Ofloxacin induced multiple fixed drug eruptions – A case report

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ABSTRACT
Adverse drug reactions are the major hazards of modern medicine. Among all these, cutaneous drug reactions are given utmost importance as they are sometimes life threatening. Ofloxacin is a first generation fluoroquinolone and these class of drugs can cause Fixed drug eruption , which is a adverse reaction , is commonly seen with antimicrobials , analgesics and anticonvulsants etc. In the pathogenesis intraepidermal CD8+ T cells play a major role and may represent double-edged swords of the skin immune system with protective and destructive capacity. Here we report a case of a 38 year old female patient with Multiple Fixed Drug Eruptions due to Ofloxacin administration.

Key Words: Fixed drug eruption , Ofloxacin , Adverse reaction.

INTRODUCTION
Ofloxacin is a fluorinated quinolone which is an intermediate between ciprofloxacin and norfloxacin in activity against gram negative bacteria , but it is comparable to or more potent than ciprofloxacin for gram positive organisms and certain anaerobes.It is particularly suitable for chronic bronchitis and other respiratory or ENT infections[21].Fixed drug eruptions(FDE) are known to arise from a variety of medications such as analgesics , anti convulsants , sedatives , antifungal medications and antibiotics[1][2].Among antibiotics , trimethoprim/sulfamethoxazole and tetracyclins are most commonly associated with FDE , but rarely fluoroquinolones result in FDE[3].The prevalence of drug eruptions has been reported in the range of 2-5% . Fixed drug eruptions may account for as much as 16-21% of all cutaneous drug eruptions[4].However , there have been reports of cases in which ciprofloxacin and norfloxacin has been implicated in fixed drug eruptions , but very few were reported with Ofloxacin.Here we present a case of bullous fixed drug eruptions following treatment with ofloxacin as this type of presentation is very rare.

CASE SUMMARY
A 38 yr old Indian female patient who is suffering from upper respiratory tract infection went to a private practitioner and she was prescribed with ofloxacin 200mg twice daily. Within few hours of
first dose administration of ofloxacin patient experienced itching and burning sensation on few areas of hands and feet. These areas turned into purplish plaques upon the administration of second dose. She still continued taking the drug and experienced erosions on her lips which are prominent on her lower lip, swelling of eye and few more new lesions on her limbs. Within 48 hours her lesions became painful and now few are filled with pus. Upon further questioning it was revealed that patient has a similar history when she has taken the same drug 13 months back for the treatment of upper respiratory tract infection. Lesions were developed after 48hrs of ofloxacin administration in the past which were few and are asymptomatic , so no treatment was sought. But on the current admission lesions developed within hours of drug administration. For the present painful lesions she consulted a private dermatologist and the diagnosis of fixed drug eruptions was made. On the current consultation she was prescribed with topical hydrocortisone cream and her antibiotic regimen was stopped. These lesions were improved over 2 weeks , leaving post inflammatory hyperpigmentation.

**DISCUSSION**

In 1889, Bourns described a series of sharply demarcated hyperpigmented lesions on the lips and tongue of a patient who had recently ingested 20g of antipyrine. A few tears later , Brocq coined the French term , eruption erythematopigmentee fixe , from which we derive the term , fixed drug eruption[^5].Fixed drug eruption is characterised by the sudden onset of round and/or oval, oedematous, dusky-red macules on the skin and/or mucous membranes accompanied by burning and/or itching. The acute phase is usually followed by residual pigmentation[^6].Typically FDE begins with a sharply demarcated oval of circinate macule[^7].Less commonly FDE appear as plaques , bullae or eruptions and may koebnerize[^8].It characteristically
recurs in the same sites each time a particular drug is taken. FDE is usually solitary in the initial attack, but with each subsequent exposure, the number of involved sites may increase and pre-existing ones may increase in size. The lesions usually develop within 30 minutes to 8 hours of taking a drug. The lesions are often painful, clearly demarcated oval or round erythematous plaques, becoming violaceous 1-2 days later. As healing occurs, after about 1 week, crusting and scaling are followed by a persistent dusky brown colour. The lesions are often painful, clearly demarcated oval or round erythematous plaques, becoming violaceous 1-2 days later. As healing occurs, after about 1 week, crusting and scaling are followed by a persistent dusky brown colour. The genetalia, lips and hands are among the most commonly affected sites, although any site may be affected including conjunctivae and oropharynx. The diagnosis of fixed drug eruption is not always easy; as in the case of nonpigmenting fixed drug eruptions, which do not have any residual hyperpigmentation. The development of molecular biology may help to unfold the exact pathogenesis of fixed drug eruptions but as of now the reason for their recurrence on the same sites is still unknown. Importantly, the causative drug and cross-reactants should be avoided to prevent recurrence. Till date, rechallenge is the most reliable method of identifying causative drugs, but increasingly the use of skin tests has gained the attention of many investigators. A genetic susceptibility to developing a fixed drug eruption with an increased incidence of HLA-B22 has been reported. Pathogenesis of FDE is unknown, antibodies, antibody-dependent cell mediated cytotoxicity, and serum factors have been implicated. Recent findings shows that intraepidermal CD8+ T cells with an effector-memory phenotype resident in fixed drug eruption lesions have a major contributing role in the development of localized tissue damage. Activation of these CD8+ T cells is sufficient for triggering the lesion, however, but not sufficient to cause extensive tissue damage observed in the fully evolved lesions; additional recruitment of CD4+ and CD8+ T cells to a specific tissue site would also contribute to the late stage of lesion development. The influx of regulatory T cells into the epidermis observed in fully evolved lesions would serve to limit harmful immune reactions. Consistent with this, positive patch test reactions are only observed at the site of previous lesions harboring significant numbers of intraepidermal CD8+ T cells. The peak incidence of FDE is 21-30 years, although any age may be affected, ratio of male:female incidence is generally equal. Genetic predisposition to FDE appears to occur in individuals with a family history of diabetes mellitus, atopy and drug allergies. Causality assessment for this ADR was made using Naranjo causality assessment scale and the naranjo score was found to be 9, thus ADR was classified as definite one. To the best of our knowledge there are only 3 previous reports of FDE to this drug ofloxacin.

CONCLUSION
Because of widespread use of fluoroquinolones, it is important to consider these as possible etiologic agents of FDE. Therefore, one must include ofloxacin also in the etiologic differential when making the diagnosis of FDE.

ACKNOWLEDGEMENT
We take this golden opportunity to express our thanks to Dr. Rama Rao Nadendla, Principal, Chalapathi institute of pharmaceutical sciences, and also Raghu Ram V and Shaik Shafiya, Assistant professors of chalapathi institute of pharmaceutical sciences, for providing necessary facilities, valuable guidance and continuous encouragement.

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