

CONTACT POINTS

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Systemic contact dermatitis due to fusidic acid

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Key words: drug allergy; fusidic acid; systemic contact dermatitis.

Fusidic acid, obtained from the fungus *Fusidium coccineum*, is a steroidal bacteriostatic antibiotic widely used in skin infections. It is preferentially used as a topical preparation. It may develop side-effects such as diarrhoea, nausea or vomiting, when administered by oral route. Several cases of local contact dermatitis by topical fusidic acid have been described previously (1–3).

Case Report

A 51-year-old man with no atopic history developed pruritic micropapular generalized exanthema, 4 hr after first oral dose of fusidic acid (250 mg). Symptoms and lesions resolved after 5 days of steroid treatment, with later skin desquamation on his neck and arms. 4 days before the oral antibiotherapy was started, the patient had been treated with topical Fucidine® ointment (fusidic acid 2% in lanolin and pet.) to an impetiginized skin lesion with good tolerance. It was the first time he was exposed to fusidic acid.

Allergologic Study

Patch test with Fucidine® (fusidic acid 2% in lanolin and pet.) was performed, being read at days 2 and 4. The

response was positive at day 4: papulovesicular lesions over erythematous skin (Fig. 1). Patch test with lanolin and pet. (both as is) was negative.

Discussion

Although widely used, hypersensitivity reactions to fusidic acid have been rarely described, usually associated with long exposures during treatments of chronic skin diseases such as atopic and stasis dermatitis (1–3). This is the first case reported of a systemic contact dermatitis by fusidic acid precipitated through the oral route. A previous short topical treatment with this antibiotic was the sensitizing event, without previous exposure to the drug. Not only long-term but also short-term treatments may be a risk factor for type IV sensitization to fusidic acid.

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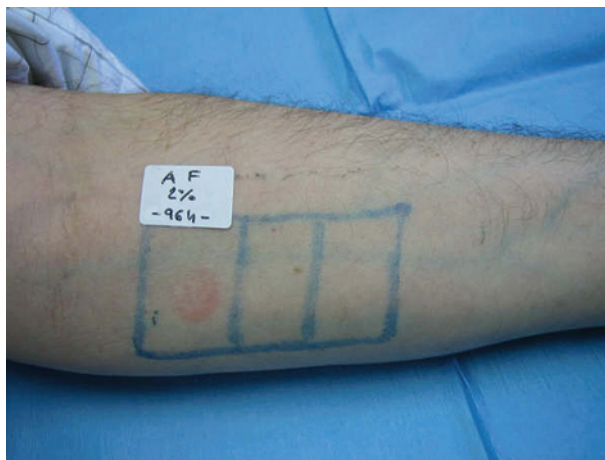


Fig. 1. Patch test positive response at 96 hrs

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Loss of eyelashes after use of a tinting mascara containing PPD

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Key words: eyelash; hair loss; mascara; *p*-phenylene diamine.

p-Phenylene diamine (PPD) is the most frequent sensitizer in hair dyes with increasing concentrations found

in the darker shades (1). A further well-documented source of exposure is from semi-permanent henna tattooing, a popular trend of recent years (2, 3). We report a case of sensitization and allergic reaction to PPD from use of tinting mascara.

Case Report

A 26-year-old office worker was initially referred for patch testing because of long-standing hand dermatitis. Positive reactions were found to PPD 1% pet. (D2+, D4+), disperse orange 3 1% pet. (D2+, D4+), nickel 5% pet. (D20, D4+) and hydroquinone 1% pet. (D2+, D4+). She had blonde hair that had never been dyed and had never used a skin lightening cream. On discussion of these results, she remembered temporarily losing all her eyelashes following the use of tinting mascara prior to a holiday, the previous year. There was no history of associated dermatitis or blepharitis. The mascara contained 2-chloro-*p*-phenylenediamine and carried a warning of allergic reaction and advice to perform a 48-h skin test prior to use.

Discussion

We conclude that the most likely explanation for eyelash loss in this case was concurrent sensitization and allergic reaction to PPD in the mascara, as the product was applied over a long period of time. Sosted et al. (4) have reported 2 cases of hair loss in the scalp due to PPD allergy associated with dermatitis lasting more than 3 weeks. In this consumer-based study, Sosted also demonstrated that many cases of PPD allergy go unreported, probably because the patient is able to make the connection between the reaction and the hair dye and then does not seek medical advice. It is also possible that less obvious exposures are simply missed as the cause of a reaction to this potent contact allergen, as in our case.

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Reduction in the frequency of sensitization to thiurams. A result of legislation?

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Key words: CEN 455-3; frequency; glove allergy; legislation; thiurams.

The number of patients sensitized to thiurams reflects the exposure to this group of chemicals. The major source of exposure originates from rubber gloves, especially single-use rubber gloves (1) but also from use in domestic and industrial settings.

The epidemic of sensitization to natural rubber latex (NRL) proteins from the mid 80s was followed by efforts in the NRL-glove industry, strongly supported by recommendations from European Committee for Standardization (CEN, TC 205, WG3) and The European Commission (2, 3), to reduce the content of proteins, mainly by leaching. At the same time, the manufacturers were encouraged to reduce the content and use of thiurams, the rubber accelerators, which are considered to be the most frequent contact sensitizers in rubber gloves.

According to information (Susanne Winkelmann, personal communication) from the industry as well as analyses of gloves (4), use of thiurams has now been considerably reduced in most quality, single-use NRL-gloves in the health care sector. Instead, carbamates, especially dibutyldithiocarbamate, are being used. This compound is considered to have a much lower sensitizing potential than others of the hitherto used sulphur accelerators (5). Cross-reactivity may however, still cause problems for patients already sensitized to thiurams or other carbamates (Lerbaek A, Knudsen B, Menné T, present issue).

Records from 1995 to 2004, from the patch test clinic at Gentofte Hospital, University of Copenhagen, Denmark, comprising a total of 8369 patients, tested with thiuram mix 1% in pet. (Hermal, Reinbek, Germany), showed a statistically significant decline in the frequency of sensitization to thiuram-mix (Fig. 1) ($P < 0.0001$; Armitage-Cochran's test for linear trend).

Discussion

We found a statistically significant decrease in contact allergy to thiuram over a 10-year period in our clinic. During this period, no major alterations were taking place in the area of referral concerning indication for referral to the clinic, occupational shifts in the population, population size, etc. The number of patients referred varied between 500 and 950 per year. The relative proportion of women was unchanged during the period, varying from 62 to 67%.

Focus on NRL-allergy in our clinic may have resulted in referral of selected cases, giving a high frequency of health care workers with both NRL- and thiuram-allergy, especially in the mid 90s. However, our findings are in accordance with the trend in other publications (6–8). The decline in frequencies of sensitization to sulphur accelerators from the thiuram group is coincident with a reduction in the content of NRL-allergens in single-use gloves used in the health care sector. The thiurams have been reduced due to substitution with (less sensitizing) carbamates and process control, but the reduction is probably also a spin-off from better leaching of the products in order to reduce the NRL-protein content.

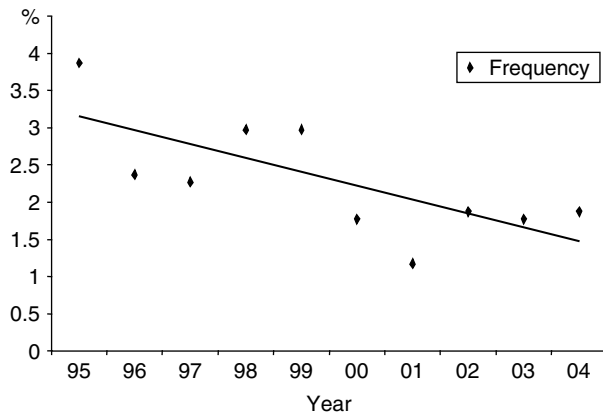


Fig. 1. Frequency of sensitization to thiuram-mix 1% in pet. at the patch test clinic, Gentofte Hospital, Denmark during the period 1995–2004. Armitage-Cochran's test for linear trend is significant with $P < 0.0001$.

The current standard, CEN455-3, does not contain a recommendation for quantitative measurements of residual chemicals (9), but it is satisfying that the industry has listened to the clinicians, resulting in gloves with a less sensitizing potential.

It is worth mentioning, however, that rubber additives still were the most frequent contact allergens reported in occupational contact dermatitis in 2001–2 (10).

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Chromate allergy from contact with leather furnishings

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Key words: allergic contact dermatitis; leather sofa; potassium dichromate.

Case Report 1

A 69-year-old woman presented with a sudden severe flare-up of previous mild eczema, affecting the hands, feet, back calves and posterior aspect of her arms. She promptly responded to in-patient treatment on 3 occasions with oral prednisolone, azathioprine and cyclosporin but deteriorated on discharge despite compliance.

On patch testing with the British Contact Dermatitis Society's (BCDS) standard and steroid series, a positive reaction was seen to potassium dichromate at D2 (+++) and D4 (+++).

Direct questioning elicited that 2 months prior to the exacerbation the patient had purchased a new leather sofa that was consistently used for more than 8 hr a day as a bed. She infrequently wore leather shoes. The skin cleared dramatically with no further exacerbations over the next 18 months, after she avoided all contact with leather.

Case Report 2

A 40-year-old woman with a 20-year history of pompholyx eczema presented with worsening hand eczema for 2 months associated with new patches affecting the lower back, extensor arms and legs and a limited response topical treatment.

She was patch tested with the BCDS standard and steroid series as well as her own medicaments. Positive patch tests were noted to potassium dichromate at D4 (++) (Fig. 1) and nickel at D2 (++) and D4 (+).

Collaborative history confirmed that she occasionally wore leather shoes and coat and had purchased a new leather sofa prior to the flare-up. Patch tests to samples from the leather sofa and the leather upper and inner from a pair of shoes demonstrated a positive reaction at D2 (++) and D4 (++) (Fig. 2). She has improved since avoiding nickel and leather.

Discussion

Cr (III), the commonest agent used in chrome tanning, has strong



Fig. 1.



Fig. 2.

tanning properties and binds to the collagen in the hide to form leather. Leather often contains trace amounts of hexavalent chromium [Cr (VI)], which is formed by oxidation of Cr (III) during the tanning process.

Legally, Cr (VI) should be a negligible part (3 mg kg^{-1}) of the total content of leather, because Cr (VI) has a greater ability to penetrate and accumulate in the skin and is the predominant cause of toxicity of chromium including contact dermatitis, skin and mucous membrane ulceration and asthma (1, 2).

However, a Danish survey reported that 35% of leather-products contained an excessive concentration of Cr (VI) ranging from 3.6 mg/kg to

14.7 mg/kg. This has been attributed to changes in the manufacturing process (3).

In analytic tests chromium is extracted from leather at an alkaline pH (7.5–8.0) which is similar to that of human perspiration. It is likely that this plays a role in chromium leaching out of leather during use thereby contributing to elicitation of chromate dermatitis (4).

While the foot eczema of case 1 was due to wearing leather shoes, the rash on the back and limbs was consistent with contact dermatitis to the leather covering of the sofa in cases 1 and 2. Contact with leather in furnishings has not previously been reported to be an exacerbating

factor in chromate dermatitis. This case series highlights the importance of considering environmental factors which negate the response to conventional therapies.

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Screening for gold allergy among dental clinic employees and patch test population

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Key words: dental restoration; gold; jewelry; nickel; professional contact.

Positive patch test results to gold sodium thiosulphate (GST) occur in 1–23% of tested subjects (1), i.e. with the prevalence comparable with the nickel allergy (2). High prevalence of positive test reactions to gold coupled with low clinical relevance has inspired many investigators to explain these peculiarities. However, less attention has been paid to the professional factors.

Table 1. Patch test results to gold sodium thiosulphate and nickel sulphate in dental staff and dermatological patients

Contact allergens	Dental staff (n = 80)	Patch test population (n = 80)
Gold sodium thiosulphate (2.0%)	17 (21.3%)	22 (27.5%)
Nickel sulphate (5.0%)	16 (20.0%)	14 (17.5%)

80 employees of dental clinics were patch tested using the dental screening series. The group for comparison consisted of 80 dermatological patients of the same sex and age distribution, undergoing routine patch testing. In addition to European standard series, they were tested with GST 2.0% pet. Testing was performed on the upper back using IQ chambers on a hypoallergic tape; results were read on days 3 and 5. All test products were brought from Chemotechnique Diagnostics (Malmö, Sweden). The participants' use of gold dental alloys and golden jewelry was analysed alongside with the presence of metal-related galvanic mouth symptoms. χ^2 test was used to examine the differences between the prevalence of gold allergy among dental clinic employees and patch test population.

At least 1 positive patch test result was obtained in 33 dental workers and 48 dermatological patients; the positive reactions to 2.0% GST were on the top of the list. Altogether, 39 study participants (24.4%) gave positive test reactions to 2.0% GST, and as summarized in Table 1, dermatological patients prevailed.

In total, the sensitization to 5% nickel sulphate was confirmed in 30 cases (18.8%). Concomitant nickel and gold allergy occurred in 5 patients and in 6 members of the dental staff. Statistical analysis showed that nickel allergy was not a predisposing factor for gold sensitization. 33 dental workers and 22 patients had, or had previously had, a dental item made of gold, whereas none of 160 subjects had oral lesions related to gold. Only 2 dental workers had suffered from eczema caused by golden jewelry. The presence of gold-containing dental alloys and sensitization to gold were significantly related ($P < 0.01$). Among dental staff members, gold allergy was significantly related to the galvanic mouth symptoms as well as wearing golden jewelry ($P < 0.001$, and $P < 0.05$, respectively). In dermatological patients, these correlations

were not relevant. Neither group showed correlation between nickel allergy and galvanic mouth symptoms.

Conclusions

Our study confirmed the basic conclusions drawn by others (2, 3). Firstly, dental gold is the main source of sensitization. Secondly, despite the extensive use of gold, allergic contact stomatitis and dermatitis are rare.

According to our data, professional factors do not contribute to the sensitization to gold. The most important source of this allergen is dental restoration-containing gold. No correlation between nickel and gold sensitivity was established.

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Photocontact allergy to oxybenzone and contact allergy to lignocaine and prilocaine

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Key words: Benzophenone 3; photoallergic contact dermatitis; amide anaesthetics.

Case Report

A 60-year-old female reported a 6-year history of red itchy swollen skin on her face, neck and chest after a few days of sun exposure abroad. It resolved with hydrocortisone cream 1%. She had an episode of cellulitis following ophthalmologic surgery in 2002, which resolved with oral antibiotics and anti-inflammatory drugs.

Examination was normal. Blood tests including lupus serology and plasma porphyrin scan were normal. Patch testing showed a +++ reaction to Balsam of Peru and Caine mix IV 10% in petrolatum (amylocaine, lignocaine and prilocaine mixture) at 48 hr and 120 hr. Patch testing to sunscreens was normal, but photopatch testing (Waldmann UVA 7.7 J/cm²) showed a ++ reaction to benzophenone-3. Patch and photopatch reactions were graded on a + to +++ scale according to the recommendations of the International Contact Dermatitis group (1). Photopatch testing was in line with the recently published consensus methodology (2).

Patch testing to lignocaine, prilocaine, mepivacaine, bupivacaine and procaine at 5% in petrolatum showed a +++ reaction to lignocaine at 48 hr and a +++ reaction to all amides (lignocaine, prilocaine, mepivacaine and bupivacaine) at 120 hr; procaine was negative (Fig. 1). Prick testing with preservative-free lignocaine 1%, prilocaine 1% and bupivacaine 0.5% was negative, but intradermal testing showed a negative immediate response, an erythematous papulovesicular response to lignocaine at 48 hr and macular erythematous responses to prilocaine and bupivacaine at 72 hr. She reports no further episodes since avoiding benzophenone-containing products. We have advised her that tetracaine and procaine are suitable for topical and intradermal anaesthesia.

Discussion

Sunscreen photoallergy remains uncommon. Benzophenone-3 (oxybenzone, 2-hydroxy-4-methoxybenzophenone) photoallergy is more frequent than allergy to hydrophilic benzophenones. History, photopatch testing and



Fig. 1. Intradermal testing to local anaesthetic agents at 48 hr. B = bupivacaine; L = lignocaine; P = prilocaine.

resolution of the rash on avoidance of benzophenones confirm photoallergy to benzophenones in this patient.

Contact allergy to amide anaesthetics was discovered with patch testing to caine mix IV and confirmed with positive individual amide patch tests and intradermal testing. Her history of postoperative cellulitis probably represents type IV hypersensitivity to lignocaine, and cross-reactivity to other amides precludes their use (3, 4).

Lignocaine hypersensitivity is uncommon, and few published series estimate the prevalence. Mackley et al. (3) found 1% incidence of Type IV allergy to lignocaine in 183 patients. Ruzicka et al. (5) found no reactions in patients tested for local anaesthetic allergy, while Weightman et al. (6) describe 29 cases over a 21-year period. The North American Contact Dermatitis Group (NACDG) survey of 1030 patients demonstrated a 0.7% incidence in a general patch test population (3). The apparent variation may reflect international difference in use of anaesthetics in OTC products.

Investigation of reactions to local anaesthetics should include patch testing and should be confirmed using intradermal testing for reactions to injectable anaesthetics or to topical anaesthetics where there is diagnostic doubt. This is due to the higher specificity of intraderm testing and the practical implications of local anaesthetic allergy (7, 8).

Conclusion

We report a patient presenting with concurrent photoallergic contact dermatitis to oxybenzone and type IV

hypersensitivity to amide anaesthetics. Awareness of contact and photocontact allergy to these allergens is important because of the increasing use of products containing local anaesthetics. Testing for allergy to an injected local anaesthetic should involve a prick test and patch testing and if negative an intradermal test to include both early and late readings.

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Generalized dermatitis with eosinophilia resulting from allergic contact dermatitis due to povidone iodine

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Key words: povidone iodine; allergic contact dermatitis; generalized dermatitis; eosinophilia.

Case Report

A 58-year-old Japanese man had been treated with a 10% povidone iodine solution for secondary infection of an ulcer on the right lower leg. After about 1 month, the size of the ulcer increased, and a pruritic eruption developed around the ulcer, but application of 10% povidone iodine solution was continued. As the pruritic eruption suddenly spread throughout most of the patient's body, he presented to our hospital. Physical examination revealed scattered erythematous papules and erythema with scales on the neck, trunk and bilateral arms (Fig. 1), and erythema with serous papules around the ulcer on the right lower leg (Fig. 2). Laboratory test indicated leucocytosis (10 000/ μ l) and hypereosinophilia (2100/ μ l). We advised the patient to discontinue 10% povidone iodide solution since we suspected severe contact



Fig. 1. Scattered erythematous papules and erythema with scales on the back.



Fig. 2. Erythema with serious papules around the ulcer on the right lower leg.

dermatitis caused by the solution. We started treatment with systemic predonisolone and a topical mometa-sone furoate. The eruption diminished and the ulcer epithelized after about 2 weeks. The patient was patch tested and showed positive reaction to the dried 10% povidone iodine solution at D2, D3 and D7. We then performed an ingredient patch test with povidone iodine at 2% aq. and polyoxyethelene nonylphenyl ether at 0.2% aq. The patient showed positive reaction only to povidone iodine 2% aq. at D2, D3 and D7.

Discussion

Povidone iodine is commonly used as antibacterial agent and antiseptic, and has gained widespread acceptance because of its low toxicity and high germicidal efficacy. As irritant contact dermatitis caused by prolonged contact with a large quantity of 10% povidone iodine solution had been reported (1), it was necessary to distinguish allergic contact dermatitis from irritant contact dermatitis. In order to distinguish them, Kozuka (2) recommends to perform the patch test with dried 10% povidone iodine solution because the patch test with 10% povidone iodine solution causes irritation. In addition, Nishioka (3) reported that patients showing positive reaction to povidone iodine 2% aq. on days 3–5 were considered allergic. On the basis of these reports, we diagnosed our patient as allergic contact dermatitis due to povidone iodine because he showed a positive reaction to dried 10% povidone iodine solution and to povidone iodine 2% aq. on 3–7 days.

We considered that prolonged application of povidone iodine solution caused generalized dermatitis in our patient. There have been several cases of severe or generalized contact dermatitis due to povidone iodine (4–6). However, in these reports, they performed patch testing by neither dried povidone iodine solution nor povidone iodine 2% aq., and they did not describe about eosinophil. To our knowledge, this is the first definitive case of generalized dermatitis with eosinophilia on the basis of allergic contact dermatitis due to povidone iodine. We speculate that eosinophilia may have

participated in the formation of generalized dermatitis in our patient.

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The simultaneous occurrence of contact urticaria from sulbactam and allergic contact dermatitis from ampicillin in a nurse

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Key words: allergic contact dermatitis; ampicillin; contact urticaria; hand dermatitis; occupational; sulbactam.

Case Report

A 24-year-old nurse presented with pruritic erythematous papulovesicles and scaly plaques that affected her fingers (Fig. 1). After 3 months of work in a paediatric section, she experienced pruritus and redness on her fingers that occurred within 30 min of preparing antibiotic solutions. She also developed pruritic eczema on one finger that extended to all fingers in a few months. She had a past history of allergic rhinitis, but not of atopic dermatitis.

We performed scratch tests and patch tests with several of the antibiotic injection solutions that were most frequently used in her paediatric section, such as Sulbacillin[®] (ampicillin + sulbactam), Augmentin[®] (amoxicillin + clavulanate) and the other β -lactam antibiotic injections, including cefazedone, cephadrine, cefuroxime, cefotaxime and ceftriaxone. Small quantities of the

test solutions were placed on her upper back, and linear scratches about 1 cm long, but not sufficient to draw blood, were made through the epidermis (scratch tests) (1). Reactions were considered positive when a wheal of test antibiotic solution was larger than that of histamine control in 30 min, and a test with Sulbacillin[®] (100 mg/ml aq.) produced erythema and wheals. The patch tests showed strong positive reactions to both Sulbacillin[®] (100 mg/ml aq.) and Augmentin[®] (100 mg/ml aq.) at D2. Next, we performed scratch tests and patch tests with various concentrations (100 mg/ml aq., 10 mg/ml aq. and 1 mg/ml aq.) of ampicillin and sulbactam, which are the only 2 components in Sulbacillin[®]. She developed an immediate reaction to pure sulbactam, but not to ampicillin, and she also had a delayed vesicular eczematous reaction to pure ampicillin at D2 and D3 (Table 1). We also performed scratch tests using sulbactam, histamine and water to 10 healthy medical students and nurses as a control test to avoid false interpretation of results, and they showed no immediate response to sulbactam. On her Korean standard patch tests, cobalt chloride and thimerosal were weakly



Fig. 1. Pruritic erythematous papulovesicles and scaly plaques on the left little finger.

Table 1. Scratch tests and patch tests results

	Concentrations	Scratch test (30 min)	Patch test	
			D2	D3
Augmentin [®] (amoxicillin + clavulanate)	100 mg/ml aq.	–	++	+
Sulbacillin [®] (ampicillin + sulbactam)	100 mg/ml aq.	+ ^a	++	+
Ampicillin	100 mg/ml aq.	–	++	+
	10 mg/ml aq.	–	++	+
	1 mg/ml aq.	–	+	+
Sulbactam	100 mg/ml aq.	+ ^b	–	–
	10 mg/ml aq.	+ ^c	–	–
	1 mg/ml aq.	–	–	–
Positive control (histamine)	1 mg/ml	+ ^d	–	–
Negative control (water)		–	–	–

a = wheal 1.4 cm, erythema 2.5 cm; b = wheal 3.5 cm, erythema 6.0 cm; c = wheal 0.8 cm, erythema 2.4 cm; d = wheal 0.6 cm, erythema 2.0 cm.

positive at D2. In addition, the *in vitro* assays for antigen-specific IgE to penicilloyl G, penicilloyl V, ampicilloyl and amoxicilloyl were performed by using RAST, and the results were all negative.

On the basis of the nurse's clinical history and the scratch and patch testing, a diagnosis was made of contact urticaria from sulbactam and allergic contact dermatitis from ampicillin. She showed clinical improvement with topical steroid treatment, and we recommended her to move to another hospital section where she would not have to deal with aminopenicillins. Since avoiding aminopenicillins, the symptoms disappeared, with no relapse on follow up.

Discussion

Aminopenicillins have been associated with various types of allergic reactions, including both Type I and Type IV hypersensitivity. The main adverse reactions are urticaria, angioedema, anaphylaxis and maculopapular rashes (2). However, allergic contact dermatitis from aminopenicillins is a rare condition, and there have been just a few reports on this in the literatures (3–5).

One of the main mechanisms of this case seemed to be as a Type IV reaction to aminopenicillins, since the patch test was positive to both Sulbacillin[®] and Augmentin[®], and it was positive only to ampicillin and not to sulbactam in the patch test with using the individual components of Sulbacillin[®]. We also performed a patch test with other β -lactam antibiotics, but no delayed type reaction was observed. According to the previous reports (2, 6, 7), both the β -

lactam core structure and the side chains are recognized by T cells in delayed allergic reaction to aminopenicillins. This highly selective response prevents them from cross-reacting with cephalosporins and even penicillin G or V. Ampicillin (the constituent of Sulbacillin[®]) and amoxycillin (the constituent of Augmentin[®]) can cross-react, because these 2 penicillins carry the same side chain, the only difference being a hydroxy group at the aromatic ring of the side chain for amoxycillin (8).

We performed scratch tests instead of prick tests to evaluate the contact urticarial symptoms. The scratch test is a less standardized method than the prick test, but it is a useful diagnostic method for non-standardized allergens in contact urticaria (9). It is interesting that the other mechanism of this case was Type I reaction to sulbactam. Sulbactam has a β -lactam structure resembling that of the penicillin nucleus, and this widens the antibacterial activity of ampicillin and the other β -lactam antibiotics against the β -lactamase-producing micro-organisms. Although there have been a few reports of hypersensitivity reactions to clavulanic acid (another β -lactamase inhibitor) (10, 11), to our knowledge, no immediate or delayed allergic reaction to sulbactam has been described before.

We could not completely exclude the possibility that the preservatives contained in antibiotic injections might have been responsible for the allergic reaction to Sulbacillin[®] and Augmentin[®] in our patient because thimerosal showed a weak positive reaction on the standard patch test.

However, as our patient did not reveal any response to the other β -lactam antibiotic injections and showed positive response to pure sulbactam and pure ampicillin, the preservatives might not have been the causal substance of the hypersensitivity reaction.

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Idiopathic post-traumatic eczema

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Dyshidrotic eczema of the hands and/or feet is associated with atopy, contact allergies, mycotic infections and stress (1). Mechanical trauma may result in skin abrasion and laceration but may also give rise to urticarial, psoriatic or lichenoid skin lesions.

An eczematous (dyshidrotic) eruption caused by invasive mechanical trauma, however, is only rarely reported (2–4).

Case Report

A 30-year-old male consulted us at 2 occasions with a circumscribed dyshidrotic eruption at a site of sharp instrumental trauma. He experienced the same reaction 5 times before, each time at sites of small skin

defects after trauma. The dyshidrotic lesions quickly healed after the application of a topical corticosteroid. He denied the use of bandages. He had a history of hay fever but not of atopic dermatitis. He works in the information technology and likes gardening and collecting model trains.

He was patch tested with the European standard series and additional series including metals, plastic ingredients and glues. He had positive reactions to balsam of Peru, colophony and abitol. Prick tests were positive to grasses, house dust mite and several animals.

After the first visit, he consulted us again with multiple confluating vesicles at a site of trauma on the fourth finger of his right hand and a second time after a sharp trauma at the back of his hand (Fig. 1). No other skin lesions were present, in particular, no signs of a fungal infection on the feet or between the toes were visible. Histopathology showed a spongiotic dermatitis, and a microscopic investigation and a culture on fungi did not show fungi.

Discussion

Reports on mechanical trauma as cause of dyshidrotic or eczematous eruptions are rare (2–4). Mathias designated dermatitis at sites of previous trauma as post-traumatic eczema (1). 3 different types of post-traumatic eczematous reactions can be discerned. The eruption may be the first manifestation of an endogenous dermatitis, or it may follow the onset of the dermatitis. It may also occur as an isolated

phenomenon, which may be classified as idiopathic post-traumatic eczema.

Our patient does not show any signs of dermatitis, except after invasive trauma. Other explanations for his dermatitis like mycotic or bacterial infections could be excluded. A contact allergic reaction to a bandage (positive patch test reactions to colophony and abitol) may be a possibility, however, he vigorously denied the use of bandages. Another possibility is contact with glues or plants. However, he did not remember skin contact with these materials. Because of the clear history and the localized nature, limited to the site of trauma, of the lesion, we diagnosed his condition as an idiopathic post-traumatic eczema.

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Fig. 1. Dyshidrotic eruption at a site of invasive mechanical trauma.