Acanthamoeba is the causative agent of a painful, sight-threatening keratitis. The first case of Acanthamoeba keratitis was reported in 1972 in the USA and soon after, it was reported in the UK in 1974 (Nagington et al., 1974). Ulcerative keratitis due to Acanthamoeba is often associated with the improper use of contact lenses, but it is also reported in non-contact lens wearers (Sharma et al., 2000). At present, treatment involves hourly topical application of a mixture of anti-Acanthamoeba drugs including 0.02% polyhexamethylene biguanide or chlorhexidine for up to a week. The hourly drops may be reduced after 48 h to alleviate the epithelial toxicity, but treatment continues for up to a year (Clarke et al., 2012).

In a recent report by Agahan et al. (2009), it was demonstrated that three patients with Acanthamoeba keratitis were treated successfully without any anti-Acanthamoeba drugs. The patients had no history of contact lens wear and were admitted due to intolerable eye pain and blurring of vision. Ocular examination revealed corneal ulceration and stromal infiltrates. The patients were reportedly diagnosed with Acanthamoeba keratitis (Agahan et al., 2009). All patients were treated for photophobia with anticholinergics (atropine). In ophthalmologic practice, atropine is commonly used for ciliary muscle paralysis to block accommodation and to prevent reflex constriction of pupillary muscle. For inflammation and pain, the patients were given topical diclofenac sodium (0.1%) every hour for up to a week. The patients responded well to this treatment and the M1 receptor thus should prove highly effective in the treatment of this blinding infection.

Additionally, we propose amlodipine as a substitute anti-inflammatory agent (Kataoka et al., 2004) for diclofenac sodium eye drops. The anti-inflammatory effects of amlodipine are probably mediated by the inhibition of monocyte chemokine protein-1, tumour growth factor-1, the Rho pathway and oxidative stress (Kataoka et al., 2004). Moreover, our recent findings showed potent amoebicidal properties of amlodipine (Baig et al., 2013), and thus its use is likely to lead to dual effects in the successful management of Acanthamoeba keratitis.

In conclusion, we suggest the use of the selective muscarinic antagonist dicyclomine rather than atropine, as well as topical amlodipine to substitute for diclofenac sodium in cases of Acanthamoeba keratitis. The proposed use of these drugs needs to be tested in clinical trials to prove their efficacy in amoebic keratitis.

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