reports of keratitis, ulceration, and perforation associated with NSAIDS have focused on older formulations such as ketorolac and diclofenac, as well as a newer NSAID, bromfenac. Risk factors for adverse corneal events in these studies included systemic inflammatory diseases, concurrent topical steroid use, and epithelial keratopathy. 1-3 We report a case of a full-thickness corneal melt following uneventful cataract surgery in a patient with systemic graft-versus-host disease who was treated with topical NSAIDs.

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

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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.

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