

CONTACT POINTS

CONTACT DERMATITIS 2005; 52: 226–234

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Allergic contact dermatitis to drometrizole trisiloxane in a sunscreen with concomitant sensitivities to other sun screens

Contact Dermatitis 2005; 52: 226–227

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Key words: allergic contact dermatitis; drometrizole trisiloxane; photoallergic contact dermatitis; sunscreen filters.

Case Report

A 59-year-old non-atopic woman presented with an eczematous rash that had erupted 5 days after she had returned home from a holiday in the Canary Islands. The rash, which started on her legs and back, had spread extensively and resulted in her needing 4 days of inpatient care.

Patch testing, using an extended British Contact Dermatitis Society standard series and also a cosmetic and a textile series, was negative. Photopatch testing (5 J/cm² UVA), using a combination of commercially available photoallergens supplied by Trolab (Hermac, Reinbek, Germany) and Chemotechnique (Crawford Pharmaceuticals, Milton Keynes, UK), showed a positive result for benzophenone 4 (D2⁺/D4⁺) on the irradiated skin only. 2 of the patient's sunscreens were also photopatch tested undiluted: there were positive (+) reactions at D4 to both products on the non-irradiated and irradiated skin, the D2 reactions being ?+.

The ingredients of the patient's 2 sunscreens, referred to as A and B, were further investigated. Neither sunscreen listed benzophenone 4 as an ingredient but both contained several UV filters. Photopatch testing with the ingredients of A showed positive (+) reactions to drometrizole trisiloxane (2.5% alcohol) at D4 on both non-irradiated and irradiated skin, consistent with a non-photoallergic positive reaction. There were ?+ reactions at D2 to this UV filter with negative reactions at D2 and D4 to all of the other 24 ingredients. There were no positive results when the ingredients of sunscreen B were similarly tested. Patch testing with the drometrizole trisiloxane in 15 control patients gave negative reactions only at D2 and D4.

Discussion

Invented in 1999, drometrizole trisiloxane, otherwise known as mexoryl XL (CAS no. 155633-54-8; trade name Silatrizole), is a photostable, oil soluble, chemical UVB and UVA light filter. It is authorized for use in cosmetics up to a maximum concentration of 15%. It cannot be used alone in sunscreens because of its limited spectral activity. It is therefore combined with other chemical and physical filters to obtain high sun protection factor (SPF) values. Increasing the concentration of individual filters usually achieves little extra benefit with SPF but raises the potential for adverse irritant and allergic reactions. Combinations of filters at relatively low concentrations are therefore formulated. Due to the risk of photoinstability, with resulting reduction in SPF and the production of potentially allergic and irritant byproducts, synergy of action of the UV filters is sought. At the request of the manufacturers, sunscreen B was returned to them

for analysis. No impurities were found.

One possible explanation for the negative results to the ingredients of sunscreen B is that when combined in the product a compound allergic reaction was produced. Another possibility is that an allergen was patch tested at too dilute a concentration to elicit a positive reaction. The ingredients of sunscreen B were provided by the product manufacturer at concentrations stated to be suitable for patch testing. The concentrations of commonly tested allergens have been derived from past experience and knowledge, but with potentially new allergens, the investigator cannot be sure what is the optimal concentration for patch testing. In such situations, it is preferable to obtain the ingredients undiluted to enable serial dilutions to be made as necessary. Such an approach, however, is not always possible. In addition, some UV filters are difficult to patch test with and can produce ?+ reactions due to irritancy. This was not found in the 15 control patients who were tested with the drometrizole trisiloxane.

Multiple contact allergic or photoallergic reactions to UV filters are known to occur in patients with photoallergic reactions forming the minority (1). The use of such filters is not confined to sunscreens: they are also frequently included in cosmetics and toiletries to guard against skin photodamage and to retard photodegradation and hence prolong the product's lifespan. Contact dermatitis due to UV light filters may therefore present clinically as a rash with a distribution not typical of a sunscreen application. Photoallergic rashes usually manifest 24–72 hr after UV exposure. Our patient's rash, which erupted 5 days after returning from holidays, would therefore be more typical of a non-light-induced reaction.

The commonly used commercial photoallergens series, available in

the UK, are not sufficient for investigating contact dermatitis to the newer sunscreen filters: it is important to patch test using the patient's own suspected sunscreens. Patch testing, using the product ingredients prediluted by the manufacturer, may give false-negative results if not provided at optimal concentrations for testing.

Allergic reactions to drometrizole trisiloxane are not unknown but are very rare (personal communication from manufacturer). We have not been able to identify any previously published report of allergic contact dermatitis due to this sunscreen filter.

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Contact allergy to decyl glucoside

Contact Dermatitis 2005; 52: 227

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Key words: allergic contact dermatitis; decyl glucoside

A 25-year-old-veterinary nurse developed eczematous patches on her arms and confluent eczema of the neck, upper chest and face. Subsequently, there were also acute eczema of the scalp and associated hair loss. Inflammation of the upper chest was sharply demarcated at the lower margin and confined to skin which came into contact with the patient's shoulder length hair. Contact allergy

to the patient's customary hair mousse (Wella's Shock Waves Curl Shaping mousse, strong hold; Darmstadt, Germany) was suspected and confirmed by patch testing to the mousse 'as is'. Patch tests to the British Contact Dermatitis Group standard series and to a cosmetic and the hair-dressing series were negative.

Further patch tests were undertaken to individual ingredients of the mousse, kindly supplied by Wella. At 48 hr and 96 hr, there were positive reactions (+ and ++) to a 2% aqueous solution of decyl glucoside. There were no reactions to any other ingredient. Symptoms improved after avoidance of the mousse and treatment with topical steroids.

Decyl glucoside is a non-ionic surfactant used in rinse-off products such as shampoos, hair dyes and soaps. It is also found in some leave-on products, including sunscreens. The one previous report of contact allergy to decyl glucoside relates to its presence in a topical antiseptic, used after body piercing (1). Contact allergy to Shock-Waves hair mousse has been reported, but the three patients described all had positive patch test reactions to 3-iodo-2-propynyl-butylcarbamate.

Individual ingredients of the mousse were not tested (2). Our patient did not react to this biocide at either of the two patch test sites where it was applied.

This patient's experience serves to reinforce the importance of patch testing to patients' own products and to individual ingredients.

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Erythrodermia caused by allergy to codeine

Contact Dermatitis 2005; 52: 227–228

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Key words: codeine; cross-sensitivity; cutaneous adverse drug reactions; dihydrocodeine; drug allergy; medicaments; opioids; positive patch test.

An 85-year-old man came to see us, because he was suffering since 3 days ago an erythematous eruption that began in his legs and progressively was extended to his trunk, arms and face, accompanied with intense itching and developed with vesicular exudative lesions in some areas. The eruption persisted for 10 days and disappeared via scaling after having received treatment with oral corticosteroids (deflazacort). Some days before, the patient had been taking nolutil[®] (an analgesic belonging to pirazolone group) and efferalgan[®] (acetaminophen) to treat a pain in the back. The patient thought he could have also taken (by mistake) some pills of cod-efferalgan[®] (acetaminophen 500 mg and codeine 10 mg).

He was patch tested with European standard series (True-test[®]) that were negative. Patch tests with drugs (nolutil[®], acetaminophen 5% pet. and codeine 0.1% aq.) were positive (++) with codeine. Subsequently, he was patch tested with other opioid drugs, with positive reaction to morphine hydrochloride (+) and dihydrocodeine (+) (Table 1).

We describe an unusual case of erythrodermia due to type IV hypersensitivity to codeine included in a drug in combination with acetaminophen. A similar case with the same drug (Cod-efferalgan[®]) was also described by Estrada et al. (1). Similarly, Rodriguez et al. (2) reported a young man with several episodes of general dermatitis due to codeine contained in antitussive and analgesic drugs. In both cases, like in our patient, patch tests were positive with codeine and also with morphine chloride and were negative with other opioid drugs. These authors did not include in the patch tests the dihydrocodeine that had been

Table 1. Patch test results with opioids

	% w/v	D2	D4
Codeine phosphate	0.1% aq.	+	++
Morphine hydrochloride	1% aq.	+/-	+
Tramadol	5% pet.	-	-
Dihydrocodeine	20% pet.	-	+
Pentazocine	5% pet.	-	-
Buprenorphine	0.3% pet.	-	-

aq., aqueous solution; pet., petrolatum.

also positive in our patient. However, Cooper (3) published the case of a woman with positive patch tests to dihydrocodeine but not to codeine. All these data support that codeine can elicit late drug allergic reactions. Therefore, we must bear in mind that this drug is frequently taken by the patients without a prescription; hence, we should be aware of it by asking the patient about the drugs he/she has taken.

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**Cutaneous allergic
reaction to heparins:
subcutaneous but not
intravenous
provocation**

Contact Dermatitis 2005; 52: 228-230

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Case Report

On 5 January 2004, a 31-year-old woman presented with pruritic erythematous lesions of about 4 cm in diameter on her thighs and upper arms. The lesions appeared 3 weeks after the initiation of subcutaneous calcium heparin (Caprocin, 20 000 IU/0.8 ml), following a deep vein thrombosis of the left lower leg in the 11th week of her second pregnancy. She was hospitalized and intravenous sodium heparin (Novo Heparin, Mochida Pharmaceutical, Tokyo, Japan, 1000 IU/ml), 18 000 IU daily, was administered on 31 October 2003. She was subsequently switched to subcutaneous calcium heparin, 7500 IU $\times$ 2 a day, before she was discharged.

3 weeks after the initiation of subcutaneous heparin injection, she developed erythema accompanied by intense pruritus at the injection sites. The pruritic erythema appeared about 24 hr after the injection and disappeared within a week. The changes in colour, induration and pruritus were most significant 48-72 hr after the injection (Fig. 1). Histopathologic examination of the

Key words: calcium heparin; cutaneous adverse drug reactions; delayed-type hypersensitivity; heparin; late-phase reaction

Cutaneous allergic reactions due to subcutaneously injected heparin have been reported and are mostly unrelated to heparin necrosis. Affected patients usually develop infiltrated erythematous plaques with or without vesicles at the injection sites. In the majority of the cases reported, delayed type hypersensitivity to heparin has been suggested as an underlying pathomechanism for skin lesions (1-6). We report a case of cutaneous allergic reaction caused by subcutaneously injected calcium heparin (Caprocin, Nihon Schering, Osaka, Japan).

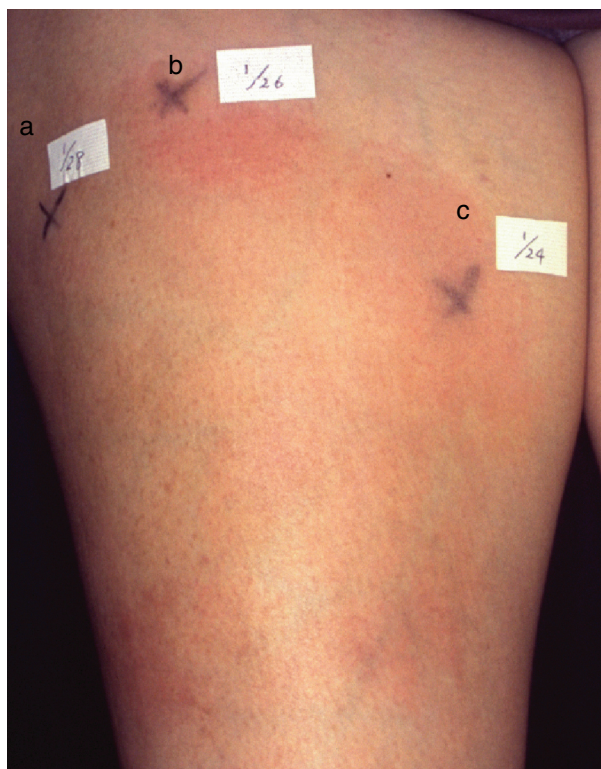


Fig. 1. Clinical appearance of injected sites at 24 hr (a), 72 hr (b) and 5 days (c) after injection. Erythema was most evident 72 hr after injection.

lesion, 3 days after the injection, showed marked perivascular oedema associated with lymphocytic infiltration, admixed with mast cells and eosinophils (Fig. 2). There was no evident change in the epidermis.

The patient was suspected to have a cutaneous intolerance reaction to calcium heparin, and the subcutaneous heparin was discontinued. Instead, intravenous sodium heparin administration was started and was well tolerated by the patient.

Skin tests were performed with calcium heparin and sodium heparin. Patch tests were all negative (Table 1). Prick tests gave a doubtful reaction to calcium heparin. An intradermal test with calcium heparin yielded a positive reaction 20 min after the test, which faded within 24 hr. Erythema with localized pruritus reappeared 48 hr later at the test site. Sodium heparin failed to elicit any response. 6 controls showed no reactions to the same tests. A subcutaneous provocation test with 0.1 ml and 0.2 ml of calcium heparin gave infiltrated, pruritic erythema at

the injection sites 48 hr and 24 hr later, respectively. Sodium heparin also showed erythema at the test site after 48 hr. These reactions persisted for 7 days.

Discussion

The patient developed pruritic erythematous plaques at the sites of subcutaneous heparin injection. Allergic reaction to subcutaneous heparin is not rare in the English literature (1–6). However, as far as we know, this is the first Japanese case report of allergic reaction to subcutaneously injected heparin. 2 points are noteworthy in this case. First, a patch test for heparins was negative. Intradermal testing with calcium heparin gave an immediate-type positive reaction characterized by the presence of erythema and wheal 20 min after the test, which was followed by the appearance of pruritic infiltrated erythema 48 hr later. Sodium heparin was unable to elicit a positive reaction either

20 min or 48 hr later, probably because of the low-heparin concentration (1000 IU/ml).

To our knowledge, there are only a few reports on allergic reaction to subcutaneous heparin in which a patch test was negative and intradermal tests were positive immediately and 48 hr after tests (7, 8). Hallai et al. reported a patient who developed immediate itching and later on erythematous plaque at the injection site of subcutaneous low-molecular-weight heparin (LMWH) (8). The patient showed positive reactions to intradermal tests with LMWH as well as unfractionated heparin, 15 min and 48 hr after testing (8). These authors suggested that both immediate and delayed-type hypersensitivity reactions were involved. In the present case, however, the clinical and histopathological findings as well as the reactions to intradermal tests suggested that the allergic reaction to subcutaneous heparin resulted from an immunoglobulin E-mediated late-phase reaction, even though the reaction occurred 24 hr after the injection and persisted for 7 days.

Recently, LMWHs have been increasingly used in many countries, and cross-reactivity between various LMWHs and unfractionated heparins has been reported (9, 10). For that reason, skin testing of a panel of heparins and heparinoids is recommended to find safe alternatives for patients allergic to specific heparins (9, 10). In the present case, however, skin testing with LMWHs was not performed because subcutaneous LMWHs are not yet fully available for the treatment of deep vein thrombosis in Japan.

Second, intravenously administered sodium heparin was well tolerated by the patient, without any adverse reaction, even after provocation of the allergic reaction to subcutaneous heparin. In cases of allergic cutaneous reaction to subcutaneous heparin, it has been emphasized that changing heparin administration to the intravenous route should be avoided as it may be unsafe and promote a worse outcome (11). Conversely, Boehncke et al. reported a case of delayed-type hypersensitivity to heparins in which intravenous heparin was tolerated by the patient (3). A possible explanation for this divergence is that delayed-type hypersensitivity to heparins requires

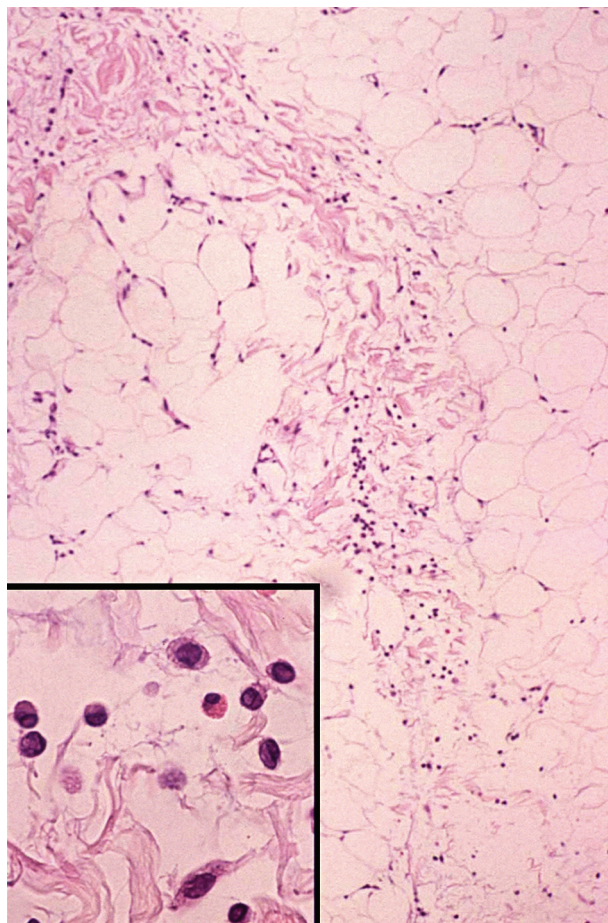


Fig. 2. Histological findings of lesion, showing marked perivascular oedema associated with infiltration of lymphocytes, mast cells and eosinophils.

Table 1. Heparin skin test results

Tests	Calcium heparin (20000 IU/0.8 ml)		Sodium heparin (1000 IU/ml)
Patch as is			
48 hr	—		—
72 hr	—		—
Prick as is 20 min	±*		ND
Intradermal	625 IU		50 IU
20 min	+ +		—
24 hr	—		—
48 hr	+		—
3 days	+		—
7 days	+		—
Subcutaneous	2500 IU	5000 IU	500 IU
24 hr	—	+	—
48 hr	+	+	+
3 days	+	+	+
7 days	±	+	+

*doubtful reaction; + = erythema; ± = faint erythema for intradermal and subcutaneous tests at 24 hr to 7 days; ND = not done.

binding of allergens to dermal or subcutaneous proteins (4). In addition, intravenously administered heparins may not be sufficient in amount for the provocation or induction of erythematous infiltrated plaques.

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Peri- and subungual burns caused by hydrofluoric acid

Contact Dermatitis 2005; 52: 230–232

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Key words: calcium gluconate; onycholysis; subungual blisters.

Hydrofluoric acid (HF) is the inorganic acid of elemental fluorine and is one of the strongest and most corrosive acids, widely used not only in industrial settings (glass etching, electronics and the petrochemical, plastic and dye industries) but also in domestic products, such as cleaning solutions and rust removers (1).

Hydrofluoric acid handling is highly dangerous both because it can produce severe cutaneous burns and because it has potential systemic and lethal toxicity (2).

Prompt first aid is mandatory to reduce damage after HF cutaneous exposure (3).

We report here the occurrence of peri- and subungual blisters after contact with HF in anti-rusting solution.

Case Report

A 49-year-old man was admitted to our outpatient consultation of the Department of Dermatology of the University of Bologna in May 2004, complaining of the recent onset of painful haemorrhagic vesiculobullous lesions involving the peri- and subungual regions of the fingers. He reported that the afternoon before he had cleaned the engine of his boat with a cloth soaked in anti-rusting agent without use of gloves, and he could not avoid contact between his hands and the anti-rust substance.

12 h after this, a burning pain had started in the fingertip of the second right finger and in the fingertip of the first, second, third and fourth left fingers.

The clinical examination revealed mild burn of the pulp appearing as severe dryness and haemorrhagic bullae in the hyponychium and nail bed of several digits (Fig. 1).

The patient brought the anti-rusting agent utilized, that contained 15% HF.

A few hours after the onset of nail lesions, the patient went to his doctor who consulted the Antipoison center of Milan and was told to apply a gel containing 2.5% calcium gluconate on the digits (Table 1). The procedure did not, however, improve signs or symptoms. We sent the patient to a plastic surgeon who



Fig. 1. Mild burn of the pulp appearing as severe dryness and haemorrhagic bullae in the hyponychium and nail bed.

excised the necrotic tissue, debrided the blisters and applied 2.5% calcium gluconate gel directly on the base of the lesions to be massaged into the burned skin every 2–3 hr. Evaluation of the patient after 1 week showed marked improvement of the nail lesions (*Fig. 2*). The patient referred complete disappearing of the pain soon after the first topical treatment.

Discussion

Hydrofluoric acid burns are uncommon but typically characterized by intense pain and severe progressive tissue damage due to the unique properties of the acid that remains relatively non-ionized even at diluted concentrations. Unlike other acids, HF does, in fact, not cause significant surface damage but rapidly crosses lipid membranes and has a potent ability to diffuse into tissues, where it releases the freely dissociable fluoride ion, producing liquefaction and necrosis of the soft tissues and bone corrosion (4). This secondary deep tissues damage may continue for days, if not treated.

Delay in treatment is common in patients with HF burns due to the fact that the more diluted the solution, the more time it takes for the pain to appear after exposure, allowing time

for the acid to progress deeply (1). Surface decontamination is not sufficient as treatment, because HF-induced necrosis only stops when the acid is neutralized.

The strong pain that is typically associated with the skin damage is due to the capacity of fluorine ions to immobilize calcium ions in the tissues stimulating the nervous system.

The severity of HF burns depends on the concentration and total amount of HF (the solution may reach a strength of 75%), the surface area involved, the duration of contact and the penetrability and resistance of the tissues exposed to the acid. Solutions up to 20% HF may not produce erythema and pain until late as 24 hr after exposure. Therefore, contact with diluted solutions is especially dangerous, because it may go unnoticed until patient develops pain and tissue necrosis. These burns are the most common presentation of HF burns.

Death due to severe HF burns, involving a large surface of the skin, has been documented (5). Severe multiple organ failure due to the direct or indirect effects of local or systemic fluoride toxicity has also been reported. Death may also occur as a result of the consequences associated with fluoride ion poisoning, such as arrhythmias, hypocalcaemia and hypomagnesaemia and are favoured by the delay in the diagnosis and treatment. Sudden death and fatal cardiac arrhythmias, such as ventricular fibrillation, may rarely occur (2).

Certain areas of the body, such as the palms and the soles, are relatively resistant to the action of HF due to the thickness of their horny layer, while others, such as the subungual tissues, are rapidly penetrated, because the onychocorneal band, which attaches the distal plate to the nail bed, possesses a very thin horny layer (6). Once reached the subungual space, if allowed to progress, HF may destroy a great deal of the soft tissue and bone of the distal phalanx, because the process may go on for months. Painful onycholysis is a typical sign of HF burns, which frequently affects the digits and the subungual areas (6). Onycholysis may be preceded by blue green discolouration of the distal nail plate (7).

The use of gloves does not necessarily protect from HF burns, because the chemical may pass through pinholes in the rubber gloves (3).

According to the series reported by Hatzifotis et al. (1), 40% of HF are due to cleaning products utilized at



Fig. 2. Clinical improvement after applying 2.5% calcium gluconate gel directly on the base of the lesions.

Table 1. Calcium gluconate gel for cutaneous exposure to hydrofluoric acid

For 100 g	Gram
Calcium gluconate powder	2.5
Lidocaine chloride	2
Methyl hydroxy ethyl cellulosa	5
(Tylose MH 300 – Hoechst R)	
Propyl glycol	15.5
Water	75

home or boat and 24% are work related. Development of onycholysis is usually very rapid, in our patient it occurred 12 hr after exposure to HF due to the low concentration of the acid in the anti-rusting agent.

When HF burns do occur, prompt recognition and specific initial treatment are extremely important. After dilution and removal of the acid with water, the treatment of choice is, in case of peri- and subungual burns, calcium gluconate. Calcium ions can neutralize the fluoride ions forming insoluble calcium fluoride but should be applied after debridement of the lesions to allow deep penetration of the antidote and prevent the development of more serious burns. Cessation of pain can be considered as a sign of correct intervention.

The addition of dimethyl sulphoxide to the calcium gluconate gel may help the gel to reach the affected areas, when onycholysis is severe and the nail plate can not be removed (1).

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Jellyfish dermatitis caused by *Porpita pacifica*, a sign of global warming?

Contact Dermatitis 2005; 52: 232–233

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Key words: global warming; jellyfish dermatitis; *Porpita pacifica*.

A 37-year-old surfer, enjoying surfing frequently and having enough knowledge about jellyfish, felt a painful sensation on the left forearm while paddling along the coastline of Chiba Prefecture, which faces the Pacific Ocean. He found many *Porpita pacifica* Lesson (raft hydroid) in the area. Although there was no eruption on the first day, pruritic erythema appeared the next day. Itchy papules and vesicles developed on the erythema 4 days later. Although the patient had experienced contact with *P. pacifica* several times recently, he had never noted such eruptions. The eruption spontaneously subsided 10 days later.

Porpita pacifica (order Chondrophora, class Hydrozoa, phylum Cnidaria) is found in the warm areas of the Pacific Ocean. The jellyfish consists of a disk, gastrozooids and dactylozooids. The gonozooids appear during the breeding season. *Porpita pacifica* is called 'Ginkakurage', meaning 'silver coin jellyfish', in Japan. While generally not very hazardous to humans, this venom can cause mild eruptions. This is the first reported case of dermatitis caused by this jellyfish.

The number of *P. pacifica* observed in Japan has skyrocketed recently. On 2 September 2004, an estimated 9000 *P. pacifica* were washed ashore along a 300 m long stretch of Kitahama beach in Shirahama, Wakayama Prefecture (S. Kubota, unpublished

observation). Until 2002, the most ever found at this location was just 363 on 27 April 1996 (1). On 31 July 2003, however, 1213 were found (2). Very recently, *P. pacifica* began appearing in large numbers along the coastline of the Sea of Japan, in the northern region of Japan. The sudden increase in *P. pacifica* around Japan is probably due to higher water temperatures, which reflect global warming. Similar northward drift has also been observed for at least three kinds of jellyfish: *Eugymnanthea japonica* Kubota, *Eutima japonica* Uchida and *Nemopilema nomurai* Kishinouye. Also in the Atlantic Ocean, marine biologists have discovered a northward shift of plankton species by global warming (3, 4) and predicted that future warming will change the spatial distribution of primary and secondary pelagic production (4).

Skin injuries from jellyfish are due to toxic and/or allergic reactions (5). The present case showed pruritic erythematous papules and vesicles 4 days later, although the previous several contacts with *P. pacifica* produced no eruptions. It is therefore conceivable that this is delayed-type allergic reaction. Purification of the jellyfish toxin(s) should be needed for further investigation. Among the many jellyfish toxins, some of the most dangerous are produced by *Chiropsalmus quadrigatus*, *Carybdea alata* and *Carybdea rastoni*, all of which were recently characterized (6). These toxins are novel, heat-labile proteins and are homologous.

Dermatitis from *P. pacifica* may increase in the future, and if so, it may be a sign of global warming.

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Allergic contact dermatitis from triethanolamine polypeptide oleate condensate in eardrops and shampoo

Contact Dermatitis 2005; 52: 233

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Key words: allergic contact dermatitis; Cerumenex[®]; eardrops; oleyl polypeptide; shampoo; Soropon[®]; triethanolamine polypeptide oleate condensate; Xerumenex[®].

Case Report

A 62-year-old woman developed a severe, oedematous and oozy dermatitis of the right ear canal, with extension to the external ear and to the right side of her face and neck. This occurred within 72 hr of a first exposure to Cerumenex[®] eardrops (Purdue Pharma, Pickering, Ontario, Canada). The lesions resolved after a few days of treatment with oral prednisone.

8 years previously, the patient had experienced a similarly severe dermatitis of the scalp, face and neck after using Soropon[®] shampoo. This product, no longer available, was also manufactured by Purdue Pharma, and its composition was very much the same as Cerumenex[®] (Table 1).

The patient was patch tested to Cerumenex[®] eardrops 'as is' as well as to the NACDG standard series and to a cosmetic series (Chemotechnique Diagnostics AB, Malmö, Sweden) using Finn Chambers[®] (Epitest Ltd Oy, Tuusula, Finland) on Scanpor tape (Norgesplaster, Vennesla, Norway). At D2 and D4, she had a brisk 2+ reaction to Cerumenex[®] but did not react to propylene glycol 10% pet. or to triethanolamine 2% pet.

The manufacturer kindly provided the individual ingredients of Cerumenex[®], diluted in propylene glycol and water at the concentration present in the finished product. The patient underwent a second series of patch tests and, at D2 and D4, she displayed 2+ reactions to triethanolamine polypeptide oleate condensate 10% and to oleyl polypeptide 8.7%, the latter being a breakdown product of the former.

Discussion

As the name implies, Cerumenex[®] (Xerumenex[®]) is a topical preparation to dissolve earwax. A few cases of allergic contact dermatitis have been reported and the allergen identified as the triethanolamine polypeptide oleate condensate (1–7). Our case confirms this finding.

Interestingly, the patient described by Dooms-Goossens et al. seems to have become first sensitized to Xerumenex[®] eardrops and later reacted to a shampoo containing

potassium-coco-hydrolysed animal protein (PCHAP). Patch testing revealed 2+ reactions to PCHAP, triethanolamine-coco-hydrolysed animal protein, potassium undecylenoyl hydrolysed animal protein and triethanolamine polypeptide oleate condensate. Cross-reactions between these hydrolysed animal protein derivatives seem to be common. Our case is very similar to the patient described by Dooms-Goossens et al. except that the sensitization to the shampoo came first.

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Table 1. Ingredients of Cerumenex[®] eardrops and Soropon[®] shampoo

Cerumenex [®]	Soropon [®]
Triethanolamine polypeptide oleate condensate 10%	Triethanolamine polypeptide cocoate condensate 12%
Water	Water
Chlorobutanol 0.5%	Thyrothricin 0.1%
Propylene glycol	Propylene glycol
Triethanolamine	Lactic acid
	Methyl paraben
	Propyl paraben
	Fragrance

Mud bath dermatitis due to cinnamon oil

Contact Dermatitis 2005; 52: 234

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A case of long-lasting, extensive eczematous and bullous dermatitis affecting exposed areas (arms and legs), beginning within 24 hr after having a mud bath with cinnamon essential oil in a spa, in a 74-year-old woman, is reported. Patch tests with the GEIDC standard battery and the dental battery (including clove essence and eugenol), cinnamon essence and its components were carried out 5 years later. Fragrance mix, cinnamon essence, eugenol, cinnamic alcohol and cinnamic aldehyde yielded a positive result. To our knowledge, this is the first case of cinnamon dermatitis after a mud bath.

Key words: adverse effects; cinnamon; contact dermatitis; mud therapy; stomatitis.

Cinnamon (*Cinnamomum zeylanicum*) is a widely used spice in the feeding, cosmetic and pharmaceutical industries. Adverse reactions with their use have been described: carcinogenesis, dermatitis (frequently occupational) (1–4) and stomatitis (5).

Case Report

A 74-year-old woman reported that, 5 years before, within 24 hr after

amud bath in a spa, she experienced an extensive eczematous and bullous dermatitis affecting all exposed areas (arms and legs), with desquamation and residual hyperpigmentation. That dermatitis lasted several months. The patient was referred to another hospital in which she received steroid treatment. Information about the mud composition was required to the medical department of the spa: only cinnamon essential oil was added to the mud. No further studies were carried out at that time. Since then, although the patient has avoided any kind of mud bath, she continues showing oral symptoms (burning mouth with chronic papulous and aphtous lesions).

Patch tests were performed with the GEIDC (Spanish Contact Dermatitis Investigative Group) standard battery and dental battery (including clove essence and eugenol). Only fragrance mix was positive (++ at D2 and D4). Testing with cinnamon essence and its components showed reactions to cinnamon essence (negative at D2 and ++ at D4), eugenol (+ at D2 and D4), cinnamic alcohol (+++ at D2 and D4) and cinnamic aldehyde (+++ at D2 and D4).

Despite cinnamon avoidance, oral symptoms ameliorated only partially. That incomplete improvement caused the search for alternative sources of cinnamon exposure. A significant improvement was achieved after avoiding confectionery products and cola drinks.

Conclusions

A case of generalized dermatitis after a therapeutic mud bath with cinnamon essential oil is described. Although the sensitizing power of cinnamon is

well known (1–3), symptoms and signs are often indolent (aphtous stomatitis) (5), and the possible sources of exposure are very extensive and often hidden, making the diagnosis and the avoidance difficult. Positive patch tests in the standard battery to fragrance mix (2) could lead to cinnamon allergy diagnosis in those cases. To our knowledge, this is the first case of cinnamon dermatitis after a mud bath. Whether the patient had symptoms with cinnamon before that episode is unclear, but chronic oral symptoms due to hidden cinnamon exposure are still present.

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