Atypical Skin Inflammation in a 2.5-Year-Old Girl With Atopic Dermatitis

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Atopic dermatitis (AD) is a recurrent inflammatory skin disorder. Acute AD is characterized by erythematous and exudative lesions, whereas the chronic form is characterized by lichenification and crusting. According to the results of an epidemiological study conducted in Spain, AD was considered idiopathic in 58% of cases and associated with sensitization to allergens in 42% (the responsible allergens were foods in 10% and aeroallergens in 26%) [1]. The role of the skin in the development of allergic reactions, including life-threatening ones, is increasingly significant [2].

Adverse drug reactions are a serious public health problem in that they are associated with high morbidity, socioeconomic costs, and potential fatality [3]. The gravity of this problem can be seen in the increasing incidence of accidental or intentional drug-induced toxicity in children. Drug-induced toxicity (usually through the digestive tract, but also through the skin) is a common problem and one requiring continuing education.

A 2.5-year-old girl with AD was referred to the outpatient allergology clinic with intensified, atypical inflammatory skin lesions and accompanying pruritus.

The history showed that the girl had been diagnosed with AD at the age of 2 months. She was therefore given standard treatment for moderate AD and recommended a dairy-free diet, which led to partial improvement. At 14 months of age, she underwent allergy testing (sIgE with food and inhalant allergens was negative; an atopy patch test with milk was positive) and an open food challenge with cow’s milk. Given the observed exacerbations of AD, she returned to a dairy-free diet. She had typical symptoms of AD on her wrists, popliteal areas, and neck, although during the 4 months before her most recent consultation, she did not experience flare-ups. In the opinion of the parents, the recent exacerbations had no connection with the consumption of food or the action of other external factors. Despite the presence of skin lesions, no other symptoms were observed at admission.
treatment for psoriasis, mistakenly applied her dithranol 2%
parents revealed that the child’s grandmother, who was under
establish the diagnosis. On the third day of hospitalization, the
data in the history indicated exposure to cosmetics, chemical
and with no general symptoms or inflammatory markers. No
ruled out, since the skin changes were too extensive, multifocal,
accompanied by characteristic itchy wheals. Erysipelas was
atypical shape and clear margins. In urticaria, skin changes are
revealed the presence of confluent inflammatory patches
end of the first week of hospitalization. The clinical data suggest irritant contact dermatitis caused
dithranol in a child with AD. With appropriate treatment and
monitoring of the atopic changes, the patient’s skin returned
to normal 4 weeks after the accidental exposure to dithranol.
Medical errors, including incorrectly administered drugs,
are the third leading cause of death in the USA [4]. As many
as 44% of all cases of toxicity in children below 5 years of
age are caused by drugs used in medical care, especially
those prescribed to their caretakers [5]. The most common
route of drug toxicity is through the gastrointestinal tract;
transdermal drug toxicity is uncommon. The predisposing
factors for toxicity in children, even if induced via skin contact,
are differences in metabolism and immaturity of anatomical
barriers in the developing child. Chemical substances applied
to a child’s skin can induce both topical and systemic effects.
Dithranol is an organic chemical compound from the
polyphenol group. It was originally extracted from the araroba
tree and remains one of the most widely used and effective
treatments for psoriasis. According to De Jager et al [6],
dithranol is used in children at low concentrations (0.016%-
0.625%). In adults, it is usually applied at concentrations
ranging from 0.1% to 5%. The present case involved 2%
dithranol ointment. Around 4% of patients stop using the
drug owing to adverse effects. Reported adverse effects after
topical administration of dithranol include burning sensation, irritation, and redness of the skin [7]. The main difficulties with its use are irritation, staining of the skin, and allergic contact dermatitis. Patch testing is important in patients previously described as dithranol-intolerant [8-10]. However, the irritant potential of dithranol hampers patch testing performed to differentiate between allergy and irritation. Moreover, the threshold for detection of contact hypersensitivity may theoretically be above the irritant threshold. It has been suggested that dithranol allergy is more common than generally suspected and that patients who do not tolerate dithranol should undergo patch testing with the allergen [8]. While a positive reaction may only be obtained in a minority of patients, those with a negative result, and therefore a presumably irritant response, could be treated more confidently with lower concentrations rather than having to discontinue a potentially useful treatment. In contrast, the minority who are truly allergic to dithranol should avoid this compound in all its formulations. Löffler et al [9] showed that a pronounced skin reaction to rather low concentrations of dithranol is common, although it is not clear whether this represents an allergic or irritant reaction. The authors concluded that increased reactivity to dithranol most likely reflects genuine increased skin susceptibility, rather than an allergic response.
When applied to a large skin area, dithranol might induce toxicity and lead to symptoms such as those of central nervous system disturbances, kidney damage, and methemoglobinemia. The drug should be used with caution in children with psoriasis. Physicians should make every effort to inform patients about the prevention of drug-induced toxicity.

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Conflicts of Interest
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References
Deflazacort-Induced Erythema Multiforme Exudativum Successfully Treated With a Single Dose of Etanercept

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Corticosteroids can cause immediate and delayed hypersensitivity reactions, with a much higher prevalence in the case of delayed reactions [1]. The corticosteroids most frequently involved in delayed reactions are dexamethasone, betamethasone, and triamcinolone acetonide [2]. Reports of hypersensitivity to deflazacort are rare and include maculopapular rash, acute exanthematous pustulosis, and toxic epidermal necrolysis (TEN) [3-5]. No cases of deflazacort-induced erythema multiforme exudativum (EME) have been reported.

Severe drug-induced delayed reactions, such as TEN or EME, may be extremely difficult to treat. Etanercept, a TNF blocker, has been used successfully in the treatment of TEN [6-10]. While the pathogenesis of TEN is unclear, it was observed that activated T cells secrete large amounts of TNF-α and interferon γ, resulting in apoptosis of keratinocytes [7]. Etanercept blocks this inflammatory pathway via inhibition of TNF.

We present a case of EME caused by deflazacort that was successfully treated with a single dose of etanercept.

A 45-year-old man with no significant personal history came to the emergency department with a 5-day history of erythematous-violaceous rash characterized by infiltrated papules on the scalp, face, neck, trunk, and upper limbs. He also presented with palpebral and labial edema accompanied by erosions on the oral mucosa and a sore throat. Four days previously, he had finished a course of oral amoxicillin-clavulanic acid (875 mg tid) and loratadine after nasal surgery, although he continued taking deflazacort (30 mg per day). After admission, he was unsuccessfully treated with a high dose of systemic methylprednisolone. Eosinophilia was not present, and no other organs or systems were involved. Tests were performed to rule out infections caused by Epstein-Barr virus, cytomegalovirus, human immunodeficiency virus, hepatitis viruses, and Mycoplasma pneumoniae.

Because of the rapidly spreading rash and worsening of the oral erosions, 2 cutaneous biopsies were performed and...