

Short Communications

Clothing dermatitis from Naphthol AS

CHRISTOPHE-J. LE COZ¹ AND JEAN-PIERRE LEPOITTEVIN²

¹Unité de Dermato-Allergologie, Dermatologie Professionnelle & Photobiologie; ²Laboratoire de Dermato-Chimie associé au CNRS, Clinique Dermatologique des Hôpitaux Universitaires de Strasbourg, 1 Place de l'Hôpital, F-67091 Strasbourg, France

Key words: allergic contact dermatitis; CAS 92-77-3; CI 37505, Naphthol AS; clothing; patch testing technique. © Munksgaard, 2001.

Case Report

A 23-year-old woman, with a past history of dermatitis from costume jewellery, was referred with subacute eczema, which had begun 4 weeks earlier on her neck and décolleté and spread to the upper limbs. She reported the onset as a few days after she had worn a new blue woollen pullover. Patch tests with the European standard series, additional allergens, disperse blue dyes (Chemotechnique®), her nail lacquer and a piece of her pullover showed a + reaction to nickel sulfate only. As the patient had often worn another pullover with a zip likely to contain nickel, she was advised to avoid wearing this garment, and lesions cleared with topical corticosteroids.

Dermatitis relapsed acutely 2 months later, however, within 4 h of the patient wearing a strongly-coloured cotton scarf (black with a teddy bear motif) made in India, which had also been worn before the preceding episodes. Further patch tests with the garment and textile dyes and finishes series (Chemotechnique® and Trolab®), showed at D2 a +++ reaction to both the scarf and Naphthol AS 1% pet. (Trolab®). The presence of Naphthol AS in the scarf was confirmed by comparative thin-layer chromatography followed by high-performance liquid chromatography, after extraction with chloroform.

Discussion

Naphthols are coupling agents used to dye cotton in combination with diazonium salts. Because its lower affinity to cellulose, β -Naphthol (2-naphthol, C.I. Azoic coupling component 1) has been replaced by Naphthol

AS (Fig. 1) (1), also called Naphthazol A, 3-hydroxy-2-naphthalinide, 3-hydroxy-2-naphthoic acid anilide and C.I. Azoic Coupling Component 2 (CI 37505; CAS 92-77-3).

Naphthol AS, though not tested alone, has been reported as an occupational allergen in combination with Diazo Component 51 (2), and as a cross- or associated sensitizer with Pigment Red 23 in tattoos (3). Naphthol AS itself was first reported as a cause of pigmented contact dermatitis in textile workers in Mexico in the 1970s (4). It was identified as the cause of hand dermatitis in a needlewoman probably occupationally sensitized from clothes (5), and occasionally in other cases of textile dermatitis (6–8).

In some situations (6), and particularly in patients with high phototype (7, 8), Naphthol AS may cause pigmented contact dermatitis. Both the textile and Naphthol AS are generally positive on patch testing (5, 9). As some unreacted Naphthol AS may persist in fibres, even after washing (6, 7), the residual concentration can be as high as 4 mg/g (5), constituting a risk of sensitization.

We emphasize: (i) clothing dermatitis may not present with a typical pattern; (ii) Naphthol AS allergy is not detected by patch testing with 'standard' textile allergens, though it is included in the Trolab® textile dyes series; (iii) though tending to be used for cheap cotton fabrics made in emerging countries, the presence of Naphthol AS in such fabrics may be difficult to establish; (iv) contrary to textile dyes and resins, there is no legislation or labelling concerning Naphthol AS (11, D. Fuess, Institut Textile de France, personal communication); (v) though possibly still underestimated, the sensitization rate to Naphthol AS seems lower than to azo dyes, such as Disperse Blue 106 or 124 (10).

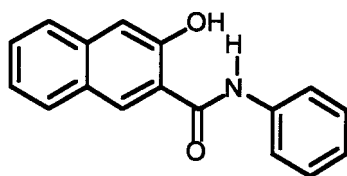


Fig. 1. Chemical structure of Naphthol AS.

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IgE-mediated allergy to castor bean dust in a landscape gardener

G. METZ, D. BÖCHER AND J. METZ

Klinik für Dermatologie und Allergologie, Städtisches Klinikum, HSK Wiesbaden, Ludwig-Erhard-Str. 100, 65199 Wiesbaden, Germany

Key words: castor oil seed; immediate-type allergy; organic fertilizer; agricultural occupational disease; contact urticaria; horticulture. © Munksgaard, 2001.

Case Report

A 38-year-old man, 15 years a landscape gardener, presented with a 2-year history of work-related symptoms. Without there being any atopic history, 10–20 min after exposure to castor-bean-containing organic fertilizers, he developed urticaria on the exposed skin, with conjunctivitis. Wearing protective goggles, breathing mask and gloves while working failed to prevent symptoms. However, he had no symptoms when working with animal horn dust or pure mineral fertilizer.

Prick tests with a standard series of inhalation allergens were negative and total IgE was within the normal range. On scratch testing, neither native nor hydrated castor oil was positive. However, scratch tests with natural castor bean and shredded castor bean were both positive: within 3 min, the patient showed a local urticarial reaction, after 20 min he developed urticaria on the neck, and after 40 min conjunctivitis and Quincke edema. With antihistamine, the symptoms cleared within 2 h. 3 controls scratch-tested were negative to castor bean seed. Specific IgE against castor bean was detected in the patient's serum (CAP-RAST: class 3).

Discussion

The castor oil plant (*Rizinus communis*) belongs to the Euphorbiaceae family of plants, and is cultivated commercially in India and South America. The castor oil seed (bean) is 50–60% oil and 18–20% protein. When pressed cold, pure castor oil is obtained, which is used widely in the pharmaceutical and cosmetic industries. With hot pressing and solvent extraction, a brownish-yellow oil is produced, which is used in other technical fields (colours, lacquers, synthetic materials, lubricants and textiles). The remaining mass after pressing can be

used in granular form in viniculture and as an additive in many organic fertilizers. It is not required by law to declare shredded castor bean in fertilizers. The manufacturers are asked only to advise caution to sensitive people handling such fertilizers.

There have only been a few reports recently on contact dermatitis from castor-oil containing products, such as lipsticks, make-up removers, ointments and military camouflage sticks (1–3). In contrast, respiratory allergies of the immediate type have frequently been reported from castor bean dust at work or environmentally (4–6). Castor bean dust allergy is also known in agriculture, horticulture and motorway-verge workers (7).

Allergic symptoms in those working on plant cultivation, which till now were thought to be related to the pollen count, may instead be induced by castor bean dust.

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Cold panniculitis – an unusual differential diagnosis from aluminium allergy in a patient hyposensitized with aluminium-precipitated antigen extract

GEORG MOOSER, HELMUT GALL[†], LUTZ WEBER AND RALF UWE PETER

Department of Dermatology, University of Ulm, Oberer Eselsberg 40, D-89081 Ulm, Germany

Key words: cold panniculitis; aluminium-precipitated antigen extract; differential diagnosis; aluminium allergy.
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Case Report

A 52-year-old woman, with an allergy to wasp venom, was hyposensitized to ALK-depot SQ802[®], containing wasp venom precipitated on aluminium hydroxide. Each subcutaneous injection of the extract was followed by cooling the acute local reaction (with ice packs wrapped in absorbent gauze) at the injection site for about 1 h. As soon as 24 h after the 2nd maintenance dose, the patient developed tender, erythematous, ill-demarcated, indurated plaques at the injection sites on both lateral thighs, as well as over the ventral and medial aspects of the thighs. The lesions resolved spontaneously after several weeks, leaving residual hyperpigmentation and subcutaneous atrophy. They reappeared with each treatment even after having changed the vaccine to a lyophilized, aluminium-free extract. No further lesions occurred when we stopped cooling the injection sites, even though we continued using the aluminium-hydroxide-adsorbed vaccine ALK-depot SQ802[®].

The patient was patch tested with the used vaccine ALK-depot SQ802[®] and its components, aluminium hydroxide 0.3% aq., human albumin and phenol 4.0% aq. In addition, she was tested with an empty and a petrolatum-filled Finn Chamber[®] and with aluminium chloride hexahydrate (AlCl₃ · 6H₂O) 1.0% aq., 2.0% aq. and 5% aq.: all tests proved negative. Histological examination of a biopsy of the skin lesions showed a lobular panniculitis, with necrosis of lipocytes, and a mixed cellular infiltrate of neutrophils, lymphocytes, and histiocytes. Ultrasound (7.5 MHz) examination of the atrophic areas showed significantly thinner subcutaneous tissue compared to normal contralateral skin (9.1 versus 13.0 mm). 24 h after rechallenge with an ice pack alone on the upper arm, the patient developed a similar skin lesion that corresponded histologically with cold panniculitis.

Discussion

At present, the most commonly-used hyposensitizing semi-depot extracts are aluminium-precipitated antigen solutions (1), the well-known local side-effects of which are transient subcutaneous nodules, which occur in about 20 to 30% of all patients. Less frequently, persistent symptomatic nodules are described (about 4%). Foreign body reaction to aluminium hydroxide, local delayed hypersensitivity to allergen and delayed hypersensitivity to aluminium (2) have all been proposed.

However, in our patient, the lesions described above derived from cold panniculitis, provoked by the application of ice packs – even though these ice packs were wrapped in absorbent gauze. With negative patch testing to the aluminium-precipitated antigen solution used and its components, well-tolerated subcutaneous injection of the same vaccine alone and reproduction of the skin lesions by application of the ice packs alone, we demonstrated histologically-confirmed cold panniculitis.

We could find no previous case report of cold panniculitis following subcutaneous injection of aluminium-hydroxide-adsorbed vaccines, even though the application of such ice packs is widely used in treating the acute local reaction after vaccination.

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Allergic contact dermatitis due to the β -blocker befunolol in eyedrops, with cross-sensitivity to carteolol

MASSIMILIANO NINO, FRANCESCO SUPPA, FABRIZIO AYALA AND NICOLA BALATO

Department of Dermatology, University of Naples Federico II, Via S. Pansini 5, 80131 Naples, Italy

Key words: eyelid dermatitis; β -blockers; befunolol; carteolol; allergic contact dermatitis; medicaments; ophthalmics; cross-sensitivity. © Munksgaard, 2001.

Case Report

A 68-year-old woman, with no history of atopy or allergy, had long been treated for open-angle glaucoma with eyedrops containing the β -blockers befunolol (Betaclar[®]) or timolol (Timoptol[®]), when she developed acute eyelid dermatitis, associated with hyperemia and conjunctival chemosis. Her condition rapidly improved after suspending befunolol and following therapy with topical corticosteroids.

Patch testing with the European standard series, a preservatives series and an ophthalmics series (including no β -blockers) was negative. On patch testing initially with the patient's own ophthalmics and then with ophthalmics containing befunolol or other β -blockers, the positive reactions in Table 1 were found.

Comment

Contact allergy to topical β -blockers is a well-recognized side-effect of glaucoma treatment (1, 2). Sensitization may be singly to agents such as timolol (3–6), befunolol (7), levobunolol (8), or, more rarely, to multiple β -block-

ers in a single patient (9, 10). Our patient developed contact allergy after many years of befunolol exposure and patch testing appeared to demonstrate cross-sensitivity to carteolol. Evidence of such cross-reactivity has not previously been reported (11, 12).

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Table 1. Patch test results

		D2	D3	D6
Betaclar [®] (befunolol HCl)		++	+++	+++
Timoptol [®] (timolol maleate)		–	–	–
Betoptic [®] (betaxolol HCl)		–	–	–
Carteol [®] (carteolol HCl)		++	++	++
Turoptin [®] (metipranolol HCl)		–	–	–
Vistagan [®] (levobunolol HCl)		–	–	–
befunolol	1% aq.	+	++	++
betaxolol	1% aq.	–	–	–
carteolol	1% aq.	+	+	++
levobunolol	1% aq.	–	–	–
metipranolol	1% aq.	–	–	–
timolol	1% aq.	–	–	–

Simultaneous photocontact sensitivity to ketoprofen and oxybenzone

AKIRA KAWADA, YOSHINORI ARAGANE, MUTSUO ASAI AND TADASHI TEZUKA

Department of Dermatology, Kinki University School of Medicine, Ohno-Higashi 377-2, Osaka-Sayama City, Osaka 589-8511, Japan

Key words: photocontact sensitivity; ketoprofen; oxybenzone; benzophenone-3; cross-sensitivity; medicaments. © Munksgaard, 2001.

A 21-year-old woman was seen in August 2000 with pruritic well-demarcated rectangular erythema on the flexor aspect of the right arm. In July 2000, she had started using a poultice (Miltax®, Daiichi Pharmaceut. Co. Ltd., Tokyo, Japan) containing 0.3% ketoprofen. 3 weeks later, she had developed erythema where the poultice had been applied.

Patch and photopatch tests (1) were performed on the inner aspect of the upper arm with ketoprofen 0.5% and 5% pet. and 4 constituents of the base, oxybenzone (benzophenone-3) 0.1%, 1% and 10% pet., thymol 1% and 10% pet., trisodium ethylenediaminetetraacetate 1% and 10% pet., and castor oil 1% and 10% pet. 4 other related chemicals, tiaprofenic acid 5% pet., zaltprofen 5% pet., 5% ibuprofen ointment, and 1% sprofen ointment, were also patch and photopatch tested.

All the above substances were negative at D2 and D3 on patch testing. On photopatch testing, ketoprofen 0.5% and 5% pet., oxybenzone 0.1%, 1%, and 10% pet., and tiaprofenic acid 5% pet. elicited erythema and papules 1 day after UVA irradiation (4.5 J/cm²). Patch and photopatch tests with ketoprofen 0.5% and 5% pet., oxybenzone 1% and 10% pet., and tiaprofenic acid 5% pet. in 5 normal subjects showed no reactions, either 2 days after application or 1 day after irradiation.

Discussion

Arylpropionic acid derivatives (APADs), a group of non-steroidal anti-inflammatory drugs (NSAIDs), are used both systemically and topically. Ketoprofen and tiaprofenic acid, APADs, frequently cause allergic and photoallergic contact dermatitis. Benzophenone and its closely-related thiophene-phenylketone structures are held to be responsible for photocontact allergy to ketoprofen and tiaprofenic acid (Fig. 1), respectively (2). Cross-photosensitivity between ketoprofen and tiaprofenic acid has also been reported (3). Our case showed cross-photosensitivity between ketoprofen, tiaprofenic acid, and oxybenzone, implicating the benzophenone structure. Horn et al. (4) reported a patient with contact dermatitis from ketoprofen and photosensitivity to oxybenzone, whereas our case did not show contact sensitivity to ketoprofen or oxybenzone.

This case showed simultaneous positive photopatch tests to ketoprofen and oxybenzone. We postulate either photosensitization to each substance or photosensitization to one and cross-sensitization to the other. Ketoprofen and oxybenzone have benzophenone in common between their structures (Fig. 1), suggesting cross-sensitivity between them. Oxybenzone (benzophenone-3), a chemical UVB sunscreen, is widely used in food and drugs, e.g., poultices. Our case, therefore, calls for awareness of the simultaneous use of benzophenone-related APADs and oxybenzone in medicaments.

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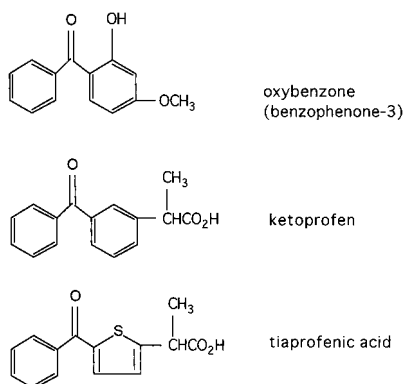


Fig. 1. Chemical structures of oxybenzone, ketoprofen and tiaprofenic acid.

Contact irritation provoking Hailey-Hailey disease

CHRISTINA M. RUDOLPH, BIRGER KRÄNKE, THEO D. TUREK AND WERNER ABERER

Department of Dermatology, Karl-Franzens-University Graz, Auenbruggerplatz 8, A-8036 Graz, Austria

Key words: Hailey-Hailey; familial benign chronic pemphigus; provoking factors; contact irritation. © Munksgaard, 2001.

Hailey-Hailey disease (familial benign chronic pemphigus, HH) is a rare autosomal dominant acantholytic disorder with chromosomal defect at 3q21–3q24. It is characterized by recurrent vesiculopustules, moist vegetative plaques, and patchy scaling, predominantly in intertriginous areas. Although the Hailey brothers, who first described the disease in 1939, originally excluded the pathogenetic influence of exogenous factors (1), many are now recognized as provoking factors, including irritants (2).

Case Report

A 64-year-old man, with no previous history of dermatitis, presented with crusted, erosive axillary erythema and swelling (Fig. 1). Over the past 6 weeks, he had repeatedly applied *Colognette Zitrusfrisch* refreshing tissues for malodorous slight erosions that had recurred there for many years. Mild erosions were also found in both groins. He was on no systemic medication. Recurrent episodes of 'inflammation of the axillary sweat glands' had occurred in his father. Histopathologic examination of axillary biopsies showed the characteristic

suprabasal clefting with acantholytic cells of HH. Complete healing was achieved with systemic antibiotic and bland topical therapy.

Patch testing with the standard, medicament and fragrance series of the DKG and with 5 commercially available refreshing tissues was performed when the patient was free of signs. Only the *Colognette Zitrusfrisch* refreshing tissue (ingredients: alcohol, aqua, fragrance, and menthol) showed peculiar erythema with purpura on D3, without crescendo phenomenon, interpreted as an unusual irritation reaction. Histopathologic examination of a biopsy of this area showed an inflammatory reaction with patchy extravasation of erythrocytes in the upper dermis.

Discussion

As early as 1962, Chorzelski (2) demonstrated that local irritants could induce typical HH lesions. It has since been postulated that HH patients have an increased frequency of contact sensitization (3), and recent authors have stressed contact allergens, rather than contact irritants, as exacerbating factors (4, 5). In our patient, however, contact irritation was what resulted in the disease being diagnosed for the 1st time.

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Fig. 1. Crusted, erosive erythema and swelling in the axillary region

Occupational contact allergy to tetrazepam

GENEVIEVE CHOQUET-KASTYLEVSKY, FRANÇOIS TESTUD, PHILIPPE CHALMET, SYLVIE LECUYER-KUDELA AND JACQUES DESCOTES

Poison Center and Pharmacovigilance Unit, Hôpital Edouard Herriot, 69437 Lyon Cedex 03, France

Key words: allergic contact dermatitis; tetrazepam; occupational; pharmaceutical manufacturing; benzodiazepines; medicaments; airborne; lack of cross-sensitivity. © Munksgaard, 2001.

Case Reports

Case no. 1

A 22-year-old woman had been working in a pharmaceutical factory since 1998, with no history of contact dermatitis. In October 1999, the factory began to manufacture tetrazepam. Although she handled tetrazepam powder with gloves and a face mask, she developed erythema of the face and neck, with itchy eyelid edema. She subsequently had flares each time that she handled the powder. The lesions healed during weekends and holidays.

6 months later, patch testing in Hayes test chambers with tetrazepam (Myolastan® tablets crushed in pet. and 10% in pet.) was positive (++) at D2, and negative to bromazepam (Lexomil® tablets crushed in pet.). Negative controls with Myolastan® tablets crushed in pet. have already been published (1, 2).

Case no. 2

A 42-year-old woman was head of the same pharmaceutical factory. She presented with erythema of the fingers and edema of the face after handling tetrazepam tablets. The skin lesions persisted for months. 5 months later, similar patch testing was positive (++) at D4 to tetrazepam and negative to bromazepam.

Only 2 workers were handling the powder and tablets in small-scale production facilities, with no general or exhaust ventilation. After these 2 cases of contact allergy, the factory suspended the manufacture of tetrazepam, and neither patient ever relapsed.

Discussion

Delayed-type hypersensitivity to benzodiazepines is rare and has only been proven by patch testing for tetrazepam (with 1 case of co-sensitization (1) and 1 case of cross-reaction (3) to diazepam), and clobazam (4). Sensitization following oral treatment with tetrazepam has been reported since 1990 (1, 2, 5), patch tests, when performed, being positive. Only 2 previous reports of contact dermatitis have been published, a nurse handling tetrazepam tablets without gloves (1) and a woman crushing tetrazepam tablets with her fingers (6). These are the 1st cases reported in pharmaceutical manufacture.

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Contact sensitization to chlorproethazine can induce persistent light reaction and cross-photoreactions to other phenothiazines

A. BARBAUD¹, E. COLLET², S. MARTIN¹, F. GRANEL¹, P. TRECHOT³, D. LAMBERT² AND J. L. SCHMUTZ¹

¹Dermatology Department, ³Pharmacovigilance Department, Fournier Hospital, 54000 Nancy; ²Dermatology Department, Bocage Hospital, 21000 Dijon, France

Key words: allergic contact dermatitis; chlorproethazine; phenothiazines; médicaments; cross-photosensitivity; patch testing technique. © Munksgaard, 2001.

In France, Neuriplege[®] ointment is a widely used myorelaxant containing chlorproethazine (10%), glycerol, magnesium stearate, lactic acid and paracymene. Its phenothiazine component can induce contact dermatitis, sometimes photoaggravated, or photoallergy.

Material and Methods

We collected data retrospectively from 10 patients (5 F, 5 M, average age 65.5 years, referred to either Nancy or Dijon) with contact allergy to Neuriplege[®] ointment. All had been patch tested with Neuriplege[®] as is, and chlorproethazine 10% pet., 1% pet. also in 9 cases and 0.1% pet. also in 6 cases. Additionally, photopatch tests were performed with chlorproethazine 1% and 0.1% pet. in 3 cases. Patch tests and photopatch tests were also performed, respectively, in 5 cases and in 4 cases with both chlorpromazine (0.1% pet) and promethazine (0.1% pet). The phenothiazine photopatch tests were performed on D1 with 5 J/cm²

Results

A severe contact dermatitis had been observed at the site of application of Neuriplege[®] ointment in all 10 cases. There was secondary spread to distant sites in 7/10 patients, with photodistribution in 4. During follow-up, relapse occurred in 2 patients who used Neuriplege[®] to treat a relative. 2 other patients developed a persistent light reaction, during the 6 months following contact photosensitization to Neuriplege[®], with a positive UVA phototest in both cases.

Patch tests were positive in all 10 patients to Neuriplege[®] as is and chlorproethazine 10% pet., causing a focal flare in 2. The excipients were negative in all patients. Chlorproethazine 1% pet. was positive in 8 of the 9 tested. Photoaggravation of photopatch tests with Neuriplege[®] as is and chlorproethazine 10% and 1% pet. occurred in 3/3 cases. Though patch tests with chlorpromazine (0.1% pet) and promethazine (0.1% in pet) were negative in 5/5 patients, photopatch tests were positive to chlorpromazine in 4/4 cases and promethazine in 3/4 cases.

Discussion

Chlorproethazine was responsible for drug-induced photosensitivity in 25/1239 cases (2%) reported to the French Agency for Drug Pharmacovigilance (1). This phenothiazine can induce contact dermatitis which is

generalized and photoaggravated. As previously reported in 2 cases (2, 3), we also observed persistent light reaction in 2 of our patients, which has also been reported with other phenothiazines such as dioxopromethazine (4). None of the 10 patients developed any immediate reaction after application of topical chlorproethazine, as was reported by Loesche et al. (5). Fenard-Naud (2) reported false-positive results in 52/52 of the negative control subjects when testing with Neuriplege[®] as is, and in 8/10 negative control patients tested with chlorproethazine 10% pet., but none such in 61 patients tested with Neuriplege[®] 10% pet. It is therefore advisable to patch test with Neuriplege[®] 10% pet. and chlorproethazine 1% pet. However, we observed 1 false-negative result among 9 patients tested with chlorproethazine 1% pet. Therefore, we consider it necessary to patch test first with chlorproethazine 1% pet., and then at 10% pet. if 1% is negative.

According to Fenard-Naud (2), photopatch tests with 5 J/cm² UVA should be done with Neuriplege[®] 1% pet. and chlorproethazine 0.1% pet., as she observed 2 false-positive results out of 9 negative control subjects when testing with chlorproethazine 1% pet. We saw no false-negative result when photopatch testing with chlorproethazines 0.1% pet. Photo-cross-sensitivity has been reported among phenothiazines, including chlorproethazine, and patients with (photo)contact sensitization to chlorproethazine should therefore be photopatch tested to other phenothiazines, such as chlorpromazine (0.1% pet.) and promethazine (0.1% pet.).

Phenothiazines are variously metabolized, 1 of the main metabolites of chlorproethazine being norchlorpromazine, which is also the most phototoxic metabolite of chlorpromazine (6). Photo-cross-sensitization observed in 5/5 of our patients between chlorpromazine and chlorproethazine might be explained by the formation of this common photosensitizing metabolite. Such cross-sensitivity contraindicates phenothiazines widely in patients with photocontact dermatitis from chlorproethazine.

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Occupational contact dermatitis due to the epoxy hardener m-xylylenediamine

S. SOMMER AND S. M. WILKINSON

Department of Dermatology, Leeds General Infirmary, Great George Street, Leeds LS1 3EX, UK

Key words: m-xylylenediamine; epoxy hardeners; industrial flooring; occupational; allergic contact dermatitis; material safety data sheets. © Munksgaard, 2001.

A 37-year-old man presented with a 6-month history of eczema on the forearm and hands. He fitted industrial flooring and had linked the start of his eczema with the introduction of a new tile adhesive from Germany 7 months earlier. Some colleagues had reported a similar problem. The patient had no personal or family history of atopy and was otherwise well.

He was patch tested to standard, acrylate adhesive, epoxy and plastics series, together with samples of his own rubber gloves, resin and hardener, with the following positive reactions at D2 and D4: thiuram mix ?+/+ and his own hardener 10% pet. ++/++. He was then tested to the individual compounds of the hardener and reacted ++/++ to m-xylylenediamine (MXDA) 0.1% pet. 16 controls were negative to MXDA. A latex prick test was negative. The data sheet warned of the risk of sensitization from skin contact, though it was available only in German.

Discussion

This case confirms that the patient's own hardener may have to be used for patch testing (1). It also illustrates the inadequacies of material safety data sheets in foreign languages. MXDA is used in the production of polyurethane, polyamide and epoxy products, especially epoxy-based colours. It is also used in certain textile finishes, rubbers, waxes and polishes.

The first 9 cases of occupational contact allergy to MXDA were observed between 1950 and 1962 in Germany (2). Since then, 3 further reports have been pub-

lished. Of 135 Dutch construction workers exposed to epoxy resins, 4 reacted to MXDA 0.1% pet., 2 of whom had eczema (3). A man coating floors with 2-component paint presented with airborne dermatitis, and reacted to both epoxy resin and MXDA (4). 4 Germans working on the production of polyurethane silk rapidly developed airborne dermatitis from MXDA. All also reacted to benzylamine, and 2 to ethylenediamine (5).

1 patient tested to MXDA 0.03% pet. failed to show a reaction, but reacted to a dilution of 0.1% pet. (4). Other reports, including ours, support 0.1% pet. as the best preparation for patch testing (3, 4).

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Allergic contact cheilitis from di-isostearyl malate in lipstick

JERE D. GUIN

18 Corporate Hill Drive, Little Rock, AR 72205, USA

Key words: allergic contact cheilitis; di-isostearyl malate; lipstick; cosmetics. © Munksgaard, 2001.

Lipstick-induced allergic contact cheilitis is uncommon (1). Prior to 1960, sensitivity to eosin in lipstick occurred, and, while this can still happen (2), other sensitizers have appeared, including gallates (3, 4), lanolin (1, 5), castor oil and related materials (6, 7), oils (8), waxes (9, 10), fragrances and flavors (11, 12), sunscreens (13), shellac and colophonium (5).

Case Report

An 18-year-old girl described bouts of swelling of the lips and adjacent skin. A lipstick that she had set aside was thought too old to use, so her mother purchased a new one for her. She described swelling of the lips and perioral skin within a few hours of applying the newly-purchased lipstick. The manufacturer agreed to supply ingredients in appropriate patch-test concentrations.

A 20-min application was first made and found negative. Patch testing then showed, at D2 and D4, a ++ reaction to the whole product, as well as to 1 ingredient, di-isostearyl malate 7.7% pet.

Comment

Swelling of the lips and face from a lipstick is unusual, but has been reported from quinazoline yellow (14) and from di-isostearyl malate and glyceryl di-isostearate together (15). The authors of the latter report also found sensitivity to glyceryl monoisostearate and tri-isostearate impurities in the (commercial grade) glyceryl di-isostearate, and to isostearyl alcohol in di-isostearyl malate, concluding that glyceryl monoisostearate and isostearyl alcohol were the principal sensitizers. Di-isostearyl alcohol is a C18 branched aliphatic alcohol and 2 of these are bound to a dicarboxylic acid in di-isostearyl malate.

Patch testing in the previous case as well as in this one points to allergic contact dermatitis rather than contact urticaria, despite the history of swelling. Di-isostearyl

malate is a fatty ester used as a conditioner in some other products, but in this one, for 'cushioning' and texture.

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