

CONTACT POINTS

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Allergic contact dermatitis from melphalan and chlorambucil: cross-sensitivity or cosensitization?

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Key words: allergic contact dermatitis; chlorambucil; cross-sensitivity; laboratory workers; melphalan; occupational; pharmaceutical manufacturing.

Case Report

A 21-year-old cytotoxics laboratory technician developed an itchy rash on his face 1 week after starting work in a new laboratory of a pharmaceutical company. He had been working for the company for 3½ years but had only recently been transferred to the quality assurance department. The rash appeared initially over areas on his face where his respirator dust mask touched the skin and later over uncovered areas. When it was reported to Occupational Health they instituted a withdrawal from the laboratory till the rash had cleared. They then started a 'trial of work' under close supervision. Initially, it was thought that the respiratory dust mask was the cause of the rash as the nose clip was made of latex and this would have fitted the profile of the rash. He was then tried using a paper dust mask but the rash had still recurred. He was again restricted from the laboratory and the rash cleared but recurred again when handling paperwork from the same laboratory.

His job involved working with cytotoxic drugs in a fume-hooded compartment. He would weigh the chemicals, dry them in ovens and run high-performance liquid chromatography. The drugs he worked with included azathioprine, chlorambucil, melphalan, 6-mercaptopurine and busulfan.

At the time of onset of his rash, he had himself been working with melphalan, but chlorambucil was also present in the laboratory at the time.

He had a 4-year history of psoriasis. He also reported previous skin sensitivity to nickel-containing objects. He had no history of asthma, hay fever or eczema but his mother had a history of eczema.

On examination, he had plaques of psoriasis on his elbows, knees and occiput. His face was clear as he had been away from work for some time at the time of consultation. He was patch tested to an extended European standard series and all the drugs that he had been working with. He showed

a ++ reaction to chlorambucil 1% pet. at day (D)2, which became a ++ by D4, and a ++ reaction to melphalan 1% pet. at D2 and D4, as well as a ++ reaction to nickel sulfate and a + reaction to cobalt chloride. He was diagnosed as having allergic contact dermatitis from melphalan and chlorambucil. He had also had psoriasis and allergic contact dermatitis from nickel and cobalt.

Discussion

Allergic contact dermatitis from the handling of drugs has frequently been reported, though there have been relatively few reports of allergic contact

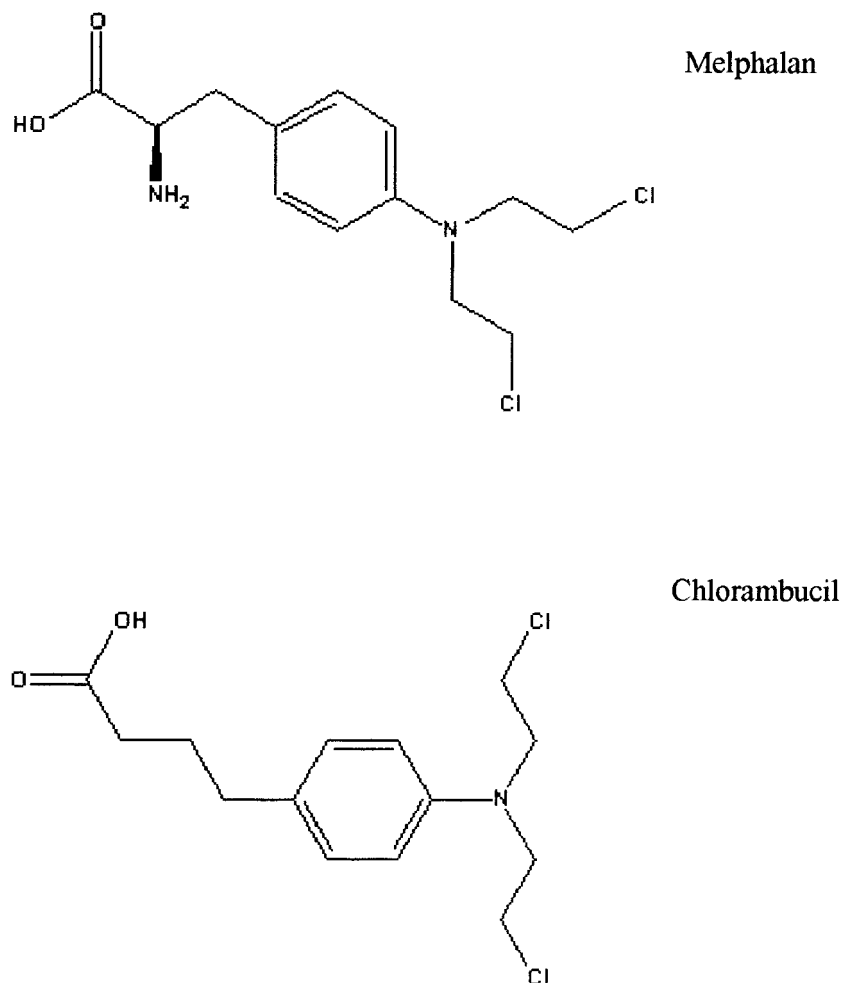


Fig. 1. Chemical structures of melphalan and chlorambucil.

dermatitis from cytotoxic drugs. To date, there have been such reports from mechlorethamine (1), nitrogen mustard (2), mitomycin C (3) and carmustine (4). A Medline search for allergic contact dermatitis from melphalan or chlorambucil found no previous reports.

Even though there was a stronger patch test reaction to chlorambucil than to melphalan, we thought that the primary sensitization was probably to melphalan, with cross-sensitivity to chlorambucil, as melphalan was the main drug with which the patient had been working prior to the onset of the rash. The molecular structures of these 2 drugs are similar enough to entertain the possibility of cross-sensitivity (Fig. 1), though co-sensitization also remains possible, as both drugs were present in the laboratory immediately prior to the onset of the dermatitis.

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Urticaria after topical immunotherapy with diphenylcyclopropenone

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Key words: alopecia areata; diphenylcyclopropenone; topical immunotherapy; urticaria.

Topical immunotherapy with contact allergens such as diphenylcyclopropenone (DPC) and squaric acid dibutylester (SADBE) has been shown to be effective in severe alopecia areata (AA) (1, 2). The mechanism has been debated (3–9).

Case Reports

Case no. 1

A 33-year-old man with a 2-year history of universal AA was sensitized with DPC 2% in acetone (acet.) on the left volar forearm. 2 weeks later elicitation was performed with DPC 0.0001%, 0.001% and 0.01% acet. on the contralateral volar forearm. Readings were performed at 2 days and topical therapy was then administered for 3 days/week with 0.001% DPC. After the 3rd application, the patient developed eczema on the scalp and forearm, as well as diffuse urticaria over the body, with intense itching and dermographism. DPC was stopped and antihistamines and betamethasone were administered for 6 weeks. After this time, we started applying DPC again at lower concentrations (0.0001% and then 0.00005%) and no cutaneous reactions were observed.

Case no. 2

A 37-year-old man had had AA since childhood, which had become universal by the age of 26. After sensitization and elicitation performed as in case no. 1, DPC 0.005% acet. was applied daily for 10 days, but was stopped when the patient developed an intense diffuse urticaria. For this case too, oral antihistamines and betamethasone followed. After ap-

proximately 2 months, the patient was administered DPC again at lower concentrations, but the appearance of the same cutaneous reaction did not allow further treatment.

Case no. 3

A 15-year-old girl had a history of universal AA since she was 2 years old. We applied DPC 0.0001% acet. 8–9 days after the start of treatment; she developed eczema and diffuse urticaria. The treatment was stopped and oral betamethasone was administered daily for 50 days. After that time, we resumed DPC 0.00001% acet. once a week without any reaction.

Discussion

Most of the side-effects associated with the application of DPC do not require discontinuation of therapy (10–14). Vitiligo (15), persistent hypo/hyperpigmentation (16) and erythema multiforme-like lesions (17,18), psoriasis of the scalp and contact urticaria (5) are rare. Only 1 previous case of diffuse urticaria during local immunotherapy with DPC has been reported, the pathogenesis of this phenomenon remaining unknown (13). The data from our patients led to 2 interesting considerations: (1) the phenomenon appears to be dose-dependent, since 2 patients out of 3 tolerated resumption of therapy at a lower dose; and (2) it is not a contact urticaria since symptoms do not start at the application site.

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Immediate and delayed hypersensitivity to ceftriaxone, and anaphylaxis due to intradermal testing with other β -lactam antibiotics, in a previously amoxicillin-sensitized patient

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Key words: antibiotics; beta-lactams; ceftriaxone; cross-sensitivity; cutaneous adverse drug reactions; hypersensitivity, occupational eyelid dermatitis.

Anaphylaxis, urticaria or maculopapular exanthemas from β -lactam antibiotics are common adverse drug reactions that have, at least partly, a comprehensible immunological basis. Contact dermatitis from these antibiotics, and in particular from ceftriaxone, is less common.

Case Report

A 31-year-old man had been working for 8 years in a pharmaceutical factory, packing powdered ceftriaxone. He had a previous history of asthma and hay fever, but not of atopic dermatitis. Prick tests were positive to pollen, animal epithelia, various latex proteins and *Dermatophagoides pteronyssinus*. He was on inhaled salmeterol and fluticasone propionate.

Four years ago, he had tolerated a 7-day course of amoxicillin/clavulanic acid. Six months previously, he had developed itchy periocular dermatitis while working, with resolution during vacations. At work he was frequently in aseptic areas wearing latex gloves, but 1 month without latex gloves did not improve his dermatitis. Two weeks without contact with powdered ceftriaxone, however, cleared the dermatitis within a few days.

Prick and intradermal tests with ceftriaxone 1:100 (stock solution 100 mg/mL) showed strong positive immediate (positive and negative

control: histamine/NaCl) and delayed reactions, an immediate weal developing into an infiltrated plaque after 24 h. Patch tests were ++ at D2 and +++ at D3. Lymphocyte stimulation test was increased for ceftriaxone, with a stimulation index of 2. A diagnosis was made of periocular dermatitis due to contact hypersensitivity to ceftriaxone. The patient continued working in the same area, wearing protective glasses, without further symptoms.

Because of the potential cross-reactivity among β -lactam antibiotics, further tests were performed 4 weeks later. Prick tests with penicilloyl-polylysine (Allergopen, Reinbek, Germany), benzyl penicillin–penicilloate (Allergopen), amoxicillin (5 mg/1 mL), amoxicillin/clavulanic acid (5.5/0.5 mg/mL), benzyl penicillin (10 000 IU/1 mL) and cefuroxime (0.75 mg/mL) showed only a slight erythema to cefuroxime. In contrast, subsequent intradermal tests resulted in erythematous weals within a few minutes. Shortly thereafter, the patient complained of nasal itching and obstruction, and shortly afterwards of nausea and feeling hot. Then he developed generalized urticaria and erythema of the face. Blood pressure and heart rate remained normal and he did not develop signs of bronchial obstruction. Immediately, 2 mg epinephrine s.c., and 2 mg clemastine and 100 mg hydrocortisone i.v. were administered. All symptoms completely disappeared within 4 h. There was also a positive immediate skin reaction at the site of the previous positive patch test with ceftriaxone, conducted 4 weeks earlier. Subsequent lymphocyte stimulation test with all intradermally tested antibiotics resulted in positive stimulation indexes for all drugs except amoxicillin. After the anaphylactoid reaction, the specific IgE was found to be slightly elevated for ampicillin (0.53 kU/L = class 1), while *in vitro* results for penicillin G, V and amoxicillin remained negative.

Discussion

β -lactam antibiotics (mainly penicillins and cephalosporins) may give rise to many kinds of adverse reactions, including both Type I and Type IV hypersensitivity. Beside the routine detection of specific IgE anti-

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bodies to various penicillins, interleukin-5 producing specific T-cell clones for amoxicillin and ceftriaxone were recently detected (1). Presumably because of similar 2nd ring and side-chain structures, cross-reactions between cephalosporins and penicillins occur, though probably not as frequently as previously estimated (2).

There are reports of occupational allergic contact dermatitis from all the cephalosporins, including 3rd generation ones (3). Some of these reports also show reactions to other 3rd generation cephalosporins, either by multiple sensitization or by cross-reaction (4); some reports do not show such reactions (5). Cross-reactivity among cephalosporins is theoretically due to the sensitizing tetrazolic group and thiazolyl-alkoxyl-iminicol side-chain (6) though some of the antigenic structures still remain unidentified (7).

The overall frequency of cutaneous hypersensitivity to ceftriaxone is 3% (8). Ceftriaxone-specific IgE antibodies may recognize the entire molecule of ceftriaxone (9), or side-chain structures in various β -lactam antibiotics (10); for example, the relatively stable (methoxyimino) acetyl side chain (2-amino-4-thiazolyl) in ceftriaxone and cefotaxime (11).

Hypersensitivity reactions to cephalosporins are generally less common than to penicillins, and anaphylactic reactions are rarer (12). Because 1st generation cephalosporins like cephalothin contained trace amounts of penicillin in the past, cross-reactivity was probably overestimated (13). Cross-reactions are presumably much less frequent with 3rd generation cephalosporins because of their 2 large side chains (14). The antigenic structures in β -lactam antibiotics for T-cell-mediated delayed hypersensitivity are not yet identified. Side-chain specific cross-reactivity rather than the β -lactam ring structures presumably account for most clinically observed immediate and delayed cross-reactivity, even though the majority of immediate reactions to penicillins are attributed to major and minor determinants of the penicillin molecule (15, 16). Overall, cross-reactivity between penicillin and cephalosporins with the same side chain varies between

12% and 38% (12, 15, 17). Cross-reactive side-chain specific IgE antibodies are known for example for amoxicillin and cephadroxil and for ampicillin and cefaclor (14). In a recent study, 41 well-characterized penicillin-allergic patients diagnosed by positive skin tests and/or provocation tests were all negatively challenged with 3 cephalosporins (cephazoline, cefuroxime and ceftriaxone) that did not share the same side chain with the penicillin that induced the reactions (18). Penicillin skin tests do not predict the likelihood of allergic reactions to cephalosporins in patients with a history of penicillin allergy (12, 19).

Salkind et al. (19) recently showed that among clinicians there is a tendency to overdiagnose penicillin allergy (only 10–20% of such patients are truly allergic on skin testing) because of the fear of penicillin anaphylaxis. They recommended that 2nd and 3rd generation cephalosporins could safely be administered to all patients except for those with a well-characterized history of immediate-type penicillin allergy confirmed by positive skin tests, and that even in these patients a cephalosporin could still be given after negative skin tests and/or after oral/i.v. challenge with a single test dose.

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Contact allergy to Tween 80 in an inhalation suspension

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Key words: asthma; breath spacer; CAS no. 9005-65-6; contact allergy; corticosteroids; emulsifier; medicaments; polysorbate 80; Tween 80.

Case Report

In a 7-year-old boy with severe recurrent asthma since the age of 2 months, treatment had been initiated at 10 months with budesonide (Pulmicort®, Draco, Lund, Sweden) pressure metered dose inhaler (pMDI) 200 µg twice daily in a spacer (Nebunette®, AstraZeneca, Lund, Sweden), prophylactically, and a beta 2 agonist, either salbutamol (Ventoline®, GlaxoSmithKline, Mölndal, Sweden) inhalation fluid 1 mg in a nebulizer (Portaneb®, Medic-Aid, Bognor Regis, United Kingdom) or terbutaline (Bricanyl®, Draco, Lund, Sweden) pMDI 0.25 mg in a spacer (Nebunette®), when required. Neither this nor beclomethasone (Becotide®, GlaxoSmithKline, Mölndal, Sweden) inhalation suspension during a 2-month period administered through the same nebulizer, plus the beta 2 agonists above, reduced the frequency of exacerbation. A change from Becotide® to budesonide (Pulmicort®) inhalation suspension 500 µg twice daily administered with the same nebulizer from the age of 2 years was equally ineffective. During

Table 2. Scratch test results

Substance	Concentration	Vehicle	20 min
Tween 80	5.0	Pet.	–

his 4th year of life, he had experienced monthly wheezing episodes lasting over 1 week. He had no other signs of atopy and prick tests (timothy, birch, mugwort, pollens, house dust mites, *Alternaria alternata*, *Cladosporium herbarum* and animal dander from cat and dog) were negative. When he was 5 years old, treatment was changed to pMDI fluticasone propionate 250 µg twice daily, in addition to the beta 2 agonists above. Within 2 weeks, he was free of symptoms and has since remained almost free while continuing on the same prophylactic medication. Seeking to explain this, he was patch tested to a paediatric series including the corticosteroid markers tixocortol pivalate, hydrocortisone-17-butyrate and budesonide, the various corticosteroids that he had used and an asthma/rhinitis series of constituents of medications for such diseases, including Tween 80, using Finn Chambers on Scanpor (Epitest, Tuusula, Finland) left on the back for 2 days, with readings at D3 and D7. Only Tween 80 was positive (Table 1). 2 years later, patch tests with Tween 80 and the budesonide inhalation solution were repeated (Table 1), and an additional scratch test with Tween 80 was negative (Table 2).

Discussion

The patient described developed contact allergy to the emulsifier Tween

80, present in the budesonide preparation used for asthma prophylaxis. Tween 80 is also called polysorbate 80 (INCI name) and polyoxyethylene sorbitan monooleate. Contact allergy to it is not common (1), even though it is widely used (2). Contact allergy in a child has been reported only once before and then in a topical medicament (1). No immediate allergy to Tween 80 was found. The negative patch test to Pulmicort® suspension can probably be explained by the inhibitory effect of budesonide on the patch test reaction (3).

Although inhaled contact allergens may cause both respiratory (4) and dermatological effects (5–8), the positive patch test to Tween 80 was unexpected. We believe that inhaled Tween 80 may have adversely affected the patient's lung function. For ethical reasons, we did not confirm this by an inhalation provocation test.

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Table 1. Patch test results

Substance	Concentration	Vehicle	D3	D7
Tween 80 ^a	5.0	Pet.	(+)	+
Tween 80 ^b	5.0	Pet.	++	NT
Budesonide ^a (paediatric series)	0.01	Pet.	–	–
Budesonide ^a	1.0	Pet.	–	–
Budesonide ^a	0.1	Pet.	–	–
Budesonide ^a	0.002	Pet.	–	–
Beclomethasone dipropionate ^a	1.0	Eth.	–	–
Fluticasone propionate ^a	1.0	Eth./DMSO (50:50)	–	–
Pulmicort® inhalation solution 0.25 mg/mL ^b	As is	–	–	NT

^a1st patch test session. ^b2nd patch test session 2 years later

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