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Pompholyx: A Still Unresolved Kind of Eczema

Despite its frequency, pompholyx or dyshidrotic eczema presents many unresolved problems of aetiology, pathogenesis and therapy. As its invariable histopathological picture of spongiotic dermatitis indicates, pompholyx is a non-specific reaction somehow related to the particular anatomy of the palmoplantar skin [1]. It may be, therefore, a manifestation of different pathologies such as atopic and contact dermatitis [2–4], oral allergy to metals [5, 6] and dermatophyte infection [7]. Rarely, even pemphigus and bullous pemphigoid may masquerade as dyshidrotic eczema [8, 9].

A recent paper has reviewed the available evidence and provided some new ones [10]. Age does not matter, as the disease is equally represented in the 16–40 and 41–65 age groups. Hands and feet may be involved in different ways. Pompholyx may affect either one hand or foot, both hands or both feet, or simultaneously hands and feet. Most patients have two homologous areas affected. Atopy is present in 50% of patients. The IgE serum level is frequently increased, irrespective of the family or personal history of atopy. Pompholyx may well be the first manifestation of atopy.

Contact allergy to nickel plays an important role, especially considering that routine diagnostic procedures (patch tests with nickel sulphate) are probably insufficient to detect all of the allergic subjects. The high perspiration rate of the palms and soles favours a high concentration of metal salts (i.e. nickel) in these regions, which could be sufficient to cause an eczematous reaction. Further evidence comes from the oral provocation test with nickel salts: positivity is interpreted as a marker of latent hypersensitivity. In such cases, low-nickel diets have significantly improved the number and severity of pompholyx relapses. Other double-blind studies, however, did not find such a significant correlation. In subjects positive in the oral provocation test there

may be an immune-complex-mediated reaction or a delayed hypersensitivity with false-negative patch tests, or even a non-immunological reaction. In the last instance, nickel salts could trigger a further unknown pathogenetic factor.

Other contact allergens have been reported as significantly associated with pompholyx, namely carba-mix, diaminodiphenylmethane, cobalt chloride and fragrance mix.

Prick and intradermal tests with staphylococcal and other pyogenic bacteria antigens, dermatophytes and candida have frequently been found positive. They are probably related to the prolonged contact with these microorganisms and not necessarily to actual infection. Likewise, house dust and pollens give frequent positive tests, but they have no apparent immunological correlation with an eczematous disease like pompholyx (type I vs. type IV hypersensitivity reaction). Acari may be a possible exception that deserves further investigation by patch tests with concentrated acaridic extracts.

Interdigitoplantar dermatophytosis has been long ago recognized as a cause of pompholyx through an 'idic' mechanism, but no convincing evidence has been provided [7]. In fact, dyshidrotic patients have tinea pedis and macerative or desquamative interdigital lesions more often than controls, irrespective of the presence of fungi. The incidence of fungal infection does not significantly differ considering only patients and controls with interdigital lesions. A specific antifungal therapy has been shown to cure pompholyx in less than 1/3 of patients who showed also a vesicular reaction to intradermal trichophytin. Such patients worsened at the beginning of the treatment but experienced a complete regression at its end (true 'idic' pompholyx). In other patients, however, the course of pompholyx has not been influenced by treating the concomitant tinea pedis. The presence of tinea pedis in a dyshidrotic patient,

therefore, cannot be necessarily regarded as a causal factor. Frequent interdigital macerative lesions of pompholyx can simply favour the development of fungal infection.

Other cases, in which fungi have been found directly in the vesicular fluid, should be more correctly defined as having pompholyx-like tinea pedis.

Environmental factors are sometimes considered by both dermatologists and patients as able to worsen the disease, namely changing season, heat, humidity and cold. None of them, however, proved to be the only responsible one in the same patient.

Emotional hyperhidrosis may increase the reactivity of palms and soles, and stressful episodes may lower tolerance

to pompholyx, in the same way as it occurs in other atopy-related diseases. No evidence has ever been provided, however.

In conclusion, the role of the aforementioned factors is still unclear. Different noxae may produce the same clinical and histological picture. Several antigens, either derived from foods, such as nickel, or directly present on the skin, such as contact haptens or fungi, may play a role. They may have a particular tropism towards proteins of the stratum lucidum of palms and soles and, in predisposed subjects, they may bind to tissue receptor sites starting the eczematous reaction.

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