Hidradenitis suppurativa (HS) is a chronic inflammatory disease for which long-term therapy, often with topical medication, is necessary. Reports of allergic contact dermatitis (ACD) to topical treatments in patients with HS are rare (1). We report here a case of ACD caused by topical clindamycin.

CASE REPORT

A 36-year-old woman with longstanding mild Hurley stage II HS, and no history of atopy or allergy, was referred for treatment of an acute and intensely itchy dermatitis characterized by redness, swelling and blister formation localized symmetrically on the groin and genitalia. A flare-up occurred after 2 days of treatment with clindamycin phosphate (Dalacin® 10 mg/ml cutaneous emulsion) applied to the affected areas twice daily. Analysis of a skin lesion biopsy demonstrated histological features of contact dermatitis and the patient was treated successfully with betamethasone with clioquinol cream.

Patch testing was performed with the Swedish baseline series, a corticosteroid series, a topical treatment series, clindamycin phosphate (10% pet.), and the patient’s own intimate care products. Patch test readings were performed on day (D) 3 and D7 according to the European Society of Contact Dermatitis (ESCD) guidelines (2) and showed positive reactions to clindamycin phosphate (+++), formaldehyde (+), carba mix (+), and linalool/limonene (+). Subsequent patch testing with the rubber lining of the patient’s underwear “as is” and with an ultrasonic bath extract were negative. The patient’s own intimate care products, including the cutaneous emulsion of clindamycin phosphate (Dalacin®), were tested for the presence of formaldehyde by the chromotropic acid method: none were positive.

DISCUSSION

HS is a chronic, recurrent, and painful disease, with an estimated prevalence of 1% worldwide (3). Topical therapy is the mainstay of treatment for early-stage HS, and most patients are using or have used topical clindamycin in the past (4). Commonly, clindamycin is used repeatedly for long periods of time and often on skin areas of occlusion (e.g. axillae, inframammary folds, and inguinal). Moreover, the substance is applied to inflamed skin characterized by barrier disruption. Nevertheless, to the best of our knowledge, there is only one other case report of ACD caused by topical clindamycin in a patient with HS (1).

The current patient also tested positive for formaldehyde, carba mix and oxidized linalool/limonene, which are common causes of contact allergy; however, none of these were evaluated as clinically relevant. In retrospect, it is impossible to know whether the patient was sensitized to these substances during her acute dermatitis caused by the topical treatment with clindamycin, or if these sensitizations had occurred at an earlier stage, perhaps due to the disrupted skin barrier owing to her HS. Upon analysis the patient had, however, no current exposures of relevance to formaldehyde, rubber, or perfume.

In general, reports of clindamycin allergy are remarkably rare despite its widespread use for treating common skin diseases, such as acne vulgaris, for more than 40 years (5). Thus, clindamycin is regarded as a weak allergen, which could be the sole explanation for the few reports. However, it is possible that the diagnosis of ACD is missed since symptoms of contact allergy could be misinterpreted as a side-effect to the treatment or possibly even mimic a flare-up of the patients’ skin disease (6). Therefore, patch testing may be particularly relevant in patients with HS receiving topical treatment, such as clindamycin, when an eczematous component is suspected.

The authors have no conflicts of interest to declare.

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