CORNEAL MELT FOLLOWING LIFITEGRAST USAGE: A CASE REPORT

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PURPOSE

We report the case of a patient regularly taking ketorolac tromethamine who developed vision-threatening bilateral corneal melting upon initiation of lifitegrast ophthalmic solution. To date, there have been no reported cases of corneal melt with lifitegrast ophthalmic solution.

METHODS

A 72-year-old female presented with ocular pain and decreased visual acuity for 3 days. The patient's past medical history was significant for CREST syndrome and Sjögren syndrome. Past surgical history was significant for bilateral CE/IOL 10 years prior to presentation. The patient had been followed for several years for moderate dry eye. She had been seen 1 month prior to presentation (May 1, 2017) with Schirmer's of 7 mm OU as well as moderate PEEs OU. She had previously been treated with aggressive lubrication with preservative-free artificial tears and cyclosporine BID OU (which had been discontinued several years prior). The patient was seen the week prior to presentation, and there had been minimal change in her dry eye condition, so she was started on lifitegrast 1 gtt BID OU in addition to continuing the ketorolac tromethamine 1 gtt qid OU. She noted a painful, burning sensation in both eyes, which began 7 days prior to presentation, commensurate with the initiation of lifitegrast. The patient had previously been taking ketorolac tromethamine for many months without significant side effects. Upon instillation of lifitegrast, she noticed immediate worsening of the ocular pain and a decrease in visual acuity. At initial presentation on June 3, 2017, visual acuity was 20/150 in the right eye and counting fingers at face in the left eye. On examination, she had corneal melting OU, a corneal ulcer OD, and corneal perforation OS as well as significant PEEs OU. Cyanoacrylate glue and bandage contact lens were placed over the perforation. She was instructed to discontinue ketorolac and lifitegrast gtt. The patient was started on moxifloxacin q2h OU as well as aggressive preservative-free lubrication. One month from initial presentation, vision is counting fingers at 3 feet OD and counting fingers at 2 feet OS. Her corneal examination had stabilized; exam showed healed ulcers with intact epithelium and residual dense central scarring OU. Her dry eye disease has remained stable with preservative-free artificial tears, and on last exam she had mild dryness OU.

RESULTS

Keratolysis, or corneal melt, is most frequently associated with preexisting tear film disease including Sjögren syndrome or collagen vascular disease. Severe corneal melt associated with the use of topical nonsteroidal anti-inflammatory drugs has also been reported. It has been theorized that matrix metalloproteinase expression was the cause of corneal melt. This patient had long-standing CREST syndrome; the most common ocular manifestations of this systemic disease are eyelid skin change and keratoconjunctivitis sicca. The diagnosis of Sjögren syndrome also predisposes the patient to keratoconjunctivitis sicca; at its most severe stage, the disease process can cause corneal ulceration, perforation and residual scarring. This patient was not on any systemic immunosuppressive medication for at least a year prior to presentation. This patient's dry eye had been stable on ketorolac drops until 1 week prior to presentation. The rapid ulcer formation and subsequent perforation corresponded to the initiation of lifitegrast drops. There have been previously reported rare cases of corneal melt secondary to topical ketorolac. Lifitegrast is a newer drug, and, to date, there have been no reported cases of corneal melt with its use prior to this report. This drug is an immunomodulator that binds to cell surface protein found on T cells (LFA-1) and blocks LFA-1 and ICAM-1 interactions, which is understood to downregulate inflammation mediated by T cell lymphocytes. The patient has an autoimmune disease that causes immunosuppression, which may have worked concurrently with the drops to promote keratolysis. The severity and aggressive development of corneal melt and subsequent perforation with initiation of this new medication suggests that lifitegrast contributed to the corneal melt in a previously stable patient. It may also suggest that adverse drug-drug interaction between the two drops may have been the cause of the corneal melt. There may be a necessity of further studies in relation to the use of lifitegrast in already immunosuppressed patients such as this case as well as studies using both lifitegrast and topical NSAID drops concurrently.

CONCLUSION

The patient's comorbid rheumatologic conditions, namely CREST syndrome and Sjögren syndrome, are confounding variables in this case. There is a known association between rheumatologic diseases and corneal melting, which does not implicate the use of topical ophthalmic drugs. Therefore, it is difficult to fully attribute the cause of corneal toxicity in this case to adverse effects of ophthalmic drugs. However, the rapid onset of symptoms upon initiation of lifitegrast, in addition to the lack of prior history of corneal melt, raises the possibility that lifitegrast initiation or drug-drug interaction with ketorolac caused corneal melt in this patient. To our knowledge, this is the first reported case of corneal melting associated with the use of lifitegrast ophthalmic solution.