

CONTACT POINTS

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Nickel allergy mimicking basal cell carcinoma

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Key words: basal cell carcinoma, contact dermatitis, mimicking, nickel allergy.

Case Report

A 61-year-old man presented with a solitary ulcerated plaque below the right eye, which had been gradually increasing in size for 8 months. On examination, there was a 1-cm diameter ulcer with a rolled edge inferiorly, and a clinical diagnosis of basal cell carcinoma was made (Fig. 1). An incision biopsy from the rolled edge showed non-specific inflammation, so a second biopsy was taken from a different area to rule out a sampling error. This showed an acute inflammatory infiltrate within the epidermis with acanthosis and a more chronic infiltrate within the dermis. The patient



Fig. 1. Ulcer with rolled edge.

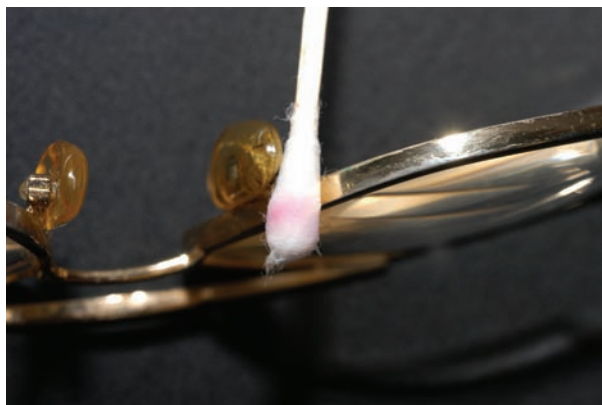


Fig. 2. Positive nickel spot test.

was then referred for patch tests, which demonstrated a strongly positive (+++) reaction to nickel. Examination of the patient's spectacles revealed that the gold plating on the right side had worn off on the rim which rested on the ulcerated area on his cheek. The dimethylglyoxime spot test from the worn area was strongly positive for nickel but was negative from where the gold plating remained on his spectacles (Fig. 2).

Discussion

We have found one previous report of contact dermatitis mimicking a basal cell carcinoma. The case involved a lesion near the medial

canthus of the right eye, and the patient was found to have an allergy to para-phenylenediamine (PPD) from their hair dye (1). The lesion resolved after application of a topical steroid. A response letter later published suggested a basal cell carcinoma was still the most likely diagnosis and had resolved spontaneously by chance (2). They argued that one biopsy was insufficient to exclude carcinoma and that a PPD allergy would be unlikely to cause an isolated lesion in that site. We feel that we have strong evidence that our patient's lesion was in fact a contact dermatitis to nickel, mimicking a basal cell carcinoma. In our case, two biopsies were performed from different sites along the edge of the lesion. In addition, the worn area of our patient's spectacle rim was strongly nickel positive and directly corresponded to the ulcerated area. Many spectacles are now advertised as 'nickel free'; however, leeching of nickel from old worn glasses may occur (3).

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Allergic contact dermatitis from timolol and dorzolamide eye drops

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Contact dermatitis to topically applied glaucoma agents is a recognized but infrequently reported side-effect of treatment. We report a case of contact sensitization from timolol and dorzolamide eye drops.

Case Report

An 80-year-old lady presented with conjunctival inflammation and severe eczematous rash affecting the eyelids and cheeks. Her eye drops for glaucoma were changed from XalatanTM (ingredients: latanoprost and benzalkonium chloride) to CosoptTM (ingredients: dorzolamide hydrochloride 2%, timolol maleate 0.5% and benzalkonium chloride) 3 weeks before presentation. She noticed conjunctival irritation and itchy rash on both eyelids within a week, and symptoms gradually got worse as she continued to use CosoptTM eye drops.

CosoptTM eye drops were stopped, and dermatitis was treated with topical steroids and emollients. Her symptoms settled quickly.

XalatanTM eye drops were restarted for treating glaucoma. However, as her glaucoma was not

adequately controlled, PropineTM eye drops (ingredients: dipivefrine hydrochloride 0.1%, benzalkonium chloride and disodium edetate) were introduced. Unfortunately, within few days, symptoms recurred in mild form and settled after stopping PropineTM eye drops.

Patch testing was performed with British standard series, medicament series, cosmetic series and CosoptTM eye drops. A ROAT was performed with CosoptTM eye drops applied to the volar aspect of the forearm for 2 weeks. All tests were negative.

A supply of pure dorzolamide, timolol and dipivefrine was obtained, courtesy of the manufacturers. Patient was tested with different concentrations in pet. There was ++ reaction to timolol 10% and dorzolamide 40% on D4 (Table 1). Patch testing in 10 control subjects with timolol 10% and dorzolamide 40% did not show any reaction.

Discussion

Contact dermatitis from topically applied timolol, a non-selective β -blocker, is observed in a number of instances and diagnosis confirmed by patch testing in concentrations ranging from 0.25% to 5% (1–3). Contact dermatitis from dorzolamide, a carbonic anhydrase inhibitor, is rare. Two cases of contact dermatitis to a 0.5% solution of dorzolamide, confirmed using patch test are reported (4).

There is no structural similarity between timolol and dorzolamide. The positive reactions indicate 2 separate sensitizations. The apparent worsening of symptoms with PropineTM remains unexplained. Allergy to excipients, benzalkonium chloride and disodium edetate, was excluded on the medicament and cosmetic series.

Table 1. Patch test results

	D2	D4
Dorzolamide (%)		
10	–	–
20	–	–
40	–	++
Timolol (%)		
2.5	–	–
5	–	–
10	–	++
Dipivefrine (%)		
0.5	–	–
1	–	–
2	–	–

The need to test with higher concentrations to avoid false-negative reactions has been stressed (5). Our report re-emphasizes this.

The incidence of contact dermatitis to combination solutions is not significantly different from that of the agents when used individually (6). However, contact dermatitis to 2 different agents, as in our patient, limits treatment options and causes difficulty in management.

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Bikini dermatitis due to mercaptobenzothiazole

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Key words: bikini dermatitis; contact eczema; mercaptobenzothiazole; rubber allergy



Fig. 1. Contact allergic reactions due to Mercaptobenzothiazole in the rubber bands of a bikini.

Case Report

One day after having worn a bikini a 19-year-old patient experienced eczematous lesions in streaks around the hip (Fig. 1) and under the shoulder plate. These lesions appeared on skin areas, which had had contact with rubber bands set in the bikini fabric. Patient's history revealed that 8 years before, wearing of socks had been suspected to cause similar lesions above the ankles. Wearing of sport shoes had been supposed to provoke pruritic plaques on the backs of the feet 10 years before. The pattern of the affected skin, showing lesions where skin had had contact with the rubber bands, whereas skin areas, which only had come into contact with the bikini fabric, remained unaffected, indicated that the putatively causative allergen was contained in the rubber bands and not in the bikini fabric. Patch testing revealed a +++ reaction to mercaptobenzothiazole, a chemical accelerator in rubber products.

Discussion

Even though contact allergy due to rubber as well as to textiles is not rare, contact allergic reactions due to rubber components in textiles are exceptional, in spite of their widespread use. Joseph et al. (1) already described a patient in which mercaptobenzothiazole in a spandex brassieres caused contact allergy. Water and sweat might have solved

mercaptobenzothiazole molecules from the rubber bands in the bikini. Our patient could have been sensitized to mercaptobenzothiazole, likely contained in the shoes. Lynde et al. (2) investigated 119 patients with foot eczema, revealing positive patch test results to mercaptobenzothiazole in 28% of the patients.

As a consequence, sensitization to mercaptobenzothiazole might have led to contact allergic reactions due to mercaptobenzothiazole possibly contained in the rubber bands of the socks 2 years later. In this context, mercaptobenzothiazole was already described as a contact allergen in socks (3).

To the best of our knowledge, this is the first case of contact allergic reactions due to mercaptobenzothiazole in a bikini.

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Mastix, a known herbal allergen, as causative agent in occupation-related dermatitis

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Key words: herbs dermatitis; mastix; occupational eczema; *Pistacia lentiscus*; skin adhesive

Case Report

A 38-year-old professional theatre actor routinely affixed false beards, hair and face masks with 'mastix'.

According to contact points of 'mastix', the patient developed an acute, sharply demarcated eczema with erythema, papules and squamas at the forehead, periorbital, upper lip and chin. Therefore, an as yet unknown contact dermatitis caused by 'mastix' was suspected.

A tapering systemic steroid treatment starting with prednisolone at 30 mg a day was applied. After 4 days, the eczema resolved completely. A 2nd treatment cycle consisting of prednicarbate cream was administered for 3 days in the outpatient clinic due to relapse of the eczema after re-exposure to 'mastix' by the actor himself.

We performed extensive patch tests including the German standard series, gum series, synthetic resins and acrylates series (German Contact Dermatitis Research Group, DKG), the two 'mastix' skin adhesives and the patient's make-up equipment. The tests revealed a strong sensitization and crescendo time course against turpentine, colophony and both 'mastix' skin adhesives. No sensitization was seen against the synthetic resins, the adhesives containing acrylates, the gum series and the make-up colours.

Discussion

To our knowledge, this is the first report of a mastix-related allergic contact dermatitis as an occupational

disease, although mastix is widely used as a skin adhesive by actors and make-up artists.

Mastix, also known as mastic, is the resin of the mastic tree *Pistacia lentiscus* that grows in the countries around the Mediterranean Sea and on the Canary Islands. In traditional medicine, the resin is used for a variety of predominantly gastrointestinal disorders such as stomach pain and ulcers and is also used to alleviate pain and swelling caused by traumatic injuries. An antibacterial effect of mastix gum is known against *Helicobacter pylori* (1). In a double-blinded randomized study, it was shown that mastix chewing gum reduces the plaque formation on teeth and bacterial growth in saliva (2). The monoterpenes verbenone, linalool and α -terpineol have been identified as the compounds contributing mostly to the antibacterial activity of mastic oil (3).

The resin contains as main components a broad variety of different triterpenes. An essential oil consisting mainly of monoterpenes can be found in the resin at a concentration of about 1–3%. Several allergic reactions as adverse effects of topical mastix therapies are reported (4, 5).

On the basis of the patient's occupational use of 'mastix' over the last 12 years, sensitization occurred and finally led to the eczema. Patch testing of the 'mastix' skin adhesives displayed a crescendo-type pattern that is more likely in allergic than in irritant reactions. It is important to note that there are skin adhesives available for actors that are also called 'mastix' but contain acrylates instead of the resin of the mastic tree. Therefore, it is necessary to test acrylates as well as the mastic tree-based adhesives.

Besides the allergy to mastix, patch testing also revealed newly emerged sensitizations against colophony and turpentine suggesting a cross-allergy between mastix, turpentine and colophony that has not been reported previously.

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Allergic contact dermatitis from iodine in a topical ointment

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Key words: allergic contact dermatitis; iodine; medicaments; topical ointment

Case Report

A 41-year-old male sales clerk was treated for a yellowish necrotic ulcer on the left ankle, with Cadex[®] ointment 0.9%, containing iodine (0.9%) and cadexomer (Torii Pharmaceutical Co. Ltd, Tokyo, Japan). The patient had no personal and familial history of dermatitis herpetiformis. Although the topical medicament was effective for the necrotic ulcer, an itchy

erythema suddenly developed around the ulcer 6 months later.

Patch testing showed a positive reaction to the ointment itself on D2 (+), D3 (+) and D4 (+). To identify the precise allergen, further patch testing was done with ingredients provided by the manufacturer. 0.33% iodine/10% glycerine w/v was positive on D2 (+++), D3 (+++) and D6 (+). All other ingredients including 10% glycerine aq., pet. and purified water were negative.

Control patch tests were performed on 6 healthy volunteers using 0.33% iodine/10% glycerine w/v, 10% glycerine aq., pet. and purified water. All volunteers showed negative reactions.

Discussion

Iodine-containing topical medications are available in both solution and ointment forms. They are commonly used not only as antibacterial agents but also for treating necrotic decubitus, leg ulcers and severe third-degree burns. Cases of allergic and irritant contact dermatitis due to iodine have been reported (1, 2). Most cases were caused by disinfectant solutions (1). Only 2 cases of allergic contact dermatitis due to an ointment containing iodine have been reported (2).

The present case of allergic contact dermatitis apparently occurred during treatment for a leg ulcer. Absorption of iodine through the leg ulcer, which had been used under occlusive conditions for a long time, is likely to have promoted the contact sensitization. Because allergic contact dermatitis from iodine in solution is not uncommon and iodine-containing ointments are usually applied under occlusive conditions, allergic reactions to iodine-containing ointments may be more common than has been reported. This case suggests that dermatologists should be attentive to allergic reactivity to iodine in topical ointments.

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Photoallergic contact dermatitis from topical diclofenac in Solaraze[®] gel

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Key words: diclofenac; photoallergic contact dermatitis; photoallergy; photosensitivity; Solaraze[®] gel

Solaraze[®] gel (Shire Deutschland GmbH & Co. KG, Cologne, Germany) containing 3% diclofenac has been licensed in 2001 as a topical treatment for actinic keratoses. It is commonly used in dermatological practice. Undesirable effects are believed to be rare but include pruritus, paresthesia and application-site reactions (dry skin, rash, erythema, contact dermatitis and vesicobullous eruptions). Recently, a few cases of contact dermatitis due to three different allergens including diclofenac have been reported (1,2).

Case Report

A 77-year-old female patient with a single superficial actinic keratosis at her right cheek developed an infiltrated and exsudative, stinging and itching erythema at this site in early

summer following 7 weeks of treatment with Solaraze[®] gel. Consequently, Solaraze[®] gel was discontinued, and a symptomatic treatment with a topical corticosteroid for the dermatitis was initiated which led to the clearance of the dermatitis but leaving the pre-existing actinic keratosis.

Patch tests were performed to Solaraze[®] gel and its ingredients kindly provided by the manufacturer at the following concentrations: Solaraze[®] gel as it is, Solaraze[®] gel 5 and 10% pet., diclofenac 1% aq., benzyl alcohol 5% pet., polyethylene glycol monomethyl ether 350 (PEGMME 350) 1 and 5% aq. and sodium hyaluronate 0.5 and 1% aq. All patch tests were negative at D2 and D4.

An additional photopatch test was performed in parallel with the same substances at the same concentrations, irradiating the test area with 10 J/cm UVA at D2. Positive reactions were seen to Solaraze[®] gel 5% pet. at D3 (++) and D5 (++) and Solaraze[®] gel 10% pet. at D3 (++) and D5 (++) and Solaraze[®] gel as it is at D3 (++) and D5 (+++), diclofenac 1% aq. at D3 (++) and D5 (++) and D5 (++++). All other photopatchtests with benzyl alcohol, polyethylene glycol monomethyl ether 350 and sodium hyaluronate were negative.

From these results, the nature of the disease as a photallergic contact dermatitis due to diclofenac as an ingredient of Solaraze[®] gel could be disclosed by positive photopatch testing, whereas an ordinary allergic contact sensitization could be excluded by negative patch testing.

Discussion

Solaraze[®] gel is approved for the topical treatment of actinic keratoses. Active ingredient is diclofenac 3% dissolved in a gel containing 2.5% hyaluronate. Best clinical cure rates (58–85%) were observed when the product is administered at least for 90 consecutive days.

Diclofenac is a NSAID and a potent inhibitor of cyclo-oxygenase 2 and prostaglandine synthesis. Supposed mechanisms of actions in cancer are an inhibitory effect on angiogenesis and a proapoptotic effect on tumour cells.

Two cases of delayed-type hypersensitivity rash following oral intake of diclofenac with subsequent positive patch testing were reported (3). Furthermore, topical use of diclofenac as antirheumatic drug resulted in allergic contact eczema (4). Recently, Kerr et al. (1) reported three cases of allergic contact dermatitis due to Solaraze[®] gel, with diclofenac, polyethylene glycol monomethyl ether and benzyl alcohol as the relevant contact allergens, respectively. An other report on allergic contact dermatitis from Solaraze[®] gel due to diclofenac sensitization was given by Kleyn et al. (2).

As other NSAID's, diclofenac is a substance with photosensitizing potential. In a series of 75 patients with photodermatose, 5 cases showed a positive photopatchtest to diclofenac (5).

Recently, one case of a photoallergic contact dermatitis due to sodium diclofenac 1% in an antirheumatic gel was reported in this journal (6). We present a case demonstrating a photoallergic contact dermatitis to diclofenac as the active ingredient in Solaraze[®] gel for actinic keratoses. Dermatologists should be aware of further similar observations, because the necessary long treatment periods with Solaraze[®] gel in actinic keratoses increase the risk that intensive concomitant sun exposures of the usually treated skin areas might occur during summer or vacation.

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2-phenoxyethanol-induced contact urticaria

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Key words: 2-phenoxyethanol; cosmetics;
type 1 allergy; urticaria

2-phenoxyethanol has previously been described to be responsible for contact urticaria after the application of cosmetics on 2 occasions (1, 2). We describe the youngest case yet of contact urticaria caused by this chemical.

Case Report

An 11-year-old girl, with no history of dermatological problems, attended the patch test clinic as she had developed a reaction to her mother's moisturiser. The subject had been using her mother's 'age defying' product on her face and reported swelling and pruritus, which developed after 30 seconds following application. The hands also swelled, and within a few minutes, she was unable to see out of her eyes due to swelling. There was no respiratory compromise. The symptoms settled within 20 min. This had not happened with any other products she had used.

Patch tests were not performed as the history was not suggestive of this. Scratch tests with the product were positive at 10 min. The manufacturer of the product was then contacted and provided some of the base ingredients to the cream to which were applied to the skin and the scratch test repeated, using the original product as a control. At 10 min, positive wheals were seen for both the original product and 2-phenoxyethanol.

Discussion

Contact dermatitis to cosmetic products is a well-recognized phenomenon, and great strides have been made in ensuring the ingredients that are labelled on the packaging. Contact urticaria is less common, in particular to preservatives. 2-phenoxyethanol is a preservative used extensively in cosmetics and medications. It is effective against both gram-positive and gram-negative bacteria and fungi. As stated earlier, there have only been 2 other reported cases of contact urticaria to 2-phenoxyethanol, and indeed there are only 3 reported cases of contact dermatitis to the product (3–5). 2-phenoxyethanol can therefore be regarded as a relatively safe preservative in both cosmetics and pharmaceutical products, but doctors must remain aware of the possibility of both contact dermatitis and urticaria as a result of exposure to products containing it.

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Decyl glucoside contact allergy from a sunscreen product

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Key words: alkyl glucosides; contact dermatitis; cosmetics; decyl glucoside; emulsifiers; sunscreen product; surfactant

Allergic contact dermatitis from emulsifiers in topical products is occasionally reported and may appear as sudden aggravation of chronic/recurrent dermatitis in patients with long-lasting skin diseases and in the need of continuous use of topical products (1, 2). Decyl glucoside is an emulsifier, a non-ionic surfactant and cleansing agent manufactured as a condensation product from decyl alcohol and a D-glucose polymer. It is frequently used in rinse-off products because of good skin tolerance and also in leave-on products such as cleansing milks, lotions and sunscreen products. It has several CAS registry numbers [58846-77-8] [68515-73-1] [141464-42-8] [54549-25-6] (3). We describe one more case of decyl glucoside allergy from the sunscreen agent Tinosorb[®] M.

Case Report

A 67-year-old man with a long history of recurrent palmoplantar pustulosis over 25 years was treated intermittently with etretinate and topical pharmaceutical products. About 15 years ago, he started to develop hand eczema and developed

recurrent patchy itchy dermatitis on his trunk and extremities. A skin biopsy showed changes compatible with an eczema. Diagnostic patch testing in 1989 showed a clinically relevant and reproducible bromonitropropanediol contact allergy (0.5% pet.) caused by a hand cleanser used by the patient; his hand eczema cleared subsequently after withdrawal of this product.

The patient was seen again from 1995 because of recurrent dermatitis; patch tests were repeated in 1997 with the standard series, supplemented with a corticosteroid series and the skin care products used. Bromonitropropanediol was negative this time.

In the following years, the patient continued to suffer intermittently from hand and foot dermatitis and was treated by a practising dermatologist. He received systemic corticosteroids in short courses and systemic antibiotics because of secondary infections.

In 2001, he was patch tested again and showed a relevant contact allergy to quinoline mix, caused by topical treatment with a cream containing both betamethasone valerate and clioquinol.

In the spring of 2003, the patient was again referred to the department of dermatology, Odense University Hospital, due to a sudden exacerbation of widespread dermatitis, vesicular locally, related to sun exposure and the use of a sunscreen product. Subsequent phototesting in November 2003 showed a pathological UVA sensitivity below 3.5 J/cm² and borderline UVB sensitivity with MED 0.030 J/cm². Further diagnostic patch testing with the standard series and his own products showed + reactions on D3 and D8 to the following TRUE test allergens: chlormethylisothiazolinone/methylisothiazolinone, Quaternium 15 and formaldehyde, all of unknown relevance because a careful exposure analysis could not document any exposure to these chemicals in topical products used by the patient. The previously reported positive reactions to quinoline mix and bromonitropropanediol could not be reproduced.

He also had a ++ reaction to the sunscreen product, Lait Avene 60(60B/60A) (Pierre Fabre, Ramonville, France) preserved with phenoxyethanol and parabens. A repeated open-application test with

the sunscreen product became strongly positive after 2 days documenting a relevant allergic contact dermatitis from the use of this product.

The manufacturer of the sunscreen, Pierre Fabre, kindly provided all product ingredients for patch testing, and on subsequent testing, the patient reacted with a ++ reaction to a new sample of the finished product, as well as to a 6% aqueous solution of the sun-filter Tinosorb[®] M, which is a 50% aqueous dispersion of colourless organic microfine particles with a size below 200 nm. The active chemical is methylene bis benzotriazolyl tetramethyl butylphenol, whereas decyl glucoside 7.5% is added as a surfactant. The concentration of Tinosorb[®] M in the product is 6%. Tinosorb[®] M is permitted in a concentration up to 20% in cosmetic products.

Repeated patch testing with the sunscreen product and decyl glucoside in a dilution series from 5% aq. down to 0.5% aq. produced + reactions to all tests at D3.

Simultaneous tests with another sample of decyl glucoside (Oramix NS 10[®]) kindly obtained from Seppic, Paris, France and a related glucoside, i.e. coco glucoside (Plantacare 818[®]) kindly provided by Cogniz, Dusseldorf, Germany), were also positive at 2% aq.

25 consecutive contact dermatitis patients acted as controls and were patch tested with Tinosorb[®] M 6% aq. with negative results.

Comment

Even though widely used in cosmetics and topical products, contact allergic reactions to emulsifiers and surfactants are relatively uncommon. In recent years, several case reports of contact allergic reactions from decyl glucoside have been published, e.g. (4–7). The optimal patch test concentration of decyl glucoside is, however, not established. It may be higher than used in this case report – perhaps up to 5–10% aq. or pet. (4–7). As has been published previously (5, 8), the positive reactions to related glucosides may be an argument for the existence of group allergy towards alkyl glucosides, which are often mixtures of various

alkyl chain lengths. It is not yet known, however, whether the allergens concern the glucosides themselves or possible contaminants. Further work is in progress.

This case report illustrates the importance of collaboration with the product manufacturer and of the willingness of a patient to undergo repeated diagnostic tests and thorough investigation in order to identify the final allergen. The primary tests may not be conclusive indeed.

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Allergic contact dermatitis mimicking atopic dermatitis due to enoxolone in a topical medicament

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Key words: 18-glycyrrhetic acid; allergic contact dermatitis; atopic dermatitis; enoxolone; medicament

Moisturizing lotions, creams and ointments are applied for dry skin. We describe a case of allergic contact dermatitis mimicking atopic dermatitis when using a moisturizer of a topical medicament containing enoxolone (18-glycyrrhetic acid).

Case Report

A 7-year-old girl, who had been diagnosed as having atopic dermatitis, visited us with pruritic erythematous macules on the bilateral cubital fossa (Fig. 1), where she had applied an over-the-counter ointment. The lesions were only observed on the applied area. A 2-day (D) closed patch test using the ointment as is produced a positive reaction at D2 (+) and D3 (+). 42 days later, a second patch test was done using each of the ointment's ingredients, provided by the manufacturer. This second test produced a positive reaction to enoxolone 1% pet. at D2 (+),



Fig. 1 Clinical picture of allergic contact dermatitis mimicking atopic dermatitis due to enoxolone in a topical medicament for dry skin.

D3 (+) and D4 (+). Other ingredients and pet. as is were all negative.

Discussion

Enoxolone is derived from glycyrrhizic acid (glycyrrhizin), which is a component of Licorice root, a plant of the *Glycyrrhizaceae* family (1). Enoxolone is widely used in cosmetics and topical medicaments as an anti-inflammatory ingredient (2). Only 3 cases of allergic sensitization have been reported (2–4).

The present case of allergic contact dermatitis showing symmetrical distribution occurred during self treatment. Absorption of enoxolone may have been facilitated through dry skin, inducing contact sensitization. We postulate that allergic sensitization to it is more common than is generally realized and that some of the cases may be misdiagnosed as having atopic dermatitis.

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