ules, vesiculobullous lesions, and pustules,[5] predominantly on the scalp, abdomen, chest and intertriginous areas.[4] The extracutaneous manifestations include lytic bone lesions, diabetes insipidus, growth hormone deficiency, hepatosplenomegaly, and lymphadenopathy.[4] Liver involvement is seen exclusively in multi-system LCH, presenting as isolated hepatomegaly and/or liver function impairment and jaundice.[5] The histopathological analysis and positivity for CD1a, S100, and/or CD207 (Langerin) in immunohistochemistry establishes the diagnosis.

Cutaneous manifestations of LCH are variable and may be similar to other prevalent dermatoses. In the presence of intense and refractory seborrheic dermatitis-like condition, LCH should be suspected, and histopathological and multisystem involvement investigations are mandatory.

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Authors' contributions

Daniela Antoniali: Design and planning of the studied case; intellectual participation in the propaedeutic and/or therapeutic conduct of the studied case; review of the literature; drafting and editing of the manuscript.

Helena Barbosa Lugão: Approval of the final version of the manuscript; drafting and editing of the manuscript; participation in research orientation; intellectual participation in the propaedeutic and/or therapeutic conduct of the studied case; critical review of the literature; critical review of the manuscript.

Daniel Elias: Approval of the final version of the manuscript; drafting and editing of the manuscript; participation in research orientation; intellectual participation in the propaedeutic and/or therapeutic conduct of the studied case; critical review of the literature; critical review of the manuscript.

Cacilda da Silva Souza: Approval of the final version of the manuscript; drafting and editing of the manuscript; collection, analysis, and interpretation of data; intellectual participation in the propaedeutic and/or therapeutic conduct of the studied case; critical review of the literature; critical review of the manuscript.

Conflicts of interest

None declared.

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Treatment of alopecia areata with Diphenylcyclopropenone: methodology based on the principles of allergic contact dermatitis☆

Dear Editor,

Diphenylcyclopropenone (DPCP) is a chemical substance that induces a cellular immune response and, therefore, allergic contact dermatitis (ACD). Its action is based on the concept of antigenic competition, inducing the formation of TCD8 lymphocytes, which inhibit the active perifollicular immune response, allowing hair growth.[1]

DPCP is a therapeutic option for alopecia areata (AA), especially in extensive cases, with a variable response, but repilation rates in more than 50% of cases.[1] Side effects are common, sometimes severe, such as acute eczematous reactions, in addition to lymphadenopathy, pruritus, hyperpigmentation, and flu-like symptoms, among others.[2]

Drug utilization varies, lacking methodological standardization.[2–4] This service uses a methodology based on the principles of ACD. This case report aims to demonstrate the steps of DPCP use in AA. This standardization allowed comparing data and reducing side effects due to drug inappropriate use.

The product is purchased at 2% in acetone and stored in the refrigerator in a dark bottle. The dilutions are prepared

☆ Study conducted at the Clínica de Dermatologia, Irmandade da Santa Casa de Misericórdia de São Paulo, São Paulo, SP, Brazil.

during the appointments and applied weekly with moistened flexible swabs.

A 44-year-old male with universal AA was sensitized with 2% DPCP on 2 × 2 cm filter paper on the back for 48 hours, inducing the ACD induction phase. After 2 weeks, he was submitted to a patch test with DPCP, at 0.1%; 0.05%, and 0.02% concentrations, with readings after 48 and 96 hours, and responses 3+, 2+ and 2+ (Fig. 1) respectively, according to the previously established standardization. The 0.02% concentration was used on the scalp, where it remained covered and unwashed for 48 hours. The concentration was increased weekly to 0.1% when moderate pruritus and erythema were obtained. Repilation was acceptable after 24 weeks, without severe reactions (Fig. 2).

The patch test assesses whether there was sensitization to the product and predicts the best concentration with which to start treatment, choosing the one with the lowest positivity, minimizing adverse effects. If the responses are intense, the concentrations are reduced, and sometimes the applications are spaced out at intervals of two to four weeks. The concentrations depend on the response of each individual.

Therapeutic failure, that is, the absence of repilation, is considered after 180 days of regular application. If there is a response, the applications are maintained until the best possible effect is attained (up to one year).

DPCP is a drug that provides good response rates in severe cases; however, the protocols are not yet standardized. Due to common and sometimes severe side effects, strict monitoring of sensitization and control of the concentrations are necessary throughout the treatment. Moreover, no industry manufactures DPCP in accordance with regulatory standards for drug development.[5] In the present environment, there is no regulation for its use, although it has been part of the therapeutic arsenal for AA treatment for many years.

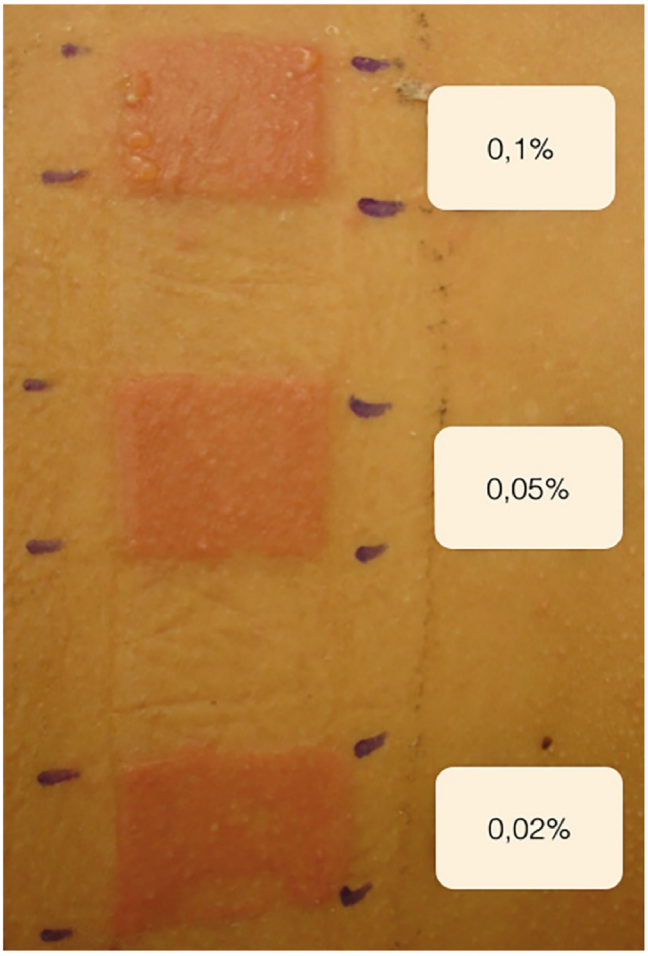

Figure 1 Reading of the patch test after 72 hours.

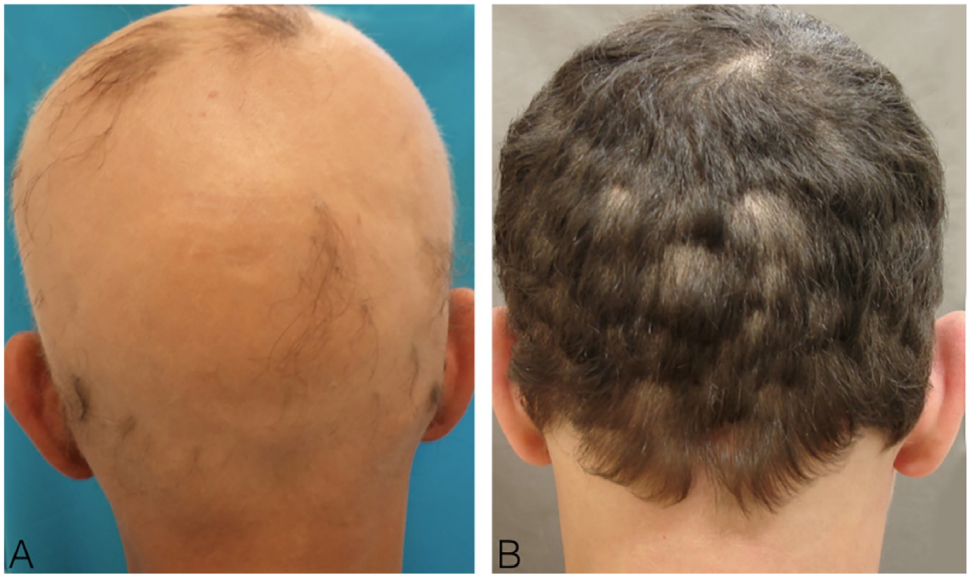

Figure 2 (A), At start of treatment; (B), after 24 weeks of DPCP treatment.

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Authors' contributions

Andressa Sato de Aquino Lopes: Approval of the final version of the manuscript; design and planning of the study; drafting and editing of the manuscript; critical review of the literature; critical review of the manuscript.

Rosana Lazzarini: Approval of the final version of the manuscript; drafting and editing of the manuscript; intellectual participation in the propaedeutic and/or therapeutic conduct of the studied case; effective participation in research orientation; critical review of the manuscript.

Conflicts of interest

None declared.

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Vandetanib induced phototoxic reaction progressed to toxic epidermal necrolysis☆

Dear Editor,

Vandetanib is a tyrosine kinase inhibitor that is approved for the treatment of medullary thyroid carcinoma (MTC). As with other tyrosine kinase inhibitors, vandetanib causes various cutaneous side effects like acneiform eruption, rash, and photosensitivity.[1] Here we describe a patient who developed a phototoxic reaction to vandetanib and then progressed to toxic epidermal necrolysis (TEN) and has been controlled with intravenous immunoglobulin (IVIg). A 43-year-old male was admitted with an erythematous eruption that developed over 3 days. On physical examination, there was a well-demarcated erythematous vesiculobullous rash on the sun-exposed areas of the skin including the face, neck, and hands. The patient had MTC, he was on oral vandetanib therapy (300 mg/d) for 15 days. He denied taking any medication except vandatanib. Few hours prior to the development of the rash, he had been exposed to sunlight for a long time without sun protection. Phototesting and biopsy were not performed. Since the distribution of the eruption was strictly restricted to sun-exposed areas and vandetanib was the only medication, this condition was assumed as a ''vandetanib-induced phototoxic reaction''. Vandetanib treatment was stopped. Oral prednisolone 1 mg/kg/day was administered. In a few days, lesions progressed to proximal extremities, back, and chest with the involvement of 30% of body surface area (Figs. 1 and 2). Oral mucosal erosions and conjunctivitis

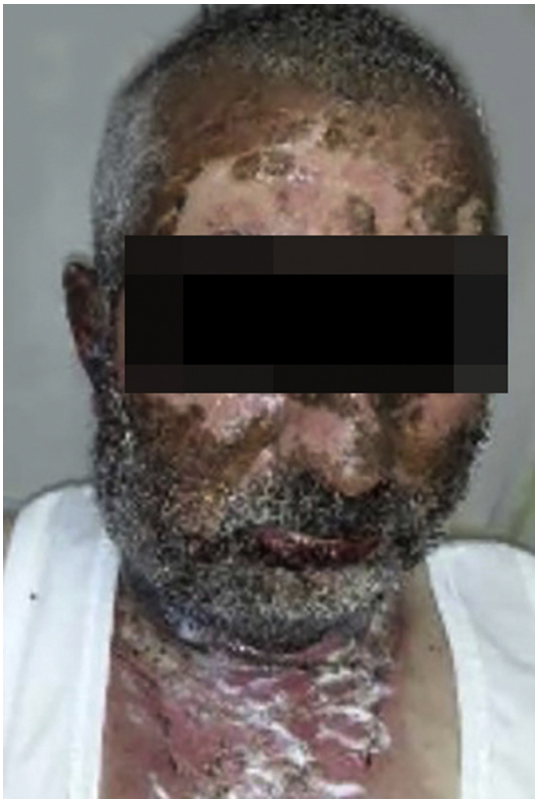

Figure 1 Diffuse epidermal detachment was seen on face.

☆ Study conducted at the Dermatology Clinic of Ondokuz Mayıs University, Samsun city, Turkey.