

Lymphomatoid contact dermatitis to an exotic wood: a very harmful toilet seat

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We report the case of a 58-year-old man who experienced a 2-year history of arciform rash of the buttocks associated with an intense pruritis. The lesion worsened despite several multipotent topical steroid courses. Punch biopsy with immunohistochemistry of the lesion was suggestive of lymphomatoid contact dermatitis, and extensive screening for cutaneous T-cell lymphoma (CTCL) remained totally negative. Lymphomatoid contact dermatitis is an inflammatory disease that can clinically simulate malignant lymphoma. Although the course of the disease is considered to be benign, it should be regarded with attention as it has been hypothesized that the condition could be a precursor of CTCL.

Key words: Lymphomatoid contact dermatitis; exotic wood.

Lymphomatoid contact dermatitis is a rare entity that refers to the phenomenon of allergic contact dermatitis, producing clinical, histological, or immunohistochemical findings mimicking

a cutaneous T-cell lymphoma (CTCL) (1). The pathogenic mechanism of the disease is still unclear but is probably underlined by a chronic inflammatory reaction involving lymphoid cells (2). We report the case of an unexpected lymphomatoid contact dermatitis to an exotic wood species (Teak, *Tectona grandis* L.).

Case Report

A 58-year-old man presented to our department with a 2-year history of a persistent, intensely itching, arciform rash of the 2 buttocks. The lesion improved each time after the use of a multipotent topical corticosteroid but flared up after stopping treatment. Clinical examination revealed a sharply well defined, slightly scaly, erythematous arciform lesion involving the 2 buttocks (Fig. 1a). In spite of concern about CTCL, 2 skin punch biopsies were performed and completed with immunohistochemistry examination. Moreover, the rest of the physical examination was unremarkable and extensive search for an extracutaneous localization for CTCL remained totally negative.

The biopsies showed an acanthotic epidermis with parakeratosis and a superficial infiltrate showing focal interface changes. There was exocytosis of lymphocytes into the epidermis, mild spongiosis, and mild cytological atypies. The immunohistochemistry showed that most of the cells in the infiltrate were CD3 and CD4 positive and failed to reveal the presence of CD 30 and CD 56. Finally, T-cell receptor gene analysis did not identify the presence of a clonal population of T cells in the 2 biopsies. Concurrently, unilesional mycosis fungoides raised-up as a hypothesis.

3 weeks later, when the patient came back to discuss biopsy results, he acknowledged having changed his domestic toilet seat from some 2 years now. Therefore, the decision to undergo patch-tests was taken. At D3, positive reactions for the exotic wood species, Teak (*T. grandis* L.), of the above toilet seat and for a wide range of usual allergens molecules including ethylene diamine, paraphenylenediamine, nickel, and cobalt were noted. Following the patch-tests, chronic lichenified dermatitis appeared on the back of the patient but not on regions protected against direct contact and scratching. The diagnosis of lymphomatoid contact



Fig. 1. a. Slightly scaly, erythematous arciform lesion of the two buttocks. b. Lesion after allergen avoidance and 3 months of topical multipotent steroid therapy. c. Exocytosis of lymphocytes into the epidermis with mild cytosis.

dermatitis to an exotic wood species was made. 1 year later, despite strict allergen avoidance and multipotent topical steroids treatment (clobetasol propionate), the patient's condition did not recover completely and the risk to evolve to a frank CTCL is still considered as a cause of concern.

Discussion

Lymphomatoid contact dermatitis was first evoked in 1976 by Gomez Orbaneja and is often a tricky diagnosis (3). The condition has been previously linked to a large variety of allergens including gold, nickel,

formalin, gluteraldehyde, ethylenediamine, and *para-tertyl*-butyl phenol resin. The clinical and histological features in this patient were similar to those reported in other case reports, and positive patch test for the suspected wood species confirmed the diagnosis of lymphomatoid contact dermatitis even if wood could be considered as a quite safe and inert material when in contact with the skin.

The immunological pathway supporting lymphomatoid reaction remains unclear but it has been hypothesized that the disease may result from the stimulation of lymphocytes by antigen recognition to undergo transformation into blast cells (4). Our patient had clinical findings that mimicked CTCL but the hypothesis was denied by histological and immunohistochemical skin examination. Concurrently, T-cell receptor gene analysis did not allow identifying the presence of a clonal population of T cells. Moreover, a chronic lichenified dermatitis appeared on the back of the patient but not on regions protected against direct contact and scratching after patch testing. This report can notify a possible malingering reaction or cross-reacting substances. In addition, our patient did not recover completely after strict allergen avoidance and multipotent topical steroid therapy. Therefore, even if the condition is considered as benign, attention should be given to the long-term follow-up. Such a prudent attitude is reinforced by a recent case report with similar clinical features, developing into T-cell prolymphocytic leukaemia (5) and by the fact that it has also been hypothesized that the disease may evolve into CTCL (1). Indeed, in our patient, the risk to evolve to a frank CTCL should be still considered as a cause of concern.

Hence, patients presenting with lymphomatoid contact dermatitis should be attentively investigated and long-term followed-up as the condition could be the presage of a more ominous disease.

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