

CONTACT POINTS

CONTACT DERMATITIS 2005; 52: 108–119

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Chloracne due to *o*-dichlorobenzene in a laboratory worker

Contact Dermatitis 2005; 52: 108

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Key words: chemical laboratory; chloracne; *o*-dichlorobenzene; occupational.

Case Report

A 27-year-old man presented with a history of acne after beginning his research for the graduate dissertation in a chemical laboratory. His work involved manipulation of *o*-dichlorobenzene once a day and no contact with any other chemical substances except acetone. 3 months after starting this work, he had begun to show initial manifestations of acne and had then accidental hand contact with *o*-dichlorobenzene because of a broken vial. Soon after this episode, acne symptoms worsened, prompting him to seek medical advice.

On examination, there were open comedones and straw-coloured cysts on the cheeks and malar crescent. No lesions were apparent on the forehead, the neck or the chest. Blood tests were normal and there was no evidence of other diseases.

The patient was removed from further exposure, treated with systemic antibiotic therapy, and after 3 months showed evident improvement. In order to help confirm the role of *o*-dichlorobenzene exposure, passive diffusion samplers (Radiello[®], Aquaria, Milano, Italy) were placed in the laboratory for the duration of the work shift, desorbed with CS₂ and then analysed by means of gas chromatography coupled with

mass spectrometry. *O*-dichlorobenzene present in the air was in the range of 0.8–2.2 p.p.b., that is hundreds of times lower than the ACGIH TLV-TWA of 25 p.p.m.

Discussion

O-dichlorobenzene is well known for its toxicity to the liver; chloracne because of *o*-dichlorobenzene exposure has been less studied. Vazquez et al. (1) reported 9 patients of chloracne in a factory that produced chemicals based on monochlorobenzene, *o*-dichlorobenzene and *p*-dichlorobenzene. In this instance, exposure to these chemicals was at 15 p.p.m., which is still lower than the ACGIH TLV.

Several chlorinated compounds have been associated with chloracne in humans. However, the degree of halogenation does not necessarily determine toxicity, whereas the position of halogen atoms on the outside of the molecule seems to be crucial (2).

In the case of the patient observed, chloracne seems to have been caused by very low exposure, possibly enhanced by accidental cutaneous contact increasing skin absorption.

To our knowledge, this is the first case of chloracne reported in a laboratory worker exposed to such low levels of *o*-dichlorobenzene. This should prompt clinicians and occupational physicians to pay attention even to low-level exposure to compounds associated with chloracne.

Acknowledgements

We are grateful to Chiara Scardoni for editing.

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Allergic contact dermatitis due to MDI and MDA from accidental occupational exposure

Contact Dermatitis 2005; 52: 108–109

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Key words: 4, 4'-diphenylmethane diisocyanate (CAS 101-68-8); allergic contact dermatitis; diaminodiphenylmethane (CAS 101-77-9); occupational.

Accidental exposure to a chemical may induce contact allergy manifesting as allergic contact dermatitis. We report here a female patient who was occupationally sensitized to both 4,4'-diphenylmethane diisocyanate (MDI) and diaminodiphenylmethane (MDA) from a single accidental exposure.

Case Report

A 33-year-old woman had worked in the manufacture of electronic

components since 1997. She had had allergic rhinitis since childhood, but no other atopic diseases. Her duties included assembling circuit boards and thereafter casting the boards with a casting mixture, the hardener of which contained more than 98% MDI. She used disposable vinyl gloves, a protective respiratory device and an apron only during the casting task, which was done about 2× a week.

In September 2000, she splashed the hardener onto the skin of her chest, abdomen and feet when opening a can of it. Eczema developed on these skin areas in the next 2 weeks. She visited an occupational health physician who recommended corticosteroid ointment and antihistamine. Her eczema worsened in spite of this treatment, and she was sent for a dermatological consultation to the local hospital. Prick testing elicited immediate allergy to birch pollen, hay pollen and animal dander, and patch testing was positive only to MDA. Specific radioallergosorbent tests to MDI, TDI (toluene diisocyanate) and HDI (1,6-hexamethylene diisocyanate) were negative.

Thereafter, she was splashed with the same hardener 2× more on her skin, and similar eczema developed again on these areas. Her skin was symptomless when away from exposure. In addition to eczema, she had work-related nasal and respiratory symptoms which she could not connect with any specific agent.

Later, she was referred to the Finnish Institute of Occupational Health (FIOH). At the FIOH, prick testing showed the same environmental allergies as before, and prick tests to diisocyanates were negative. Extensive patch tests elicited allergic reactions to both MDI [MDI 1.0% pet. (Trolab[®], Hermal, Reinbek, Germany): D2 –, D3 ?+, D4 ?+ MDI 2.0% pet. (FIOH): D2 ?+, D3 ++, D4 ++] and MDA [MDA 0.5% pet. (FIOH): D2 ?+, D3 ++, D4 ++]. Inhalation challenge tests with MDI were performed, but were negative.

Discussion

Diisocyanates are widely used in the manufacture of various polyurethane products. They are also used as hard-

eners, as in our case. Of the diisocyanates, exposure to MDI, and also to TDI and to HDI, has been reported to cause occupational allergic contact dermatitis (1–3).

Chemicals known to induce sensitization and allergic contact dermatitis after a single accidental exposure include epoxy resin, polyfunctional aziridine, methyl acrylate, phenol-formaldehyde resin and methylchloroisothiazolinone (4). Recently, diisocyanates have also been reported to be skin sensitizers as the result of a single occupational exposure. Goossens et al. (3) have reported a 31-year-old maintenance technician from a car factory, who was accidentally in contact with isocyanates and developed hand eczema. Similar to our patient, he was sensitized to both MDI and MDA.

In line with Goossens' report, our patient demonstrates that diisocyanates can be highly sensitizing, and that the sensitization can occur from a single accidental exposure.

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Contact hypersensitivity to topical corticosteroids – frequency of positive reactions in patch-tested patients with allergic contact dermatitis

Contact Dermatitis 2005; 52: 109–110

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Key words: contact hypersensitivity; corticosteroids; cross-reactivity; patch tests; relevance.

Recently, contact hypersensitivity to topical corticosteroids has become common (1). However, little is known about this problem in Poland (2). We therefore evaluated the frequency of positive patch-test results with corticosteroids in consecutive patients with allergic contact dermatitis (ACD).

Patients were patch tested with 8 corticosteroids in petrolatum: amcinonide 0.1% (AMC), bethametasone-17-valerate 0.12% (BV), clobetasol-17-propionate 0.25% (CP), hydrocortisone 1%, hydrocortisone-17-butyrate 0.1% (HB), acetamide triamcinolone 0.1%, budesonide 0.1% (BUD) and prednisolone 1% (PRED) (Trolab; Hermal, Reinbek, Germany). Of 257 patients tested, 19 (7.4%) had positive reactions to corticosteroids: 10 subjects positive for BUD, 7 for AMC, 7 for HB, 5 for BV, 4 for CP and 1 for PRED. Positive reactions to HB were clinically relevant in all cases, to BV in 3, to CP in 2, to BUD and AMC only in one case.

12 subjects had concomitant allergy to other allergens, in 6 of whom the allergy was polyvalent. 6 patients with positive patch-test results with BUD showed reactions to HB, 1 of them also to CP. 4 patients had simultaneous reactions to AMC and BV, 2 of them also to CP. Only 2 simultaneous reactions to AMC and BUD were observed.

The age of patients with allergy to corticosteroids (45, 54 and 69 for Q1, Me and Q3, respectively) was significantly higher than that of the remaining ACD patients (28, 44 and 58,

respectively; $P = 0.0046$, Wilcoxon test), and their skin lesions were more frequently widespread or localized to the legs (12/19). There were no differences between these groups concerning sex of patients, duration or localization of skin lesions.

This study supports other clinical evidence that corticosteroids are frequent contact allergens. Elderly patients with extensive skin lesions are more apt to develop a corticosteroid contact allergy. Clinical relevance of positive reactions to BUD and AMC was found in only a few cases.

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Allergic contact dermatitis from topical piketoprofen

Contact Dermatitis 2005; 52: 110–111

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Key words: allergic contact dermatitis; lack of cross-sensitivity; medicaments; non-steroidal anti-inflammatory drugs; photoallergic contact dermatitis; piketoprofen.

During recent years, the topical use of non-steroidal anti-inflammatory drugs (NSAIDs) has shown a considerable increase, resulting in allergic and photoallergic contact dermatitis from NSAIDs as emerging problems in developed countries.



Fig. 1. Acute eczematous reaction due to piketoprofen.

Case Report

A 73-year-old woman, with a past history of chronic venous insufficiency, applied Calmatel[®] cream 2× per day (Almirall Laboratories S.A., Barcelona, Spain) on her left leg, prescribed by her family physician. 5 days later, she developed an acute eczematous reaction, with intense itching, on the left lower leg and on the inner aspect of the right lower leg (Fig. 1). She denied sun exposure at that time. Calmatel[®] cream application was stopped and topical and systemic corticosteroids were prescribed, resulting in healing of the lesions within 4 weeks. Patch testing was performed with the Spanish standard series (27 allergens) and an additional study of NSAIDs battery (26 allergens) plus the active principle of Calmatel[®] cream, piketoprofen 1% and 2% pet., supplied by Almirall Laboratories and tested as recommended in the literature (1), showed positivity (++) only to piketoprofen at 2 and 4 days. 15 controls were tested with negative results.

Discussion

Piketoprofen, 2-(3-benzoylphenyl)-alpha-methylacetamide)-4-methyl-pyridine, is a non-steroidal anti-inflammatory drug, a derivative of the arylpropionic acid group, which prevents the synthesis of prostaglandins and other prostanoids through competitive and reversible inhibition of cyclooxygenase (2). In Spain, it is distributed as aerosol, cream and gel formulations like Calmatel[®] under licence for topical use (3).

To the best of our knowledge, only 1 previous case of allergic contact dermatitis from piketoprofen has been reported (1), as well as a single case of photoallergic contact dermatitis reported by our group (4). We have confirmed the lack of any cross-sensitivity with other NSAIDs, including arylpropionic acid derivatives, previously found. 8 patients with photoallergy due to piketoprofen were reported by Ortiz de Frutos and Alomar (Third Latin Meeting of Allergic Contact Dermatitis, unpublished communication) among 153 patients investigated in 2 Spanish hospitals during 1985–96, but no cases of allergic contact dermatitis from piketoprofen were seen by these authors.

Finally, we consider that piketoprofen 1% and 2% pet. are valid to demonstrate allergic contact dermatitis due to this NSAID.

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Emla[®] cream-induced allergic contact dermatitis in a child with thalassaemia major

Contact Dermatitis 2005; 52: 111

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Key words: allergic contact dermatitis; children; Emla[®] cream; medicaments; prilocaine.

Case Report

A 9-year-old boy of Afghan origin with beta-thalassaemia major requiring 5 times weekly subcutaneous infusions of desferrioxamine developed an eczematous rash at sites of needle insertions. The areas involved were mainly over the abdomen. He had been having Emla[®] cream applied to the injection sites for over a year prior to the onset of dermatitis. He had no history of atopy.

Initial patch testing 4 months later elicited an allergic patch test reaction to Emla[®] cream (AstraZeneca, Luton, UK), and he was subsequently patch tested to Emla[®] cream again, as well as to its constituents including prilocaine and lidocaine and other caines in the medicaments series. Parabens are commonly used as preservatives in local anaesthetic preparations and were also tested. Tests were read on days 2 (D2) and 4 (D4) and repeat patch testing confirmed a positive response to Emla[®] cream and prilocaine (Table 1). Lidocaine and other caines were negative.

Table 1. Patch test results with Emla[®] cream, its constituents and other caines in medicament series

Allergens	Readings	
	D2	D4
Emla [®] cream	++	++
Lidocaine hydrochloride 15% pet.	–	–
Prilocaine hydrochloride 5% pet.	++	++
Arlatone 10% pet.	–	–
Carbomer 10% aq.	–	–
Cinchocaine hydrochloride 5% pet.	–	–
Tetracaine hydrochloride 2% pet.	–	–
Paraben mix 16% pet.	–	–

D2, day 2; D4, day 4.

Discussion

The eutectic mixture of local anaesthetics (Emla[®] cream) provides dermal anaesthesia through the release of lignocaine and prilocaine from the cream into the epidermal and dermal layers of the skin. Its topical anaesthetic and analgesic properties have led to many therapeutic uses, including single application before procedures such as venepuncture and minor dermatological procedures, as well as repetitive long-term use for amelioration of pain during cannulation in patients undergoing long-term haemodialysis. Cutaneous adverse effects have been reported though Emla[®] cream-induced allergic contact dermatitis is rare (<0.1%) (1).

Both lignocaine and prilocaine are members of the amide group of local anaesthetics with marked structural similarity. Allergic reactions are rarely encountered by the members of this group, unlike the older ester group, e.g. procaine. Allergic contact dermatitis from prilocaine has been reported from Emla[®] cream (2, 3), though there appear to be no reports in the literature of it occurring in this particular context. Le Coz et al. (4) report a case occurring in a haemodialyzed patient, but this is the first documented case in a young patient with a haematological disorder requiring regular iron chelation injections. The patient is now receiving topical anaesthesia with Ametop[®] (tetracaine 4%; Smith and Nephew, Hull, UK) with no problems as yet.

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Contact dermatitis caused by allergy to dorzolamide

Contact Dermatitis 2005; 52: 111–112

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Key words: allergic contact dermatitis; dorzolamide; eyedrops; ophthalmics; patch-testing technique; scarification; stripped skin.

Case Reports

We report 2 cases of allergic contact dermatitis from dorzolamide in 2 71-year-old men. Patient no. 1 had had bilateral open-angle glaucoma and used timolol, pilocarpine and dorzolamide hydrochloride (Trusopt[®]; Merck Sharp and Dohme, Madrid, Spain) eyedrops for 2 years, developing dermatitis on the eyelids. Treatment with Trusopt[®] was stopped, with clearance of the

lesions in 1 month. Patient no. 2 presented with a 4-week history of eyelid dermatitis. He had glaucoma and had been using topical Timofitol[®] (Merck Sharp and Dohme) and Trusopt[®]. This treatment was stopped with clearance of the lesions in 3 weeks.

Both men were patch tested to the European standard series, Trusopt[®] eyedrops and their ingredients, including hydroxyethyl cellulose, sodium citrate dihydrate, mannitol and dorzolamide hydrochloride 5%, in both petrolatum and water, with stripped (tape-stripping of the upper stratum corneum by 10-fold application and removal of conventional adhesive tape) (1) and scarified (scratching epidermis with lancet) (2) skin, to simulate the higher percutaneous absorption around the eyes. The 2nd patient was also tested to Timofitol[®] eyedrops and timolol 10%. Positive reactions (++) were obtained on days 2 (D2) and 4 (D4) in both patients to dorzolamide 5% only at the scarified site. 10 control patients were tested with negative results. An accidental conjunctival provocation test to Trusopt[®] was positive when the 1st patient's wife administered it to him by mistake. A conjunctival provocation test in the 2nd case to Trusopt[®] was also positive.

Discussion

Dorzolamide is a carbonic anhydrase inhibitor associated with contact dermatitis among topically administered drugs for glaucoma (3). We report 2 cases of contact dermatitis from dorzolamide diagnosed by scarified patch-test technique and conjunctival provocation test. There are a few previous reports of contact dermatitis from dorzolamide, but in only 1 case is stripping used for diagnosis. To the best of our knowledge this is the 1st such report using scarified patch-test technique for diagnosis.

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Localized erythema multiforme-like contact dermatitis from laundry bar soap

Contact Dermatitis 2005; 52: 112–113

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Key words: colophonium; contact allergy; erythema multiforme-like contact dermatitis; laundry bar soap.

Case Report

A 25-year-old non-atopic pregnant woman presented with recurrent itchy, urticarial plaques and target-like lesions on the palms and soles for the last 6 months. The complaints had started 1 month after using a new brand of laundry bar soap. The

lesions continued to appear despite changing the brand of soap, and there was definite correlation between the occurrence of lesions and contact with soap.

On examination, there were multiple discrete targetoid lesions resembling erythema multiforme (EM) on both palms and soles (Fig. 1). All lesions were confined to the area coming into contact with the soap. There was no extension of lesions to surrounding areas or any dissemination. There were no mucosal lesions. The patient was not on any medication, and there was no history of herpes infection.

We patch tested her only with 1% aqueous soap solutions (both brands), and she was ++ to both soap dilutions on D4 (Fig. 2).

Discussion

Several allergens have been reported to cause an EM-like eruption, including *p*-phenylenediamine, natural rubber latex, sesquiterpene lactones, textile dyes, hair lotions, cutting oils, etc. (1–4). EM-like contact eruption is believed to be an id-like reaction occurring with powerful sensitizers, penetration-enhancing substances, high concentrations or repeated application of allergens (5).

Allergens in soaps include lanolin, perfumes, colophonium, antiseptics, dyes, surfactants and emulsifiers. Some yellow laundry bars owe their properties to colophonium, which consists of a mixture of complex abietic acids and esters (6). Contact dermatitis from colophonium in soap water has been reported previously



Fig. 1. Both palms showing erythema multiforme-like lesions.

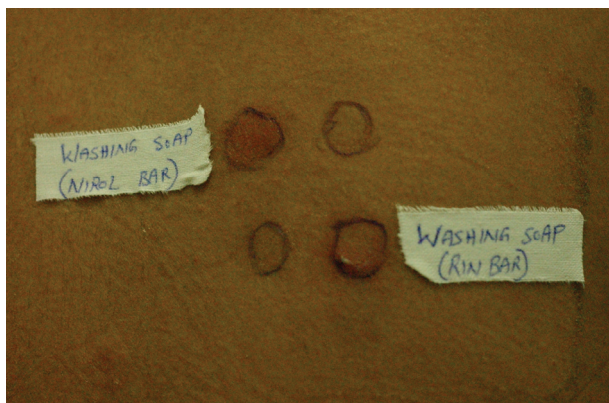


Fig. 2. Patch tests showing positive reactions to the laundry bar soaps (1% aq.).

(7). Colophonium forms an important allergenic agent in such washing soaps.

Localization to the soles relates to the habit of Indian women of washing clothes in shallow basins and sitting on a low stool with bare feet on the ground, leading to the feet becoming exposed to soap solution. Although there is a report of soap as a contact allergen, while being used in cutting fluid and producing EM (7), to the best of our knowledge, there are no reports of EM-type contact dermatitis secondary to laundry bar soaps.

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Contact dermatitis due to topical cosmetic use of vitamin K

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Contact Dermatitis 2005; 52: 113–114

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Key words: allergic contact dermatitis; cosmetics; topical use; vitamin K.

Vitamin K is a lipid-soluble vitamin used systemically in patients with hypoprothrombinemia or vitamin K deficiency and liver disease, or as an antidote to coumarin. Recently, a mixture containing vitamin K₁ and retinol has been marketed for topical use in the treatment of periorbital hyperpigmentation.

Case Report

A 27-year-old woman developed dermatitis of the face, particularly in the

periorbital area. For 4 months, she had used a cosmetic treatment of the face to decrease periorbital hyperpigmentation, Auriderm K2[®] cream (Auriga International S.A., Waterloo, Belgium), a mixture containing vitamin K₁ and retinol.

2 months later, she was patch tested with the Italian standard series using Finn Chambers[®] on Scanpor[®] (Epitest: Tuusula, Finland), and also with benzalkonium chloride 0.1% pet., oleamidopropyl dimethylamine 0.5% aq., propylene glycol 2% pet., phosphorus sesquisulphide 1% pet., triethanolamine 2.5% pet., retinol 0.01% pet. and 2 cosmetic products, Retinolo bioplus[®] face cream and Auriderm K2[®] cream, both as is.

Readings were made at 48 (D2) and 72 h (D3). Patch tests showed a positive reaction to Auriderm K2[®] cream.

The manufacturers, at our request, sent us the components of Auriderm K2[®] cream. Patch tests with the ingredients of this product were positive to vitamin K₁ 2% pet. (D2+/D3++) and *trans*-vitamin K 2% pet (D2+/D3++).

All symptoms and signs cleared after she stopped this cosmetic treatment.

Discussion

Vitamin K exists in 4 different forms: vitamin K₁ (phytomenadione), the natural form; vitamin K₂ (menaquinone) from intestinal bacterial synthesis; and vitamin K₃ (menadione) and K₄ (menadiol) as synthetic analogues (1). The lipid-soluble vitamin K (phytomenadione) causes more cutaneous reactions than its water-soluble analogues (2). In previous reports, cutaneous hypersensitivity reactions to injection of vitamin K were seen in patients with liver disease, suggesting a relationship (1). There are also reported cases of cutaneous reactions to vitamin K₁ after its intramuscular injection in patients who have it for hypoprothrombinemia without liver disease (1, 3). Reactions to water soluble vitamin K are less common, because vitamin K is absorbed through the skin (2).

Contact dermatitis due to vitamin K for topical use is uncommon and only a few cases of occupational contact dermatitis from vitamin K₃ have been so reported (4–6). All such cases

were in employees in veterinary and pharmaceutical laboratories who had contact with the vitamin in their work. In these cases, the form of vitamin K is always K₃ (menadione), which also, in high enough concentration, causes an irritant contact dermatitis (2).

No cases of allergic contact dermatitis due to vitamin K₁ for topical cosmetic use have previously been reported.

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Generalized exanthematous reaction with pustulosis induced by topical corticosteroids

Contact Dermatitis 2005; 52: 114–115

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Key words: acute generalized exanthematous pustulosis; medicaments; pustulosis; topical corticosteroids.

Case Report

A 33-year-old man had a personal history of allergic contact dermatitis from nickel, diagnosed 5 years earlier. He reported 1 or 2 episodes per month of pruriginous eczema-like eruptions on his hands over the past 5 years. He had been treated with topical corticosteroids: betamethasone valerate (Celestoderm®; Key Pharma; Spain) and methylprednisolone acetate (Adventan®; Schering; Spain) with toleration.

He consulted us with pruriginous lesions in flexural areas for 2 weeks. He was treated with fluocinolone acetonide and betamethasone valerate (Alergical®; Iquinoso Farma; Spain), betamethasone dipropionate (Diproderm®; Schering Plough; Spain) and betamethasone valerate and gentamicin (Cuatroderm®; Desarrollos farmac y cosmet; Spain). After 2 days, he developed a maculopapular rash, first in the flexural areas, then with spread to the legs, arms, abdomen and eyelids.

On examination, he had erythematous macules and plaques of annular and oval shapes. Some showed linear scaling, but no pustules were clearly seen (Fig. 1). No fever or other symptoms were noted. The patient denied the intake of other medications during the last 4 weeks.

Laboratory examinations (haemogram, biochemistry, bacterial and viral serologies, immunoglobulins A, E, G and M and anti-nuclear autoantibodies) showed no abnormalities.

In a lesional skin biopsy, under the stratum corneum and in the stratum spinosum some pools of neutrophils were found, which formed variable sized pustules. Some lymphocytes,

histiocytes and neutrophils were localized around the superficial dermal vessels. A Gram stain was performed, to rule out staphylococcal infection, which was negative.

The patient was then treated with oral prednisone, and the lesions cleared in 1 week.

Prick, intradermal and patch tests with the standard series recommended by the Grupo Español para la Investigación de la Dermatitis de Contacto (GEIDC), the suspected medicaments and other corticosteroids were performed. Prick and intradermal tests were considered as positive with a wheal reaction of 3 and 5 mm, respectively, together with a positive control with histamine and a negative control with saline. Patch tests were read after 2, 4 and 7 days, and reactions were scored as recommended by the GEIDC.

The standard series (TRUE Test™, Madrid, Spain) showed a positive reaction to budesonide. Patch tests with the suspected medicaments showed positive reactions to betamethasone valerate and fluocinolone acetonide (Alergical®), betamethasone dipropionate (Diproderm®), and betamethasone valerate (Cuatroderm®). They were negative for methylprednisolone acetate (Adventan®). Patch tests with the Aristegui Laboratories (Aristegui Laboratories, Madrid, Spain) series of corticosteroids (all 1% in ethanol) showed positive reactions to betamethasone 17-valerate, betamethasone dipropionate and betamethasone valerate (Table 1). Prick tests with the same corticosteroid preparations and with gentamicin were negative, all sites being read at 15 min, D2 and D4.



Fig. 1. Linear scaling present within erythematous macules.

Table 1. Patch test results with corticosteroids (1% ethanol) at 7 days of reading

Corticosteroid	Result
Betamethasone 17-valerate	++
Betamethasone dipropionate	++
Betamethasone valerate	++
Fluocortolone	–
Prednisone	–
Methylprednisolone	–
Hydrocortisone	–
Beclomethasone dipropionate	–
Flunisolide	–
Budesonide	++
Triamcinolone	–
Momethasone furoate	–
Deflazacort	–
Fluticasone dipropionate	–
Dexamethasone	–

From these results, we made a diagnosis of generalized pustular reaction, with histopathologic features of acute generalized exanthematous pustulosis (AGEP), in a patient with hypersensitivity to corticosteroids of class D. The patient had a Type IV cell-mediated immunoreaction as demonstrated by patch tests, prick and intradermal tests all being negative.

Discussion

False-negative patch test reactions can occur with corticosteroids and choice of vehicle may alter the patch test results (1, 2). Contact allergy to corticosteroids is of the delayed type (3). Local differences in prescribing habits may affect the frequency of corticosteroid allergy (4). *In vitro* studies have shown that the amount of degradation product, which subsequently reacts with arginine,

varies between corticosteroids and is correlated with the frequency of allergic contact dermatitis from a corticosteroid (5–7).

Acute generalized exanthematous pustulosis is an acute febrile drug eruption characterized by numerous small, primarily non-follicular sterile pustules, arising within large areas of oedematous erythema. Many cases have been confused with pustular psoriasis. More than 90% of these eruptions are drug-induced, and occasionally, AGEP may be seen as a hypersensitivity reaction to mercury or in association with an enteroviral infection. Topical corticosteroids have never before been reported as causative agents (8).

Histologically, spongiform pustules are seen within the most superficial layers of the epidermis, beneath the stratum corneum. (Fig. 2) Edema of the papillary dermis and a mixed perivascular infiltrate of neutrophils and some eosinophils are usually present. In a minority of cases, leucocytoclastic vasculitis and foci of necrotic keratinocytes have been observed.

Acute generalized exanthematous pustulosis must be differentiated from acute pustular psoriasis of the von Zumbusch type, exanthematous drug eruptions or pustular drug reactions, toxic epidermal necrolysis and drug rash with eosinophilia and systemic symptoms. We propose our case as the first report of a generalized pustular reaction induced by topical corticosteroids. Absence of fever or neutrophilia can occur in some cases of AGEP. The patient's lesions cleared with oral prednisone for 1 month. He remains asymptomatic.

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Occupational allergic contact dermatitis in a chemist from ethyl bromoacetate and bromoacetonitrile

Contact Dermatitis 2005; 52: 115–116

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Key words: allergic contact dermatitis; bromoacetonitrile; chemist; ethyl bromoacetate; occupational.

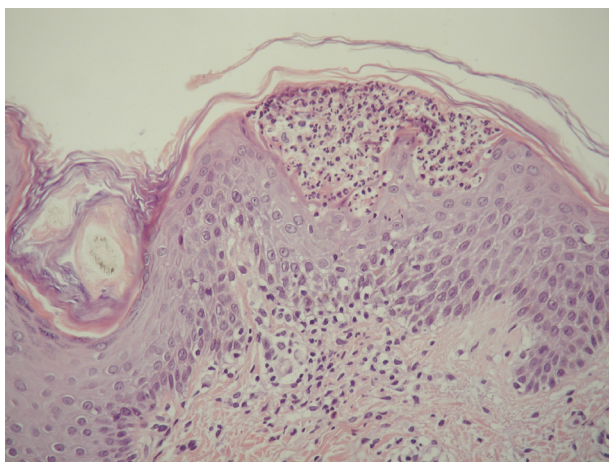


Fig. 2. Subcorneal pustules present in the epidermis. Hematoxylin and eosin ($\times 40$).

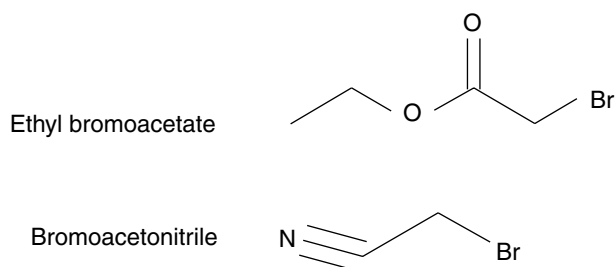


Fig. 1. Chemical structures of ethyl bromoacetate and bromoacetonitrile.

Ethyl bromoacetate and bromoacetonitrile are used in the pharmaceutical industry for the chemical synthesis of new products.

Case Report

A 28-year-old chemist, with no history of skin disease, working in a pharmaceutical laboratory, presented with eczema on the hands, with scaling and fissuring of the fingers. She was occupationally exposed to numerous chemicals and generally wore protective gloves in her work.

She was patch tested with the GEIDC standard series and with 26 different compounds that she was using at that moment in the laboratory. At D2 and D4, there were positive reactions (++) to ethyl bromoacetate (1% pet.) and bromoacetonitrile (1% pet.), but no reactions to any other substance. Patch tests in 25 controls were negative (Fig. 1).

After she was instructed to avoid further contact with ethyl bromoacetate and bromoacetonitrile, her eczema subsided within a few weeks. No recurrence was observed during 2-year follow-up.

Discussion

Because of the diversity and large number of skin-hazardous chemicals to which chemists may be exposed, it is impossible to give a comprehensive list of such chemicals (1). The diagnostic procedure in cases of contact dermatitis in these workers can therefore be considered a challenge. Allergic contact dermatitis in chemistry researchers often provides the first clue that a new chemical is a potential contact sensitizer. We report the first case of allergic contact dermatitis from

ethyl bromoacetate and bromoacetonitrile, used in the chemical synthesis of new pharmaceutical products.

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Severe allergic contact dermatitis to paraphenylene diamine in hair dye following sensitization to black henna tattoos

Contact Dermatitis 2005; 52: 116–117

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Key words: allergic contact dermatitis; henna tattoo; intensive care; paraphenylene diamine.

Henna is a vegetable dye used for hair colouring to give a reddish shade. It is obtained from the dried leaves of a shrub found in North Africa and the Middle East (1). Skin painting (pseudotattooing) with

henna is traditionally performed mainly in Muslim or Hindu persons. Allergic reactions to natural henna painting are extremely rare. Paraphenylene diamine (PPD) may be added to the mixture (black henna) to accelerate, darken and to increase the longevity of henna tattoos. This has led to increased reporting of cutaneous reactions after 'black' henna tattooing (2).

Case Reports

Case 1

A 15-year-old boy presented with facial redness after he had a permanent hair dye bleached. The next morning his whole face and neck were swollen (Fig. 1). A year before, he had a blistering reaction to a black henna tattoo. His temperature was 38°C and white cell count $15.4 \times 10^9/l$ (92% neutrophils). He developed respiratory difficulties and was admitted to intensive care for intubation and treated with intravenous steroids and antibiotics. His facial swelling settled and he was extubated and discharged.

Testing with PPD applied for 20 min showed severe blistering at 24 hr and was removed from standard series. Positive reactions to ammonium persulfate, 3-aminophenol, 4-aminophenol, hydrogen peroxide, cocamidopropyl betaine, caine mix and methylchloroisothiazolineone (Kathon CG) were recorded.

Case 2

A 14-year-old girl presented with severe facial swelling and redness few hours after a hair dye. She had a reaction to henna tattoos 2 years earlier. She stayed 3 days in hospital for treatment with oral steroids.

Paraphenylene diamine applied for 20 min developed a strong reaction at 4 hr and was removed from the standard series. Positive reactions to neomycin, formaldehyde, colophony, balsam of peru, methylchloroisothiazolineone (Kathon CG), 2,5-diaminotoluene sulfate, 3-aminophenol, 4-aminophenol and hydrogen peroxide were recorded.

Discussion

Temporary paint-on tattoos are becoming popular all over the



Fig. 1. Severe face and neck swelling after hair dye.

world. Such tattoos are marketed at holiday resorts and funfairs across Europe and the UK with children as the primary market. The tattoo makers use 'black henna', a mixture of many substances, among them PPD (concentration up to 15.7%) and/or diaminotoluene (3, 4). The long duration of skin contact, the high concentrations of sensitizing materials and the lack of a neutralizing agent increase the risk of skin sensitization (5). We report here 2 cases of severe sensitization to PPD following black henna tattoos. To our knowledge, this is the first report of such black henna-induced PPD sensitization, which has eventually caused a life-threatening reaction.

We would like to draw attention to the potential serious implications that children are being sensitized to PPD and might develop a severe allergic response. Paraphenylenediamine also cross-reacts with permanent hair dyes and rubber, which can affect future work prospects. Legislation might be needed to prevent the occurrence of similar cases.

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Occupational contact dermatitis due to a marbler's exposure to benzoyl peroxide

Contact Dermatitis 2005; 52: 117–118

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Key words: airborne contact dermatitis; allergic contact dermatitis; benzoyl peroxide; marbler; occupational.

Benzoyl peroxide (BPO) is frequently used as a topical treatment for acne, but allergic contact dermatitis has only rarely been reported in long-term-treated patients (1).

Benzoyl peroxide is used in the plastics industry, particularly as a hardener of polyester resin, and in the pharmaceutical industry as a therapeutic agent for acne (2).

Several cases of contact dermatitis due to occupational exposure to BPO have been reported, but never in marblers.

Case Report

A 48-year-old man, who had worked as a marbler for 30 years, with no personal history of atopy, presented with eczema on the face, predominantly and symmetrically involving the eyelids, and on the trunk, hands and forearms. Symptoms were clearly work related with complete clearing of the skin after stopping work. The distribution of the eczema, predominantly affecting exposed zones and extending to parts of the body protected by clothes, suggested an airborne mechanism.

Patch testing was performed on the upper back with a 2-day application and reading on the third with the European standard series, a series of plastics and glues (Chemotechnique) and materials handled at the workplace.

Positive reactions were observed with thiurams (D3++), PBO (D3+++), and a hardener used at the workplace (D3+++).

The patient used a hardener mixed with glue to stick the marble and then polished the material to remove the surplus and achieve a smooth appearance, resulting in a cloud of dust which very frequently irritated his nose.

Examination of the composition of the hardener, produced in Italy, showed that it contained BPO.

The patient was unable to obtain a change of job, which would have caused financial difficulties for the small company in which he worked.

Avoiding all contact with BPO allowed full recovery.

Discussion

Allergic contact dermatitis to PBO was reported by Dejobert et al. (3) in two podiatrists in contact with

PBO when they pumiced thermoplastic resin materials to make insoles.

Forschner et al. (4) reported airborne BPO contact dermatitis in an orthopaedic technician, who was in contact when heating and cutting plastic materials.

Bonnekoh and Merck (5) described a case of airborne contact dermatitis due to BPO in a sacristan in Cologne cathedral. Benzoyl peroxide was used as a bleaching agent in candle wax and evaporated as the candles burned.

Quirce et al. (6) reported a case of airborne contact dermatitis in an electrician, in whom BPO may have acted as a contact allergen after plastic particles became airborne when sawing plastic material, or perhaps as they melted during soldering.

We have not found any other cases of contact allergy to BPO in marblers in the literature regarding. This case therefore represents the first case of contact allergy to BPO confirmed by patch tests in a marbler.

Benzoyl peroxide is used as hardener of polyester resins for the production of glue or cake marble. Airborne dust is formed during polishing of marble and may enter the worker's clothes, as in the case reported here (explaining the lesions observed on the trunk).

We therefore believe that BPO must be systematically investigated in cases of airborne eczema in marblers.

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Ulcerative irritant contact dermatitis from lindane

Contact Dermatitis 2005; 52: 118–119

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Key words: Lindane; irritant contact dermatitis.

Case Report

The 57-year-old bedridden male with history of diabetes mellitus, chronic renal failure and old cerebrovascular accident was admitted to our Ear-Nose-Throat department due to deep neck infection. We were consulted for the generalized erythematous scaly papules mainly on the periumbilicus, axillae and groin area. Scabies was confirmed by scraping examination under microscope. We had educated his nursing assistant to apply lindane lotion on the patient's whole body surface, except the mucosal area for 8 h/day and then wash it off. The course was repeated for 3 consecutive days. However, the nursing assistant only washed off the lower half of the body during that period of time. Multiple scattered and coalescent geographical ulcerations with blackish burn-like crusted edges developed 1 week later (Figs 1 and 2) There were no



Fig. 1. Multiple scattered and coalescent polymorphic ulcerations with some haemorrhagic spots.



Fig. 2. Black crusted burn-like edges around these ulcerations.

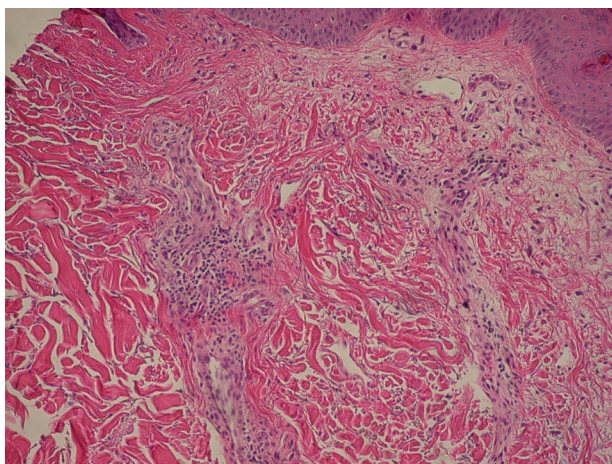


Fig. 3. Acute and chronic inflammation without eosinophil infiltrate or foreign body in dermis.

preceding vesicles nor bullae noted. The skin biopsy was showed ulcerations with papillary oedema and acute and chronic inflammation. There was neither eosinophil infiltrate nor foreign body found. (Fig. 3) We later performed a patch test. The results are unavailable because the patient expired from sepsis.

Discussion

Lindane, 1% Gamma benzene hexachloride, is commonly used to treat scabies in Taiwan. A 20% formulation used for control of borers and leaf miners is labelled as a skin irritant. No positive patch reactions to 1% lindane were obtain in 665 routine eczematous patients, patch tested in the previous study (1). The commercial product we used is Delice 1%, medical hair emulsion, that contains 1% gamma benzene hexachloride, 5% Comperlam LMD, 50% Triton X-200, 0.5% isopropyl myristate, 0.15% ethyl paraben, 0.05% ethylene diamine tetra-acetic acid disodium, 0.2% herbalialer HR and 43.1% reverse osmosis water. The value of pH is 8 ± 1 . Toxication of lindane including nausea, vomiting, convulsions, respiratory failure with cyanosis and even death have been reported. Chronic exposure may

also induce blood dyscrasias, decreased liver function, altered menses and cardiac arrhythmia (2–4). Other minor irritant symptoms including burning sensation, pruritus, erythema and vesiculation have been noted too. These adverse effects are uncommon and usually resulted from inappropriate use.

Diagnosis of irritant contact dermatitis (ICD) is a process of exclusion (5). In our case, the ulceration without preceding vesicle or bulla on occlusive site strongly indicates serious cumulative ICD. There is no other skin lesion on other contact area without occlusion. We also did not find any eosinophil infiltrate under microscope. ICD was preferably suggested than ACD according to clinical presentation. However, there was no ulceration caused by lindane reported in the previous literatures. The diagnosis will remain tenuous until other cases are identified.

The ICD is a prime disease of multifactorial causation including nature of irritating substance (pH, solubility and detergent action; gaseous, liquid or solid), host factors (occlusion, sweating, pigmentation, dryness, sebaceous activity and other skin disease) and environmental factors (temperature, humidity, friction and pressure) (5, 6). Its

develop depends on the balance between the sum of all harmful influence and repair capacity of the skin (7). The ulcerations occurred on the back, the dependant and occlusive site of the patient's usual position. Lindane lotion is not considered a corrosive substance that is sufficient enough to cause burn-like lesions in general conditions (5). Nonetheless, there appears to be fairly general agreement that prolonged exposure time, pressure and occlusion of lindane against the skin are the necessary conditions for lindane to cause an ulcerative irritant contact dermatitis.

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