

CONTACT POINTS

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Reproducible identification of the causative drug of a fixed drug eruption by oral provocation and lesional patch testing

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Key words: cutaneous adverse drug reaction; fixed drug eruption; metamizol; positive patch test.

Non-steroidal anti-inflammatory drugs are among the most common causes of fixed drug eruptions (FDEs) (1), identification of the cause of which is essential to their management. Although re-challenge remains the most reliable method, topical provocation on sites of previous lesions has increasingly been adopted (2).

Case Report

A 57-year-old man from India received combined oral treatment with diclofenac, metamizol, and tetrazepam along with lansoprazole for lumbago. 3 days after initiation of this therapy, widespread sharply marginated, bluish, oval plaques several centimetres in diameter, with an erythematous edge occurred on both legs and the trunk. These lesions were itchy and painful. The patient had been continuously on glimepirid since 1998 for non-insulin-dependent diabetes mellitus (NIDDM).

Therapy with cetirizine 10 mg/day and prednisone 40 mg/day was initiated, and the patient was referred

to our department (Fig. 1a). We diagnosed an FDE, most likely caused by the analgesic therapy, which was therefore discontinued. Pain control for the lumbago was achieved by tramadol monotherapy, and the pruritus was controlled with clemastin 3 × 1 tablet/day. The lesions cleared slowly within the next 12 days on systemic therapy with prednisone, which was initially increased to 80 mg/day. At the time of discharge,

therapy consisted of 8 mg/day prednisone and 2 × 100 mg/day ranitidine; this medication was discontinued shortly thereafter. Glimepirid continued to be taken by the patient continuously.

Allergy tests were performed 8 weeks after discharge, in the complete absence of any immunosuppressive treatment. Scratch tests with all drugs the patient had received remained negative. Oral provocation

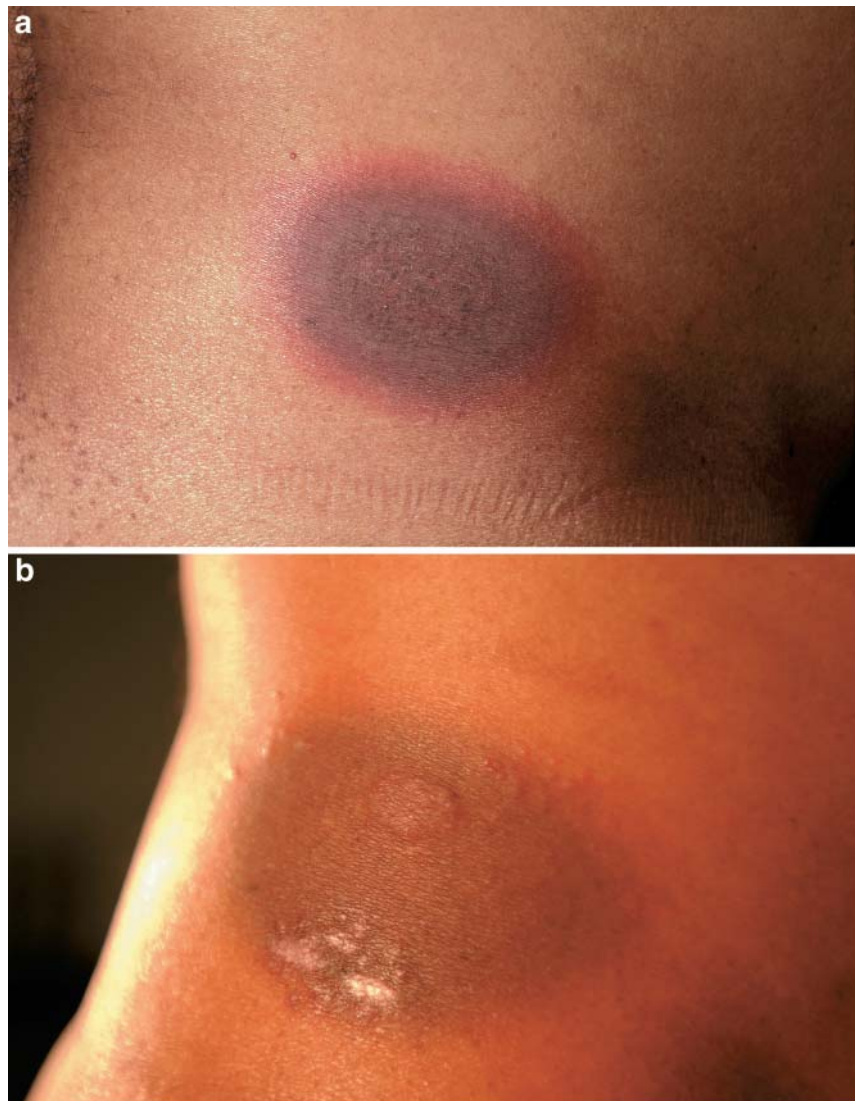


Fig. 1. Clinical appearance of the FDE on the patient's trunk (a) at the time of admission and (b) 72 h after a lesional patch test with metamizol at the same site.

was well tolerated with regard to tetrazepam and lansoprazole. 5h after exposure to 20 drops metamizol (Novalgin®,) a flare-up of the FDE occurred, with erythema developing at previously affected sites.

Lesional open patch tests were performed 2 months after this episode, as described (3). Metamizol was tested by topical application of the same metamizol-containing medication used for the initial treatment for lumbago, which had already yielded a positive result in the oral provocation (Novalgin). These tests proved negative for all drugs but metamizol, which triggered the development of papulovesicles at the test site (Fig. 1b). The allergy tests were completed by oral provocation with diclofenac, which was also well tolerated.

Comment

Identification of drugs causative of FDE is usually established by re-challenge (4), but patch tests may become a safer alternative (5). Metami-

zol is among the drugs most frequently implicated as a cause of FDE (6). In the patient reported here, identification of the causative drug was particularly difficult because there were several potential candidates besides metamizol, including lansoprazole (1). Oral provocation documented the causative role of metamizol in this case. Interestingly, the results of the re-challenge could be reproduced by lesional open patch testing. To our knowledge, this is only the 2nd documented case of successful patch testing for metamizol (7). Noteworthy and contrary to the initial report (7), this did not precipitate any generalized reaction. This case report underlines the feasibility of lesional open patch tests in the diagnosis of FDE.

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