

CONTACT POINTS

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Type IV hypersensitivity to sodium metabisulfite in local anaesthetic

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Key words: adrenaline; lidocaine; local anaesthetic; sodium metabisulfite; Type IV hypersensitivity.

Case Report

A patient received an anaesthetic injection for a biopsy of a suspected basal cell carcinoma on the left forearm. The local anaesthetic consisted of, among other constituents, lidocaine 2% with adrenaline (1:100 000). According to the patient, a burning sensation developed around the site of the injection after 7 hr, itch after 24 hr and swelling and erythema after 48 hr. The form of the erythema was round and not that of the plaster that had been there. The symptoms and signs had disappeared in about a week.

Patch tests were performed with the European standard series, lidocaine 2%/adrenaline from an ampoule, lidocaine 2% from an ampoule and all the ingredients in an ampoule of lidocaine 2%/adrenaline (lidocaine 2% in pet., adrenaline 2% pet., sodium metabisulfite 2% pet. and ethylenediaminetetraacetic acid 1% pet.). Positive reactions to nickel (++), lidocaine 2%/adrenaline from the ampoule (+) and sodium metabisulfite (++) were observed on day 3. Patch tests with sodium metabisulfite in 3 healthy volunteers were negative.

Prick tests were performed with sodium metabisulfite (0.05%, 0.1%, 1% and 10% in water). All these were negative.

Provocation test (subcutaneous injection) was performed with

lidocaine 2%/adrenaline from an ampoule. The patient experienced a burning sensation, erythema and swelling at site of the injection after 48 hr. The symptoms lasted for a week. Provocation test with lidocaine 2% from an ampoule was negative.

Discussion

Sodium metabisulfite (synonym: sodium pyrosulfite) is a frequently encountered sulfite present as an antioxidant in oral, topical and parenteral medicines. It is used as an additive to inhibit oxidation of an active component (in this case the adrenaline in the lidocaine solution). Sodium metabisulfite is also used as a food additive (E223) in the food industry (1). It is further used as a fixative in the photographic industry.

Both early and late reactions to sulfites have been reported. Oral or nasal administration of sulfites may cause various reactions such as bronchospasm (in asthmatic patients), rhino-conjunctivitis, urticaria and even anaphylactic shock. There is no unequivocal explanation of the pathogenetic mechanism of hypersensitivity to sulfite. Clear proof of an immunological (immunoglobulin-E mediated) cause is still lacking (1).

A relevant contact allergy to sodium metabisulfite is not often encountered. There are about 20 case reports to date. These involve individuals who are employed in the pharmaceutical industry, the food industry or the photographic industry. There are also several case reports of patients who were allergic to sodium metabisulfite in creams, among others, in hydrocortisone and ketoconazole creams (2). However, in an investigation by Vena et al. (3), contact allergy to sodium metabisulfite appeared not to be so rare. They conducted 2894 patch tests with sodium metabisulfite (1% pet) in patients with eczema. A positive reaction was observed in 50 (1.7%) patients. The positive reaction to sodium metabisulfite was considered to be relevant in 12

(7 occupational and 5 medicament) of these 50 patients.

In this case report, there was a Type IV hypersensitivity reaction to a local anaesthetic, which could be attributed to the sodium metabisulfite additive. Such a reaction to sodium metabisulfite additive in a local anaesthetic has been reported previously, but the reaction (swelling) there occurred as early as 5 hr after the injection (4).

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Allergic contact dermatitis from dyes in wigs following diphenylprone treatment

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Key words: allergic contact dermatitis; azo dyes; diphenylprone; para-phenylenediamine; wigs.

Case Report

A 36-year-old woman developed extensive alopecia areata in 1992 and quickly progressed to alopecia universalis. In July 1993, sensitization was attempted with 2% diphenylprone (DPCP). This was increased to 2.5% due to lack of response and she successfully sensitized to it. Treatment was started at low concentrations and she developed severe scalp eczema, which also extended onto her arms and legs, requiring topical corticosteroids for control. By January 1994, she had generalized eczema but no hair regrowth so DPCP was discontinued. By June 1994, she still had eczema of the scalp but in a band-like pattern suggestive of contact dermatitis from her wigs. The dyes or elastic in the wigs were suggested as likely culprits and patch testing was performed. This was negative for all rubber chemicals but she reacted strongly in the standard series to para-phenylenediamine (PPD). A dye series was then applied, with the following results at Day 4 (D4) for all (Table 1).

She initially tried barrier methods, e.g. Tubegauze between the wig and scalp, along with 1% hydrocortisone, but eventually had to switch from acrylic wigs to human-hair wigs without dyes and avoid any clothes containing anything other than natural fibres and bleached totally of any dyes. By July 1998, she was much improved but still getting some scalp dermatitis and she was patch tested again. She was negative to all rubber chemicals tested but positive (++) to the dyed parts of her wig. With meticulous avoidance of dyed wigs and dyed clothing, she achieved good resolution of her eczema and was discharged from follow-up in September 1998. She was not seen again until April 2004, when she still had a strip of eczema around the back of the scalp. Patch tests were strongly positive to PPD but again negative to rubber chemicals. Part of her human-hair wigs still contained dyes, causing the strip of eczema. She continues to

be free of her eczema on the body and limbs by avoiding dyed clothing.

Discussion

Topical DPCP has been used in the treatment of alopecia areata since 1983 (1). Side-effects are frequent but generally tolerable, with a mild eczematous reaction being desirable to achieve hair growth. Undesired side-effects include erythema multiforme (2) and urticaria (3, 4). Fernandez-Redondo et al. (5) describe a case of systemic contact dermatitis simulating a fixed drug reaction in a nurse who had been treated with DPCP, and a case of consort contact dermatitis in which a woman developed acute facial eczema whenever her husband was treated. However, in most of the literature no serious long-term side-effects have been reported. Cross-sensitizations between dyes are also well documented (6), but we have not found documentation of sensitization to dyes following contact with DPCP, a chemical not related to azo dyes.

Our patient developed a severe generalized allergic contact dermatitis from DPCP and later to a variety of commonly used dyes. Our explanation is that constant exudative eczema in the scalp encouraged contact sensitization to dyes in her wigs. Having become sensitized to the dyes, she then suffered generalized eczema from clothing dyes.

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Delayed-type hypersensitivity to the ultra-low-molecular-weight heparin fondaparinux

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Key words: anticoagulation; cutaneous adverse drug reaction; delayed-type hypersensitivity; fondaparinux; heparin; pentasaccharide; therapeutic alternatives.

Delayed-type hypersensitivity reactions at injection sites of heparins in the form of eczema-like infiltrated plaques, with occasional generalized reactions, are common side-effects. Cross-reactivity between high- and low-molecular-weight heparins is well known. Recently, the new pentasaccharide fondaparinux has been recommended as a safe alternative.

Case Report

An 85-year-old woman developed localized infiltrated plaques, followed by a disseminated exanthematous reaction, 10 days after starting the administration of dalteparin sodium (Fig. 1). Intradermal tests with a comprehensive series (Table 1) of

Table 1. Dye series

Disperse Red 1	1% pet.	++
Disperse Yellow 3	1% pet.	++
Aminophenol	1% pet.	+
Disperse Blue 3	1% pet.	–
Disperse Red 17	1% pet.	+++
Disperse Orange 3	1% pet.	+++



Fig. 1. Confluent infiltrated plaques from dalteparin.



Fig. 2. Erythematous superficial plaque (day 4) at the injection site of 2.5 mg subcutaneous fondaparinux.

undiluted commercial drugs yielded positive reactions at day 7 to all heparins and the heparinoid danaparoid, whereas fondaparinux and lepirudin were negative. Subcutaneous provocation with lepirudin was tolerated, but there was a positive reaction to fondaparinux at each site of 3 therapeutic subcutaneous injections (Fig. 2). Later, a second intradermal test with fondaparinux

was positive. Therefore, we interpreted the first intradermal test as false negative, as upon subcutaneous re-exposure, the plaque reaction recurred within 2 days. Skin biopsy of a plaque revealed spongiosis of the epidermis and oedema, with a perivascular mononuclear cell infiltrate in the upper dermis. Immunohistology revealed a predominance of CD4⁺ T-helper cells and Langerhans' cells within the infiltrate, suggesting delayed-type hypersensitivity.

cells within the infiltrate, suggesting delayed-type hypersensitivity.

Discussion

Fondaparinux is an ultra-low-molecular-weight heparin with a molecular weight of 1728 kDa, and its structure is an exact copy of a heparin pentasaccharide sequence (1, 2). It is a new chemically synthesized selective inhibitor of activated factor X and is effective in the prevention and treatment of deep vein thrombosis (1). Despite the structural similarities, cross-reactions with allergic reactions to heparins have not so far been observed (2). 11 cases have been reported in the last 2 years of delayed-type allergy to heparins and tolerance of fondaparinux (2–7). All patients were females with a mean age of 59.5 years (2–7). They revealed delayed-type hypersensitivity to low-molecular-weight heparins (11 patients), high-molecular-weight heparins (6 patients) and the heparinoid danaparoid (5 patients) (2–7).

So far, the antigenic determinants of heparins have not been identified. Currently, it remains unclear whether the reaction to fondaparinux represents a cross-reactivity to a part of the heparin molecule or whether it is a *de novo* sensitization to another epitope. It could be hypothesized that in this case the fondaparinux–factor X complex probably represents the allergen. In addition, the fact that most affected patients are females and the rare male patients often obese (8) still needs to be elucidated. Hormonal factors, longer persistence of heparins in subcutaneous fatty tissue (9) or a relationship to the lipase activity of heparins could explain this striking difference (10). In summary, in patients with delayed-type hypersensitivity to high- and low-molecular-weight heparins and danaparoid, fondaparinux cannot

Table 1. Results of prick, intracutaneous and subcutaneous tests

Substance	Prick tests (20 min)		Intradermal tests		Subcutaneous tests (therapeutic dose)
	As is	1 : 10	As is (20 min)	As is (day 7)	
Liquemin (heparin-Na)	–	Irritant reaction	Irritant reaction	+	Not done
Fragmin (dalteparin-Na)	–	–	Irritant reaction	+++	Not done
Fraxiparin (nadroparin-Ca)	–	–	Irritant reaction	++	Not done
Clexane (enoxaparin-Na)	–	–	–	++	–
Orgaran (danaparoid)	–	–	Irritant reaction	+	+ (>48 hr)
Refludan (lepirudin)	–	–	–	–	
Arixtra (fondaparinux)	–	–	–	First test: – Second test*: +	

*Second intracutaneous test with fondaparinux was positive at days 3 and 7.

reliably be recommended as a therapeutic alternative.

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Occupational airborne allergic contact dermatitis from azithromycin

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Key words: airborne; allergic contact dermatitis; azithromycin; occupational

Almost all airborne contact allergens are occupationally related (1). Industrial and pharmaceutical chemicals, including antibiotics, constitute a large part of that group. Antibiotics reported to cause airborne allergic contact dermatitis include penicillins, cephalosporins, aminoglycosides, benzoyl peroxide and azalides (1, 2).

Case Report

2 men were employed in the blending department of a pharmaceutical company. They were required to add water to the powdered form of various chemicals and then bake them. Each blend required 2-3 days. Drugs involved included Norvasc, Viagra, Zolof, lithium, Reactine, Cardura and Zithromax (azithromycin). Fillers such as cornstarch, lactose and magnesium stearate were used at all stations. A protective Tyvek suit and a filtered respirator were part of the safety equipment.

The 1st patient was 40 years old and had worked in the blending department for 6 years. He was first seen in October 2001, when he reported an itchy, vesicular eruption on the head and neck that later spread to the rest of the body. He was treated with a potent topical corticosteroid and an oral antihistamine.

This patient was tested to the North American Contact Dermatitis Group (NACDG) standard series as well as crushed Zithromax tablets and pure azithromycin, both diluted to 5% in pet. Readings were performed at D2 and D5. He had a + reaction at both the Zithromax and azithromycin sites at D2, and a +++ reaction at both sites by D5.

The 2nd man was 44 years old. He again had been employed in the blending department for 6 years. He presented in June 2002 with a history of an intensely pruritic acute eczema-

tous eruption on the left cheek that later spread to the rest of the face and neck, after 8 blends of Zithromax.

We proceeded with patch testing to the NACDG standard series as well as to Zithromax and azithromycin, both diluted to 5 and 10% in pet.

A + reaction was seen at both sites and concentrations at D5.

Both these patients were removed from the Zithromax room and reported clearance with no recurrence.

Comment

To our knowledge, there is one previous report of airborne allergic contact dermatitis from azithromycin in a pharmaceutical worker confirmed by patch testing and workplace challenge (3). We believe, in conjunction with the occupational health department at the company, that these employees might not have followed safety precautions when entering or exiting the blending rooms, as well as there having been an inadequate ventilation system.

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Metal allergy to implanted orthopaedic prosthesis after postoperative *Staphylococcus aureus* infection

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Key words: implant; inflammatory arthritis; metal allergy; orthopaedic prosthesis.

Case Report

An 80-year-old Japanese man underwent surgery on the left knee, during which an orthopaedic prosthesis made of an alloy of cobalt and chrome was implanted (Table 1). 3 months later, an oedematous erythema developed over the left knee, accompanied by severe local pain and systemic fever. Blood examination showed C-reactive protein (CRP) at 19.8 mg/dl (normal,

0–0.4 mg/dl) and a white blood cell count of 17 000 (normal, 3 000–9 000 μ /l). Colonies of *Staphylococcus aureus* were cultured from the aspirated fluid inside the knee. Although intravenous antibiotics were given regularly and the inside of the knee joint was rinsed repeatedly in an attempt to control the infection, the suppurative inflammation in the left knee persisted for 4 months. A local exudative erythema was also present on the skin. Cultures from the aspirated fluid were all positive for *S. aureus*. Elevated CRP persisted for 124 days. The patient underwent a 2nd operation to replace the cobalt and chrome alloy prosthesis with a temporary and metal-free prosthesis containing antibiotics. The recalcitrant inflammation and dermatitis over the left knee quickly subsided soon after the operation. We then considered the possibility that the patient had an allergy to metal.

A 2-day closed patch test with metal allergens from the Japanese Society for Contact Dermatitis standard series (numbers 1–3 in Table 2) and from Torii Pharmaceutical Co. Ltd. (Tokyo, Japan, numbers 4–16 in Table 2) showed positive reactions to Co, Ni, Cr, Mn, Pt, Ir, In, Hg, Sn and Zn (Table 2). Among these, Co, Ni, Cr and Mn were major constituents of the original orthopaedic implant (Table 1).

The patient underwent a 3rd operation to replace the temporal orthopaedic prosthesis with a permanent prosthesis, one made of an alloy

of titanium and ceramic. The patient showed no recurrence of either pruritic erythema or suppurative arthritis.

Discussion

Pruritic erythema and suppurative arthritis were persistent in this case, even though the patient was repeatedly treated with intravenous antibiotics and rinsing inside the knee joint. Because Co, Ni, Cr and Mn were major constituents of the orthopaedic implant, we diagnosed the patient as having allergic contact dermatitis and inflammatory arthritis due to the metals in the prosthesis, complicated by bacterial infection. The patient also showed positive reactions to Pt, Ir, In, Hg, Sn and Zn, which were not present in the metallic joint. The patient, however, wore dental metal that contained Pt, Ir, In, Sn and Hg. It is most likely that these positive reactions indicate prior sensitization. The reaction to Zn may just have been an irritant reaction.

Hierholzer et al. (1) pointed out that the bacterial infection adjacent to prosthesis is not only associated with higher tissue concentrations of metal but also with a local accumulation of immunocompetent cells. In inflamed tissues, reactive oxygen species are produced by phagocytes and oxidize Ni (II), from nickel in the oxidation state +2 to the higher oxidation states Ni (III) and Ni (IV) (2). Artik et al. (2) clearly demonstrated in mice that Ni of higher oxidation states, namely Ni (III) and Ni (IV), has significant ability to sensitize naïve T cells, although Ni (II) does not. Similar phenomena may well occur with other metals. It is highly conceivable that the Staphylococcal infection facilitated the elicitation of the metal allergy and that the metal allergy worsened the inflammation inside the joint as well as on the surface of the skin. This vicious circle might have lasted until the sensitizing implant was removed.

It is generally thought that the modern plastic-to-stainless-steel hip replacements do not cause cutaneous reactions (3). However, eczema can happen with the old type of static metal prostheses (3). The reason why modern prostheses are safe is that the nickel contained in stainless steel is so firmly bonded in the alloy that stainless steel is harmless even for most nickel-sensitive people.

Why did our patient show dermatitis and arthritis from the implant?

Table 1. Constituents of the metallic implant

Constituent	%
Cr	27.0–30.0
Mo	5.0–7.0
Ni	up to 1.0
Fe	up to 0.75
C	0.21–0.35
Si	up to 1.0
Mn	up to 1.0
Co	the rest

Table 2. Results of patch testing

Allergens	Concentration	D2	D3	D4
Cobalt dichloride	1% petrolatum	–	+	+
Nickel sulfate	2.5% petrolatum	+	+	+
Potassium dichromate	0.5% petrolatum	–	+	+
Chloroplatinic acid	0.5% aqueous	–	+	+
Gold trichloride acid	0.2% aqueous	–	–	–
Palladium chloride	1% aqueous	–	–	–
Iridium tetrachloride	1% aqueous	–	+	+
Indium trichloride	1% aqueous	–	+	+
Mercuric chloride	0.05% aqueous	–	+	+
Stannous chloride	1% aqueous	+	+	+
Aluminum chloride	2% aqueous	–	–	–
Ferric chloride	2% aqueous	–	–	–
Copper sulfate	1% aqueous	–	–	–
Manganese chloride	2% petrolatum	+	+	+
Zinc chloride	2% petrolatum	+	+	+
Silver bromide	2% petrolatum	–	–	–
Water	as is	–	–	–
Petrolatum	as is	–	–	–

Patch tests were applied using Finn Chambers[®] on Scanpor tape[®] (Epitest, Tuusula, Finland) for 48 h. Results were read at day 2, 3 and 4 according to the recommendations of the International Contact Dermatitis Research Group (4).

The patient received an implant of the older type, an alloy of cobalt and nickel, which would more easily release ionized metallic species. If the patient had already been sensitized to such metals, allergic reaction might have occurred first and then concomitant bacterial infection complicated the inflammation. If the patient had not been sensitized to the metals before implantation, the postoperative infection could have triggered and accelerated sensitization and allergic reaction. Allergic reaction combined with bacterial inflammation probably played an essential role in prosthesis failure in this patient. Our case suggests that patch testing is indispensable for patients with persistent inflammation after the implantation of a prosthesis made of an alloy of cobalt and chrome.

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Key words: cutaneous adverse drug reactions; hirudin; Type I and Type IV allergy; unfractionated heparin.

Heparin has now largely been replaced by low-molecular-weight heparin (LMWH) as the treatment of choice for thromboembolic disorders. LMWH is derived from fractionated heparin and has better absorption and potentially fewer haemorrhagic complications (1). As LMWH is derived from heparin, similar immune-mediated complications can occur with both preparations, including heparin-induced thrombocytopenia (HIT) and immediate and delayed hypersensitivity reactions. Alternative anticoagulants are therefore needed.

Case Report

A 72-year-old woman received subcutaneous injections of the LMWH enoxaparin as prophylaxis in the post-operative period following a hip replacement. On the 11th day of receiving daily subcutaneous injections, she complained of itching at the injection site immediately following administration of the LMWH. She went on to develop an urticated, erythematous plaque on the abdomen confined to this injection site. Successive injections caused immediate itching and further urticarial-type areas at the injection site. The plaques became eczematous after a few days and settled with moderate potency topical corticosteroids and cessation of the LMWH injections. She had no previously known allergies and had not been treated with LMWH or heparin in the past.

She was investigated 2 weeks later by patch testing to the British Contact Dermatitis Society standard series, medicaments series, heparin and enoxaparin. This showed positive reactions to benzoyl peroxide and

Myroxylon pereirae resin of unknown relevance. Prick tests to unfractionated heparin, enoxaparin, tinzaparin, dalteparin and lepirudin were all negative (Table 1). Intradermal tests to unfractionated heparin, enoxaparin, tinzaparin and dalteparin were all positive after 5 min and at 48 h (Table 1). Intradermal testing to lepirudin was negative both at 20 min and 48 h.

Discussion

At least 3 types of immune-mediated reactions have been described to heparin:

- (1) An immediate Type I hypersensitivity reaction with an urticarial rash, asthma and anaphylaxis.
- (2) HIT resulting from IgG antibodies causing platelet activation. This is a progressive immune-mediated phenomenon, Type III hypersensitivity reaction occurring within 7–10 days of therapy, or within hours in a previously exposed patient (1–3, 6).
- (3) A delayed Type IV hypersensitivity reaction causing erythematous plaques or eczematous patches within 10–20 days at the injection site (4). The affected area may progress to skin necrosis. Re-exposure in those already sensitized can result in reactions within 24 h. A generalized exanthematous rash has been described (5).

The first 2 reactions are rarely associated with LMWH, though the delayed Type IV reaction is now well recognized. Our patient presented with both Type I and Type IV hypersensitivity reactions to LMWH. This was confirmed by positive intradermal tests to the LMWH at 5 min which persisted at 48 h. The lack of positive reactions on patch testing and prick testing with heparin and the LMWHs in our patient was not unusual, intradermal or subcutaneous testing being recommended (3). Unfractionated

**Type I and Type IV
allergy to unfractionated
heparin and low-
molecular-weight
heparin with no reaction
to recombinant hirudin**

Table 1. Results of intradermal testing

Compound tested	Prick tests (15 min)	Intradermal tests (15 min)	Intradermal tests (48 h)
Unfractionated heparin	–	+	+
Enoxaparin (LMWH)	–	+	+
Tinzaparin (LMWH)	–	+	+
Dalteparin (LMWH)	–	+	+
Lepirudin	–	–	–

LMWH = low-molecular-weight heparin.

heparin and LMWH are structurally similar, differing only in molecular weight (4). Cross-reactions between heparin and the LMWHs are therefore to be expected, as in our patient.

Lepirudin is a recombinant hirudin, related to the anticoagulant produced by the salivary glands of the leach *Hirudo medicinalis*. It is structurally unrelated to heparin and LMWH and mediates its anticoagulant effect by a different mechanism. Lepirudin has therefore been suggested as an alternative to heparin-type compounds in patients with HIT (5). Our patient had no reaction to lepirudin, supporting a lack of cross-reactivity between the heparins and this recombinant hirudin.

Our case is the first to report both Type I and Type IV reactions to unfractionated and LMWH. We have confirmed the cross-reactivity between heparin and different types of LMWHs and demonstrated that the recombinant hirudin, lepirudin, may be a safe alternative anticoagulant for our patient in the future.

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Contact allergy to thiuram in a musician

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Key words: instrument case; musician;
para-phenylenediamine; thiuram.

Case Report

A 12-year-old boy presented with recurrent swelling of the lips, during periods of prolonged rehearsal when playing the cornet or trumpet. Patch testing to the British Contact Dermatitis Society standard series and a metals series showed positive reactions to para-phenylenediamine (PPD) (D2+++/D4+++), thiuram mix (D2++/D4++) and IPPD (D2+/D4+).

He clearly had no history of using hair dye, but had developed a blistering reaction on his arm a few days after application of a temporary 'black henna tattoo' on holiday 2 years earlier. He had no previous history of reactions to rubber in his underwear, rubber soles or balloons, and he denied chewing on rubber bands or rubbers at the ends of pencils.

We went on to examine his instruments and discovered that he kept his cornet in a blue fur-lined foam case, and the trumpet in a black fur-lined foam case. Further patch tests to the fur and rubber foam linings of the instrument cases showed positive reactions to the rubber foam linings (D2+/D4+), but negative reactions to the darkly coloured fur linings. Contact urticaria testing to the black lining and PPD were negative. Prick testing to natural rubber latex was negative.

We advised our patient to keep the mouthpiece of his trumpet and cornet separate from his instrument cases and his symptoms have not recurred.

Discussion

Contact allergens previously reported in musicians include nickel (used in mouthpieces), colophonium (used to wax strings), propolis (used to varnish stringed instruments) and exotic woods like Indian rosewood and African blackwood found in chin rests and recorders (2, 3). There have been

no previous reports of thiuram allergy in a musician and no previous reports of an instrument case causing such a contact dermatitis.

His positive reaction to PPD was related to a previous 'black henna tattoo' (3). The IPPD reaction was likely to be a cross-reaction with PPD. It has previously been noted that patients with allergic contact dermatitis from PPD following a temporary 'black henna tattoo' may also have a thiuram allergy. This may be due to the application of a sticky compound prior to the henna tattoo (Dr C.R. Lovell, personal communication). It is therefore possible that our patient's initial sensitization to thiuram occurred at the same time as his sensitization to PPD.

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Occupational allergic contact dermatitis from 1,6-bis (2,3-epoxypropoxy) naphthalene in a one-component epoxy coating

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Key words: 1,6-bis(2,3-epoxypropoxy)naphthalene; allergic contact dermatitis; CAS 27610-48-6; non-standard epoxy resins; occupational.

Case Reports

Case no. 1

A 39-year-old atopic man gave a history of an itchy red blistering rash on his hands, forearms, and sometimes neck and face. He worked as a coating-machine operator at a plant producing electrical components for personal data assistants. A new epoxy resin had been introduced 2 months prior to the onset of the dermatitis. Its material safety data sheet gave its composition as 10%–20% 1,6-bis(2,3-epoxypropoxy)naphthalene, 10%–30% methyl hexahydrophthalic anhydride (MHHPA), and silica. He had been exposed to the epoxy resin when washing a nozzle with acetone and had not been adequately protected. Away from such work, the dermatitis settled within a week. Examination at the time of patch testing was normal. Patch testing was performed using Hayes Test Chambers (Hayes Service BV, Alphen, The Netherlands). He was patch tested to epoxy resin 1% pet. (Trolab[®], Hermal, Reinbek, Germany), bisphenol A 1% pet. (Trolab[®]), 1,6-bis(2,3-epoxypropoxy)naphthalene 0.5% and 1% in olive oil (o.o.), and MHHPA 1% o.o. At D3, he was positive only to 1,6-bis(2,3-epoxypropoxy)naphthalene 0.5% and 1% (+ and ++, respectively).

Case no. 2

A 32-year-old nonatopic man worked as a laboratory technician at the same plant. His job involved handling a variety of electrical insulating materials, including silicone resin, on a trial base. He was in direct contact with these, because protective gloves were not always worn. He had developed an itchy red blistering rash on his hands and forearms 3 months after the new epoxy resin was introduced. 5 months later, he started to have similar lesions around his knees. His skin settled after avoidance of the epoxy resin, but recurred on inadvertent re-exposure. Examination at the time of patch testing was normal. He was patch tested to epoxy resin 1% pet. (Trolab[®]), bisphenol A 1% pet. (Trolab[®]), 1,6-bis(2,3-epoxypropoxy)naphthalene 0.5% and 1% o.o., and MHHPA 1% o.o. At D3, he was posi-

tive only to 1,6-bis(2,3-epoxypropoxy)naphthalene 0.5% and 1% (++ and +++, respectively).

Comment

Occupational contact dermatitis due to naphthalene-type epoxy resin has previously been reported by Yokota et al. (1), though no patch tests were performed. Various non-diglycidyl ether of bisphenol A (non-DGEBA) epoxy resins have previously been found to be causes of allergic contact dermatitis (2). The present cases were ones of occupational allergic contact dermatitis from 1,6-bis(2,3-epoxypropoxy)naphthalene, another non-DGEBA epoxy resin. To the best of our knowledge, there are no previous reports of sensitization to this substance. 1% pet. is recommended for future patch testing, since 10 controls were negative to 1,6-bis(2,3-epoxypropoxy)naphthalene 0.5% and 1% o.o.

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Allergic contact dermatitis in 191 consecutively patch tested children

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Key words: allergic contact dermatitis; children; nickel; patch testing; PPD; thiuram.

We studied a population of children attending for patch testing in south Wales to determine the prevalence rates of allergic contact dermatitis (ACD), and to compare this with previous studies of both children and adults. We also aimed to identify allergens particularly relevant to childhood ACD.

Patients and Methods

The patch-test databases from 1993 to 2003 in 2 centres (Swansea and Cardiff – total population 677 500) were searched for all children, 16 years and under, with a positive patch test to the European standard series. The frequency of ACD and the spectrum of allergens was recorded.

Results

We identified 191 consecutively patch-tested children. 41% had a positive result to 1 or more allergens. Of these, 51.7% were documented to be of current relevance. 19.6% had multiple positive patch tests.

The most common allergen was nickel (13%), followed by fragrance (9%), thiuram (9%), cobalt (8%), para-phenylenediamine (PPD) (6%), tixocortol pivalate (5%) and Myroxylon pereirae resin (5%).

Discussion

A review of 17 studies of ACD in children under 16 years (sample size 53–1023 patients per study) (1) showed a positive patch-test frequency of 14.5–70.7%. Current relevance was found in 56.4–93.3% of these, and multiple reactions were found in 3.2–54.4% of cases.

Our results show similar prevalence rates of positive single and multiple patch-test reactions, but a lower percentage with current relevance. Nickel, cobalt, chromate, fragrance and formaldehyde produced the highest number of reactions with doubtful or no relevance, possibly due to an irritant effect or a cross-reaction.

The order of prevalence of ACD to individual allergens was largely comparable with a general adult population (2), with nickel, fragrance and Myroxylon pereirae resin having the highest rates. However, thiuram and PPD allergy was proportionately higher in children.

The frequency of PPD allergy showed an increased rate of 6.3% compared with most previous studies. 3 cases of PPD allergy were in trainee hairdressers with allergies to other work-related chemicals (ammonium persulphate in 2 and glyceryl thioglycolate in 1). 2 children had reacted to black henna tattoos, an increasingly common presentation in children.

The sources of these allergens were often related to sports and hobbies. Examples of these include thiuram or mercapto allergy related to cricket bats, badminton rackets golf clubs and trainers. 1 case of cobalt allergy was attributed to the wearing of a blue leather hockey glove, and 1 case of colophonium allergy was seen in a violinist. 1 teenager developed an allergy to benzoyl peroxide in her acne treatment.

Our study has shown that, despite the frequency of ACD in children being comparable with previous studies, the spectrum and frequency of individual allergens has changed in recent years. PPD and thiuram allergy have a higher frequency in our study compared with adult populations.

The sources of these allergens are often different from adults, and we have highlighted the need to consider this even more in our history-taking, when faced with the younger patient in the patch test clinic.

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Occupational allergic contact dermatitis from champignon and Polish mushroom

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Key words: airborne; allergic contact dermatitis; champignon; occupational; Polish mushroom.

The overall extent of mushroom allergy is unknown (1). Mushrooms are antigenically rich, and a species may have more than one allergen; Type I, Type III and Type IV immunopathologic reactions to different species of mushrooms have been reported (2–9).

Champignon mushroom can induce all 3 types of reaction, resulting in oropharyngeal and laryngeal constriction (11), alveolitis (12) and hand dermatitis (13). Airborne occupational allergic contact dermatitis due to champignons has previously been reported by Kanerva et al. (13).

Case Report

A 54-year-old Caucasian woman was referred to us in February 2004 because of severe erythema and oedematous dermatitis of her eyelids, nose, lips, cheeks, neck, arms and hands (Figs 1 and 2). Since 1998 she had

been working in a factory for commercial production of champignon (*A. ortensis* and *A. bisporus*) and of Polish mushroom (*B. edulis*).

She had a family history of allergic disease (both her daughters had allergic asthma due to *D. pteronyssinus*) and was herself affected by allergic asthma due to cat dander. In 1994, she had developed autoimmune hypothyroidism for which she received chronic substitutive thyroid hormone therapy.

Total IgE was within normal limits and a radioallergosorbent test (RAST) for champignons was negative. Prick tests for environmental allergens were positive to cat and dog dander as well as to *D. pteronyssinus*.

Prick-by-prick test with champignons *A. hortensis* and *A. bisporus* and Polish mushroom *B. edulis* was negative at 30 min but 48 h later resulted in erythema at the test site. Patch testing with the same mushrooms soaked in physiological saline resulted in a severe local reaction (skin erythema and infiltration >2.5 cm). 2 normal controls were patch tested with champignons and Polish mushroom and were negative. Testing for precipitins to *A. bisporus*, *A. hortensis* and *B. edulis* was positive only to *B. edulis*. Patch test (TRUE Test™, Pharmacia, Uppsala, Sweden) for standard antigens was positive only to nickel.

The patient stayed away from work for 2 weeks and received corticosteroid therapy, with complete remission of the skin lesions; the day after her return to work, dermatitis reappeared on her hands, face and neck. The patient permanently stopped working in the mushroom factory, obtaining



Fig. 1. Facial dermatitis with erythema and oedema.



Fig. 2. Allergic contact dermatitis on the hands.

complete remission of her dermatitis without any medication. It is worthwhile adding that the patient could safely eat champignons.

Discussion

We report here a case of airborne occupational allergic contact dermatitis from champignons, similar to the one reported by Kanerva et al. (13). In contrast to the case that they described, our patient developed contact dermatitis also on her hands, because she did not use disposable gloves.

In our patient, the positive patch tests and the negative RAST and prick tests for champignons and Polish mushroom confirm a Type IV immunopathologic reaction.

Workers in mushroom factories should be warned about the risk of mushroom allergy and precautionary measures should be taken to prevent its occurrence.

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Occupational facial allergic contact dermatitis caused by Finnish pine and spruce wood dusts

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Key words: facial allergic contact dermatitis; occupational; pine; spruce; wood dust

Case Report

A 20-year-old man had had flexural atopic eczema until 11 years of age. He worked at a sawmill as a forklift operator with fresh-sawn Finnish pine (*Pinus sylvestris*), spruce (*Picea abies*) and European white birch (*Betula pendula*) timber.

After 2 months he developed facial eczema and swelling. Patch tests were performed with the European standard series and the suspected wood dusts. Positive reactions were found to Myroxylon pereirae resin (++), colophonium (++), abietic acid (++), fragrance mix (++), pine sawdust (++) and spruce sawdust (++). Birch was negative. Prick tests to wood dusts were negative. He gave up working at the sawmill and the symptoms stopped. Later, when he carried out some repairs to a building, he again developed some symptoms.

Discussion

Occupational allergic contact dermatitis from wood dusts is not common (1, 2). In Finland, during 1990–96, 0.9% (24/2646) of cases reported to the Register of Occupational Diseases were caused by woods and teak caused most of the allergic reactions. The allergic contact dermatitis of 5 patients was caused by sawdusts of Finnish conifers (1). Some case reports have also been published concerning allergic contact dermatitis from pine dust (3–5).

Those who have had atopic eczema as children may not always be investigated as thoroughly as they should be for allergic contact dermatitis, in the mistaken belief that their eczema as adults must be a relapse of atopic eczema.

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