A 38-year-old Singaporean Chinese male presented with a 1-month history of an intensely pruritic eruption over his upper limbs. He had no history of medical problems, atopy or exposure to contactants and had not taken any medication. He was systemically well and did not have symptoms of infection during or prior to the onset of the rashes.

Examination revealed widespread erythematous papules over his forearms and arms (Figs. 1 and 2). The papules had a shiny and translucent tinge, indicative of early vesiculation. Further examination revealed scaling of his soles. The patient had not reported the condition of his soles as it was asymptomatic and he had been having it on-and-off for years. A subsequent fungal scrapping of the soles revealed the presence of mycelium.

What is your diagnosis?
A. Endogenous eczema
B. Autoeczematisation from underlying tinea pedis
C. Polymorphous light eruption
D. Lichen planus
E. Viral exanthem

Discussion
A diagnosis of autoeczematisation secondary to tinea pedis was made. The patient was treated with mometasone ointment for the rashes over his forearms and terbinafine 250mg o.m. and miconazole cream for the tinea pedis. The eczematous rashes resolved within a week and the fungal infection resolved after 3 weeks of treatment. There was no recurrence of the rashes over the arms or occurrence of similar rashes over other parts of the body in the subsequent months.

The term autosensitisation dermatitis was first coined in 1921 by Whitfield to refer to a phenomenon where an acute dermatitis develops at a site distant from an inflammatory focus but which cannot be explained by the primary inflammation. Id-reaction is another term used for the condition, which comprises a diverse clinical picture ranging from a morbilliform appearance to an urticarial eruption to a generalised, papulovesicular dermatitis (as was present in this patient). The following sites, in decreasing order of frequency, are typically involved: forearms, thighs, legs, trunk, face, hands, neck and feet. In our patient, the papulovesicular morphology and the location of the rashes on the arms provided clues to the diagnosis.

Autoeczematisation occurs most commonly in stasis eczema, in which at least 37% of patients experience one episode of the disease. It may also be present in other conditions including, but not limited to, tuberculosis, histoplasmosis, dermatophytic or bacterial infections. The pathogenesis of this condition is unknown but is thought to be due to an immunologic or non-immunologic stimuli (in this case tinea pedis) resulting in release of a variety of cytokines. This leads to hypersensitivity of uninvolved skin and resultant cutaneous reaction to non-specific and otherwise innocuous stimuli.

The diagnosis of autoeczematisation is predominantly a clinical one, with emphasis on identification of the primary inflammatory focus. Histological examination is often unhelpful with non-specific findings of spongiotic epidermal vesicles with a superficial, perivascular lymphohistiocytic infiltrate of the dermis which may contain scattered eosinophils.

Autoeczematisation is a common, but often under-diagnosed condition that clinicians need to be aware of. Treatment involves a 2-pronged approach for
which treatment of the underlying cause (in this case with antifungal agents) is a cornerstone. In addition, glucocorticoid treatment will also often hasten the healing process. In our patient’s case, a potent topical agent was sufficient as the lesions were in the early stage. If however the lesions are more extensive, a short course of oral glucocorticoids such as prednisolone can often result in a dramatic clinical improvement. Adjunctive treatment such as potassium permanganate compress for weepy lesions and first generation oral anti-histamines to ameliorate itch can also often be helpful.

REFERENCES


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