

Short Communications

Systemic contact dermatitis due to deflazacort

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Key words: deflazacort; corticosteroids, erythema-multiforme-like; baboon syndrome; adverse drug reactions; occupational; pharmacist; medicaments; lack of cross-sensitivity. © Munksgaard, 2000.

Deflazacort (11 β ,21-dihydroxy-2'-methyl-5' β H-pregna-1,4-dieno[17,16-*d*]-oxazole-3,20-dione 21-acetate) is an oral corticosteroid now widely prescribed in Spain (1).

Case Report

A 27-year-old pharmacist, with minor thalassaemia (MT) and atopic dermatitis, had severe episodes of vesicular hand eczema, usually treated with oral corticosteroids. On 3 separate occasions, she had also developed a widespread macular exanthem, predominantly on the inner aspects of the upper and lower extremities and gluteal areas, with severe scaling, fever, nausea, vomiting, malaise, and hypotension. All 3 such episodes had begun a few hours after treatment with deflazacort 6 mg orally and prednicarbate cream (0.25%).

Skin biopsy revealed interphase dermatitis, with a scarce eosinophilic and lymphocytic infiltrate and some apoptotic keratinocytes, consistent with erythema multiforme. Direct immunofluorescence showed granular IgM deposits at the dermoepidermal junction.

Patch testing with the Spanish standard series and a corticosteroids series (betamethasone valerate and dipropionate, dexamethasone, triamcinolone, clobetasol, budesonide, flucortinbutyl, prednisolone, prednicarbate, mometasone, hydrocortisone, hydrocortisone butyrate and tixocortol pivalate) was negative at days 2, 4, 7 and 14. Controlled oral provocation tests with prednisone, methylprednisolone and dexamethasone were all negative.

A patch test with a tablet of commercial deflazacort 6 mg (1% aq.) produced a widespread erythema with intense pruritus 8 h after application, which disappeared totally after removal. Later, a patch test with pure deflazacort (1% aq.) was positive 2 h after application. 20 controls were negative.

Discussion

The majority of contact dermatitis from corticosteroids is due to topical application and occurs in 0.2–5% of patients (2). Delayed systemic allergic reactions to corticosteroids comprise various clinical patterns, sometimes reproducible with patch tests. Up to 1995, 24 such cases had been reported in the literature (3), due to oral pred-

nisone (5 cases), prednisolone (4 cases), dexamethasone (2 cases), triamcinolone (1 case) and betamethasone (1 case). 3 further cases due to oral prednisolone have since been published (4–6). Erythema-multiforme-like eruption has been described due to intra-articular injections of methyl prednisolone and triamcinolone acetonide (7, 8).

2 other cases of adverse reactions to deflazacort have been reported in Spain. One was a systemic reaction that started 1 h after the intake of 15 mg of deflazacort. Patch tests with deflazacort (1% eth. and 10% pet.), betamethasone, dexamethasone, budesonide and hydrocortisone were all positive (9). The other patient developed toxic epidermal necrolysis after 3 weeks of taking 90 mg/day of deflazacort orally. No patch tests with the drug were done (10).

Our cases showed the following features: 1. rapid onset after oral intake of deflazacort; 2. cutaneous reaction clinically similar to baboon syndrome; 3. dermatopathological picture resembling that of erythema multiforme; 4. widespread reaction after patch test with deflazacort; 5. no cross or concomitant reactions to other corticosteroids.

We think that patch testing with corticosteroids may explain some cases of cutaneous adverse drug reactions clinically diagnosed as baboon syndrome, erythema multiforme or macular exanthem, particularly to corticosteroids marketed in Spain (11). The origin of hypersensitivity to deflazacort was probably occupational in our patient, as in the case reported by Lauerma (12), and induced by handling tablets (13). The absence of cross-reactions to other corticosteroids may be explained by deflazacort being chemically so different from groups A, B, C, D1 and D2 (14).

Acknowledgment

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Pigmented purpuric clothing dermatitis due to disperse dyes

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Key words: allergic contact dermatitis; pigmented; purpuric; Disperse Orange 3; Disperse Blue 35; Disperse Blue 153; clothing dyes; disperse dyes; azo dyes; anthraquinone dyes. © Munksgaard, 2000.

Case Report

A 45-year-old housewife was referred to a dermatology clinic in 1993 with a 4-year history of an intermittent erythematous vulvar rash lasting 2 to 3 days, with stinging discomfort causing dyspareunia. She had no past or family history of skin problems. On examination, she had a discrete non-blanchable erythematous purpuric rash involving the vulva and perineum.

A biopsy taken from the perineum showed extravasation of red blood cells from dilated papillary blood vessels with a slight lymphocytic perivascular infiltrate, consistent with a diagnosis of Majocchi-Schamberg disease. Her other investigations, including full blood count, platelet count, bleeding and clotting time, liver and kidney function and auto-antibody screen, were normal.

She was prescribed dapsone 100 mg oral daily and a very potent topical corticosteroid cream, which cleared the dermatitis within a few weeks; however, dapsone was stopped as it caused haemolysis. The vulvar eruption persisted for 3 years until she was re-referred.

She was then patch tested with the European standard series, textile finishes and dyes and her own underwear. She had + reactions at 2 and 4 days to Disperse Orange 3, Disperse Blue 35 and Disperse Blue 153. The allergic reactions were not purpuric. She used to wear coloured underwear and changing to white underwear resulted in complete clearance within 4 months.

Discussion

Non-thrombocytopaenic contact purpuric dermatitis has been reported to be caused by various textile fabrics, rubber and antioxidants, various other chemicals and pressure (1–3). Many cases are attributed to dyes (2, 4). It has been suggested that allergic contact purpura may be due to an agent acting as a toxin or allergen with an affinity for vascular endothelium (3). However, the exact mechanism is still not known.

Our patient was unusual in that the purpura showed no evidence of dermatitis and occurred only on the vulva. Of the dyes that gave positive patch test reactions, Disperse Orange 3 is an azo dye and Disperse Blue 35 and 153 both anthraquinone dyes.

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Sensitivity to IPPD diagnosed a posteriori

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Key words: contact allergy; IPPD; rubber; boots; tyres; occupational; apiarist. © Munksgaard, 2000.

In Poland, sensitivity to IPPD has been becoming ever less frequent. In 1986, we had 4.10% consecutive patients positive to it, in 1999 only 0.71%.

A 59-year-old man, with no family history of atopy, for some years had worked as a bee-keeper and usually wore long rubber boots. He cut off part of them so that their tops were uneven. During work, he often defecated squatting in the meadow, when the uneven boot edges touched his thighs below the buttocks. Skin changes produced in this way persisted for various time periods and the patient never saw them. We think that this was initially irritant contact dermatitis and then allergic contact dermatitis from IPPD.

Working in the apiary, he sometimes sat on a bench covered with a piece of old tyre. He felt no symptoms if he wore long trousers, but once he wore shorts and sat down on the bench with his thighs in contact with the tyre, dermatitis developed at the sites of contact with the rubber and he was treated with a potent topical cortico-

steroid. 1 month later, on a hot day, he was wearing shorts and sat down again on the same bench. This time, after only 15 min, he felt itching and burning, and after several hours, a bulla developed at the sites of contact with the tyre. We treated him for several days with systemic corticosteroids. He never had any lesions on the hands or feet.

On patch testing, the standard series had to be removed after 12 h because of a severe bullous reaction to IPPD. He was given corticosteroids systemically. After 3 weeks, he was patch tested again (without IPPD) and was positive to PPD, 1.0% N-phenyl-2-naphthylamine (not used in Poland), and 1.0% CD-2 photographic developer, with which he had never had any contact. He was negative to benzocaine, aniline, TMTD and 2-MBT. He was also positive to rubber fragments from 4 types of tyres most popular in Poland, and 2 fragments of rubber boots that he used. During the 2nd test, flare-up developed at the site of the 1st patch test with IPPD.

Hyperpigmentation in a welder

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Key words: hyperpigmentation, heat; arc welding; occupational. © Munksgaard, 2000.

In arc welding, heat produced by electric current can cause erythema, and spurting of metals can give rise to dermatitis. The other hazards encountered are scars from burns and lacerations, skin cancer, chronic DLE and photo-ophthalmia (1).

Case Report

A 42-year-old welder had had hyperpigmentation over both extensor forearms for 8 years. He had used an electric arc-welding machine, emitting ionizing gas, ultraviolet and infrared radiation, and had worked for 3 to 4 h daily at a distance of 0.6 m from the machine. No protective clothing had been worn. 1 month after starting work, he had developed erythema followed by hyperpigmentation over the extensor forearms. Pigmentation had persisted despite stopping work.

Skin biopsy revealed a normal epidermis, with a non-

specific inflammatory infiltrate and melanophages in the upper dermis, and no evidence of amyloid.

Treatment with topical sunscreens, emollients and topical hydroquinone procured considerable improvement in 2 months.

Comments

We have previously reported hyperpigmentation in a furnace worker (2). In this case also, we attribute the hyperpigmentation to heat.

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Occupational contact dermatitis caused by a personal-computer mouse

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Key words: occupational; irritant contact dermatitis; mechanical; pressure; physical; skin; personal-computer mouse; ABS plastic; acrylonitrile; butadiene; styrene; phthalates. © Munksgaard, 2000.

Case Report

A 49-year-old woman, highly allergic to epoxy resin (1) and (meth)acrylates (2), could not continue her work as a cleaner because of occupational allergic contact dermatitis of the hands (1, 2). She had previously had positive patch test reactions to nickel sulfate (+++), cobalt chloride (++), para-phenylenediamine (++), p-tertiary-butylphenol-formaldehyde resin (++), mercury chloride (++), diaminodiphenylmethane (2+), diglycidyl ether of bisphenol A (DGEBA, ++) and various (meth)acrylates (1, 2). The patient was retrained to work with personal computers (PC). She was right-handed and used a PC mouse for up to 6 h per day. After 2 months, she started to develop an erythematous, patchy, partly vesicular dermatitis of the palmar aspect of the right hand located over the thenar and hypothenar eminences and metacarpophalangeal joints, and sparing the central palm (Fig. 1). This hand dermatitis improved when the patient stopped using the PC mouse, and relapsed on its reuse. The PC mouse was made of ABS plastic, i.e., a copolymer of acrylonitrile, butadiene and styrene.

In a further patch-test session, she was tested with scrapings (3) from her PC mouse with negative results. The acetone-soluble chemicals in the PC mouse and the PC mouse carpet were analyzed by gas chromatographic/mass spectrometric (GC/MS) and liquid chromatographic methods, as previously described (4, 5). (Meth)acrylates were not detected (detection limit 0.003%). Small amounts of styrene, α -methylstyrene, di-

cumyl peroxide and bis(6-hydroxy-3-methyl-5-tert-butylphenyl)methane were identified.

Discussion

Capon et al. (6) reported occupational allergic contact dermatitis from diethyl and dimethyl phthalates in computer mice. Our patient was negative on patch testing to dimethyl phthalate (5% pet.), dibutyl phthalate (5% pet.), dioctyl phthalate (2% pet.) and diethylhexyl phthalate (5% pet.), though patch testing with diethyl phthalate was not performed. Recently, Walker et al. (7) reported allergic contact dermatitis from headphones containing diethylhexyl phthalate. Their patient also had an allergic patch-test reaction to phenol-formaldehyde resin (7). Interestingly, our patient also had allergic patch test reactions to phenol-formaldehyde resin (+, 5% pet.), urea-formaldehyde resin (+++, 10% pet.) and melamine-formaldehyde resin (+++, 7% pet.).

Our patient was exceptionally allergic to DGEBA (1) and (meth)acrylates (2) and her dermatitis of the hand, related to handling the PC mouse, clinically resembled allergic contact dermatitis. Styrene, used in the production of ABS plastic, is a rare cause of allergic contact dermatitis (8, 9), but the amount of styrene was very low in the PC mouse and, as styrene is only a weak sensitizer, it would, in this case, be a very unlikely contact sensitizer. On patch testing, our patient was negative to benzoyl peroxide though could theoretically have been allergic to other peroxides used in plastics (10). It was concluded that the hand dermatitis was irritant and caused by contact with the PC mouse. Sweating may have been a contributory factor, though the patient did not have hyperhidrosis. It seems feasible to assume that the patient's earlier episodes of contact dermatitis of the hands (1) were a predisposing factor, as most people can use a PC mouse without skin problems. The patient had no history of dyshidrotic eczema. Mechanical trauma is not very commonly reported as a cause of occupational dermatosis. In a Finnish study, 3% of occupational dermatoses were caused by mechanical causes (11). This PC mouse dermatitis appears to have evolved as a result of friction, sweating, pressure and other physical factors in a patient predisposed to hand eczema (12).

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Fig. 1. Mouse dermatitis appearing as patchy, vesicular eruptions on the right palm.

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Allergic contact dermatitis due to beclometasone dipropionate in an inhalant for asthma

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Key words: allergic contact dermatitis; allergic contact cheilitis; beclometasone dipropionate; inhalant for asthma; corticosteroids; medicaments; lack of respiratory effect. © Munksgaard, 2000.

Case Report

A 51-year-old woman noted scales and fissures on the lips and erythema and pigmentation on the perioral area and jaw. Patch tests included her inhalant, toothpaste, lipstick and face soap, using Finn Chambers[®]. Reactions were read at D2 and D3 according to the recommendations of the ICFRG. The inhalant (Aldecin[®]) and its active component, beclometasone dipropionate, showed positive reactions (Table 1). Beclometasone dipropionate (Propaderm[®]) cream, which had been used for treatment of her eruption, was also positive.

Although she had allergic contact dermatitis from be-

clometasone dipropionate in the inhalant, no exacerbation of asthma nor stomatitis was observed. Cheilitis and dermatitis cleared after changing her inhalant.

Discussion

Allergy to beclometasone dipropionate has been reported rarely (1, 2) and allergic contact dermatitis from beclometasone dipropionate in an inhalant must be extremely rare. Although beclometasone dipropionate (Propaderm[®]) cream worsened her skin eruption, beclometasone dipropionate inhalant did not exacerbate her asthma or provoke a stomatitis, suggesting that the immune system of the mucous membranes might differ from that of the skin.

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Table 1. Patch test results

		D2	D3
Aldecin [®] inhalant	as is	+	+
beclometasone dipropionate	20% aq.	+	+
Propaderm [®] cream	as is	+	+
controls (n=20)		—	—

Immediate contact reaction to 2-hydroxyethyl methacrylate?

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Key words: (meth)acrylates; 2-hydroxyethyl methacrylate; immediate contact reaction; patch testing; scratch testing; dental patients. © Munksgaard, 2000.

Dentin-bonding (meth)acrylates are strong dental sensitizers (1). Occupational contact allergies to dental (meth)acrylates are increasing (2, 3). Long-standing finger-pulp eczema, with hyperkeratotic scaling skin and painful fissures, reduced sensation and sometimes paresthesia, is typical (4). 2-hydroxyethyl methacrylate (2-HEMA) is one of the (meth)acrylates most often found positive on patch testing (1, 5, 6).

Case Report

A 24-year-old woman was referred by her dentist. For the first time in 1996 and again in November 1999, during her dental appointment, she had developed flushing of the face and presternal region, generalized tremor, perioral sensations and itching of both hands. The dentist had treated these presyncopal symptoms with adrenaline and prednisolone, after which they had gradually disappeared. No oral or perioral lesions were observed. The patient's dermatological and general medical history were unremarkable and she did not take any medication.

On both visits, the dentist had treated her with composite materials and a dentin-bonding agent. He confirmed that the reaction appeared after application of the bonding agent. On other visits, she had never encountered any problems at the dentist. In 1997, she had been patch tested with the standard series, a dental series and materials used by her dentist. No relevant reactions were observed at that time. Immediate tests showed positive reactions to several inhalational allergens, and thus an atopic constitution.

After the 2nd episode in November 1999, she was referred for further investigation. Because no dermatitis had been observed and because the symptoms were more closely related to a Type I than to a Type IV allergic reaction, we performed 20-min patch tests with the materials supplied by her dentist. Because there was no clearly positive reaction after 20 min, we repeated the patch test for 40 min. The materials tested are shown in Table 1. We also performed a prick test with 1000, 10,000 and 100,000 BU/ml latex and the local anesthetic Septanest.

The patch tests were performed using van der Bend patch test chambers, affixed with Fixomull Stretch tape. The test substances were removed after 20 min the 1st time and after 40 min the 2nd time. The test results were read according to international guidelines immediately after the 20 min and after the 40 min. The patient was asked to return to the department if any later reaction should occur. The same tests were also performed in 5 healthy control subjects.

After 20 min patch test duration, there was a weak + reaction to Clearfil Photobond Catalyst Liquid 2.0% pet. After 40 min patch test duration, we observed a + reaction to Hytac Aplutip Compromer Bonding as is and to Clearfil Photobond Catalyst Liquid 2.0% pet. After 9 h, the patient returned to the department with a + reaction that had occurred to 2-hydroxyethyl methacrylate 1.0% pet. This local reaction persisted for 14 days. The prick tests with latex and the local anesthetic Septanest remained negative. The tests in the control subjects were all negative.

Because it was still not clear to us whether an immediate type of allergic reaction was involved, we performed scratch tests 1 month later. The skin was scratched on 4 different sites on the volar forearms. We then applied Clearfil Photobond Catalyst Liquid 2.0% pet. and 2-hydroxyethyl methacrylate 1.0% pet., respectively, for 20 min. The other 2 scratched sites were used as positive (histamine) and negative (petrolatum) controls. After 20 min, the substances were removed and the test sites were read. All sites were negative except for the positive control. About 8 h later, an itchy, erythematous reaction appeared on the scratched sites where Clearfil Photobond Catalyst Liquid 2.0% pet. and 2-hydroxyethyl methacrylate 1.0% pet. had been applied for 20 min. Both reactions disappeared again after another 12 h.

Discussion

Clearfill Photo Bond (Kuraray Co. Ltd.) is a dual-curing dentin and enamel-bonding agent for use with composite light-curing restorative materials. The components of

Table 1. Materials used by the patient's dentist and patch tested for 40 min

benzoyl peroxyde	1.0% pet.
N,N'-dimethyl-p-toluidine	2.0% pet.
2-hydroxyethyl methacrylate	1.0% pet.
BIS-EMA	1.0% pet.
BIS-GMA	2.0% pet.
BIS-MA	2.0% pet.
bisphenol A	1.0% pet.
methyl methacrylate	2.0% pet.
Clearfil Photobond Catalyst Liquid	2.0% pet.
Clearfil Photobond Universal Liquid	2.0% pet.
epoxy resin	1.0% pet.
hydroquinone	1.0% pet.
ethyleneglycol dimethacrylate (EGDMA)	2.0% pet.
primer and bond dental adhesive	2.0% pet.
primer and bond dental adhesive	as is
Hytac Aplutip Compromer Bonding	2.0% pet.
Hytac Aplutip Compromer Bonding	as is

the catalyst liquid are bisphenol A diglycidyl methacrylate, 10-methacryloyloxydecyl dihydrogen phosphate, 2-hydroxyethyl methacrylate, hydrophobic dimethacrylate, benzoyl peroxide and *dl*-camphorquinone. The universal liquid to use with it contains N,N'-diethanol-p-toluidine, sodium benzene sulfonate and ethyl alcohol.

We assume that the + reactions to Clearfil Photobond Catalyst Liquid after 20 min, and to 1 of its constituents 2-hydroxyethyl methacrylate after 9 h, are relevant, though the underlying mechanism remains unclear. The patient's symptoms seem likely to have been caused by a Type I allergic reaction, but the test reactions do not support this. On the other hand, a Type IV allergic reaction would probably have caused a different clinical picture, with gingivitis, stomatitis and perioral dermatitis, and later reactions during patch testing, as described by Kanerva (7). Very late patch test reactions to acrylates have also been described (8). There have been reports of immediate hypersensitivity and/or bronchial asthma from acrylates or acrylic acid (1, 9), though the exact mechanism remains mysterious.

It is also difficult to determine the best test method in such a case. Kanerva (10) states that dental acrylics should never be patch tested as is. He also discourages the practice of use tests, open tests, or repeated open patch tests with undiluted dental acrylics, because of the risk of active sensitization from single exposure. It is difficult to interpret the patient's symptoms and the patch and scratch test reactions. Can her symptoms be described as an immediate contact reaction? Can we interpret the reaction after 9 h to a patch-test duration of 40

min as a very late Type I reaction, or should we see it as an allergic reaction in between a Type I and Type IV allergy? The same question can be asked about the reactions to the scratch tests, which appeared after 8 h and disappeared again another 12 h later.

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Failure of patch testing with levobunolol eyedrops to detect contact allergy

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Key words: allergic contact dermatitis; medicament; eyedrops; β -blocker; lack of cross-sensitivity; patch-testing technique; repeated open application test; false negative. © Munksgaard, 2000.

Case Report

A 59-year-old woman presented with a 6-month history of bilateral symmetrical eyelid dermatitis. There was no past dermatological history and she was not atopic. Glaucoma had been diagnosed 4 years earlier and treated with BetaganTM eyedrops (levobunolol 0.5%) throughout. She had also changed her spectacles around the onset of the dermatitis.

Patch testing was performed with reagents supplied by ChemotechniqueTM on IQ ChambersTM, with a 2-day (D) application time and readings at D2 and D4. Tests included the European standard series and BetaganTM eyedrops and their excipients, including benzalkonium chloride and sodium metabisulfite. A ROAT was performed with BetaganTM eyedrops applied to the volar aspect of the forearm for 14 days. A spectacles patch test series was also added. All tests were negative.

In view of the continuing dermatitis and the clinical suspicion of allergic contact dermatitis from medicaments, the eyedrops were changed to XalatanTM, containing letanoprost, and there was complete resolution of the dermatitis. A supply of pure levobunolol was then obtained courtesy of the manufacturers, Allergan, and patch testing performed to levobunolol 10% pet. The D2:D4 readings were ?+:++.

Discussion

Allergic contact dermatitis from β -blockers in eyedrops is infrequent. Previous reports of allergy to levobunolol (1–4) have all reported positive patch test reactions to the concentration of active drug contained in the final product. This report emphasizes the need to test to higher concentrations in some cases if allergy is not to be

overlooked. Lack of cross-reactivity between β -blockers used in topical medicaments is also supported.

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Delayed prick test reaction identifies amoxicillin as elicitor of baboon syndrome

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Key words: baboon syndrome; amoxicillin; systemic contact dermatitis; adverse drug reaction; delayed prick test reaction. © Munksgaard, 2000.

Case Report

A 72-year-old man, without any previous history of allergy, started taking Flanamox[®] tablets (amoxicillin 250 mg, flucloxacillin 250 mg) 3× daily for secondary wound infection. After 2 days, he developed a pruritic erythematous eruption, which involved particularly the buttocks, inner aspects of the upper thighs, axillae and upper trunk. A mild maculopapular rash was also present on the trunk. Medication was immediately discontinued and the skin lesions completely resolved with 2 weeks of topical corticosteroid.

5 months thereafter, prick tests were performed with penicilloyl-polylysine, benzylpenicillin (Penicillin Grünenthal), Flanamox[®] tablets (amoxicillin and flucloxacillin), Amoxypen[®] tablets (amoxicillin), Staphylex[®] (flucloxacillin) and ampicillin (Binotal). There were no immediate-type reactions at the 20-min reading, but erythematous infiltrations to Flanamox[®] (measuring 1×1 cm) and Amoxypen[®] (0.5×0.5 cm) were present on day (D) 2 (Table 1).

Discussion

Andersen et al. (1) introduced the term baboon syndrome to characterize a particular clinical presentation of systemic contact dermatitis: a pruritic, confluent, maculopapular, light-red eruption localized to the gluteal

area and the major flexures, developing a few hours up to 5 days after oral, inhalational or parenteral administration of the eliciting agent. The development of blisters (2) or pustules (3) in the respective areas has also been observed.

So far, the cause of this pathognomonic distribution has not yet been elucidated, though skin occlusion or sweating might be responsible (1). Histopathologic evaluation of the lesions in most cases has revealed non-specific features of dermatitis (2–6), in 1 case there having been leucocytoclastic vasculitis (1). Thus, particularly T-cell mediated delayed-type immune reactions, but possibly also immune complex-mediated vasculitis, elicit the baboon syndrome.

Patch tests are considered to be the most reliable diagnostic procedure for allergen identification in patients with baboon syndrome (1, 5–9). In 2 patients, intradermal tests were performed in parallel, 1 of them yielding a delayed-type reaction confirming the patch-test result (5, 6). Surprisingly, prick tests have been performed in only a few patients, and positive prick test reactions have not so far been published (4, 8). In our patient, amoxicillin was identified as the elicitor by delayed-type prick test reactions and a conclusive history of exposure to this compound (Table 1). There were no such prick test reactions in 10 controls.

In our opinion, allergologic diagnosis of cutaneous drug reactions should start with prick tests irrespective

Table 1. Test results

		Prick		i.d. (1:100)	
		20 min	D2	20 min	D2
Flanamox [®] (amoxicillin and flucloxacillin)	100 mg/ml	–	+++*	NT	
Amoxypen [®] (amoxicillin)	100 mg/ml	–	++*	NT	
Staphylex [®] (flucloxacillin)	100 mg/ml	–	–	NT	
penicilloyl-polylysine	0.035 mg/ml	–	–	–	–
ampicillin (Binotal)	100 mg/ml	–	–	–	+++*
benzylpenicillin (Penicillin-Grünenthal)	0.1 Mio IE/ml	–	–	–	++*

* No reaction in 10 controls.

of the clinical presentation. Since there is no evidence of an immediate-type mechanism in the baboon syndrome, it is important to focus on the delayed reading. If appropriate sterile test material is available, intradermal tests might be done in patients with a suggestive history and without a prick-test reaction after 2 days. Otherwise, patch testing is indicated if a delayed-type mechanism is considered and the aforementioned testing yields no conclusive results.

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Occupational contact dermatitis due to gold

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Key words: occupational; allergic contact dermatitis; gold plating; electrolytic process; gold sodium thiosulfate; cobalt. © Munksgaard, 2000.

Allergy to gold or its salts has been known for more than 30 years (1). Occupational allergic contact dermatitis due to gold is infrequent in automated industrial processing.

Case Report

A 22-year-old man, with no relevant personal or family history, working at a cutlery factory in the electrolytic gold-plating section, presented with a 2-year history of dermatitis over the backs of the hands and fingers. He had no dental restorations and no previous history of intolerance to metals. When examined, he showed clearly demarcated erythematous violaceous plaques, with a shiny whitish surface, on the backs of the hands and fingers, suggestive of a lichenoid eruption. At work, he handled, without gloves, a solution containing 5% gold trichloride and 0.006% cobalt and nickel.

On patch testing, the GEIDC standard series was positive to cobalt chloride 1% pet. (++) , ethylenediamine dichloride 1% pet. (++) and thiuram mix 1% pet. (++) at 2 and 4 days (D). The Chemotechnique® cutting oils and metal series showed a positive reaction to gold sodium thiosulfate 2% (D2 ++, D4 +++), there still being a positive reaction at 10 days.

Leaving the job that he had been carrying out up to then led to complete remission of symptoms within a 4-month period.

Discussion

The morphological appearance of the cutaneous lesions provoked by gold is quite variable, including tattoo, eczema, lymphomatoid contact dermatitis and lichenoid eruption. Lichenoid eruptions are characterized by shiny-surfaced violaceous plaques, which disappear once contact with the allergen has ceased. The importance of gold as sensitizer has been much discussed during recent years, and still is being (2–5). The main exposure sources are personal jewellery and dental alloys. As to the working environment, the occupations most frequently causative of occupational contact dermatitis due to gold are photography, chinaware or glass decorating, jewellers or manufacturers of dental alloys (6).

Gold trichloride appears to have been the main cause of the lichenoid dermatitis in our patient, since the level of cobalt in his electroplating solution was low and variable.

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Contact dermatitis from estradiol in a transdermal therapeutic system

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Key words: estrogen substitution; transdermal therapeutic systems; Type-IV hypersensitivity to estradiol; contact dermatitis; medicaments; vaginal tolerance. © Munksgaard, 2000.

Case Report

A 29-year-old woman with Ullrich Turner syndrome (UTS), who therefore needed continuous estrogen replacement, was treated with oral Triquilar® (ethinylestradiol and levonorgestrel), but 2× developed intrahepatic cholestasis with jaundice. Changing from oral estradiol to oral Cycloproginova® (estradiol valerate and norgestrel) led to raised serum liver enzymes. She was therefore switched to a transdermal therapeutic system (TTS) containing estradiol.

Within a few days of application of Estraderm® TTS, she began to experience redness at the site of application, which worsened with a Menorest® 37.5 patch and an Evorel® patch. All these applied TTS contained estradiol as their active component. During estrogen administration by TTS, serum liver enzymes remained within normal limits. When we saw the patient, she had a large (3×3 cm) patch of infiltrated erythema on the right thigh, which had appeared 5 h after application of an Evorel® patch.

Following resolution of cutaneous symptoms, patch tests with all 3 complete TTS and with their separate components were performed (Table 1). Positive results after 2 and 3 days were recorded to the 3 TTS and to estradiol at various concentrations (1%, 2%, and 4% in 96% eth.). The results were negative to all other components of the TTS, including ethanol 96%. The same estradiol concentrations did not elicit positive reactions in 10 volunteers who were patch tested for other reasons.

Because of her contact dermatitis, the patient was switched to Estrifam® (estradiol) tablets for vaginal application. Within 1 year, serum liver enzymes increased again, so that she also had to stop this medication. Changing to Ovestin® (estriol) ovula normalized liver enzymes, but from the gynecological viewpoint, the effects were too slight; therefore, treatment was stopped again. The patient tolerated vaginal application of estradiol-containing tablets and the vaginal use of estriol-containing ovula, without developing any intravaginal contact reaction.

Discussion

In UTS, low-dose systemic estradiol is optimal (1), but can lead to intrahepatic cholestasis, jaundice and raised

serum liver enzymes (2, 3). In contrast to oral conjugated estrogens, TTS has no such effects (4), other advantages including avoidance of gastrointestinal inactivation and variable absorption, avoidance of first-pass metabolism by the liver, constant serum concentrations, and improved patient compliance (5).

Only 1% of those receiving transdermal estrogen withdraw from treatment because of a skin reaction (4), though rarely sensitization to the active drug of TTS does occur (4), and more often to other TTS components, including the adhesive (6), hydroxypropyl cellulose (6, 7), and ethanol (8), irritant contact dermatitis being much the more common event (4, 5). Sensitization to estradiol is particularly rare, only 2 patients having been reported with a positive patch test to estradiol itself (6, 9, 10).

Our patient tolerated vaginal estradiol without developing any intravaginal contact reaction. To our knowledge, this has not been reported before. A possible reason for this tolerance is the dependence of vaginal Langerhans cells on hormonal status (11). Additionally, our patient tolerated vaginal estriol, a decomposition product of estradiol that was also negative on patch testing. The latter is a good example of the high specificity of human Langerhans cells.

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Table 1. Patch test results

		D2	D3
Estraderm TTS and separate components:			
1. Estraderm TTS 50 patch		+++	+++
2. estradiol	1% eth.	+	+
3. estradiol	2% eth.	++	++
4. estradiol	4% eth.	++	++
5. paraffin	as is	—	—
6. adhesive	1% benz.	—	—
7. Klucel HF	1% eth.	—	—
8. Kucel HF	2% eth.	—	—
Menorest 37.5 patch and separate components:			
1. Menorest 37.5 patch		+++	+++
2. vinyl acetate resin (Elvax)	2% acet.	—	—
3. bentonite NF	5% aq.	—	—
4. mineral USP	as is	—	—
5. estradiol USP	1% eth.	+	+
6. estradiol USP	2% eth.	++	++
7. estradiol USP	4% eth.	++	++
8. oleic acid NF Emersol 6321 pure		—	—
9. Alcolac 622/propyleneglycol Phosal 50 P	5% pet.	—	—
10. dipropylene glycol	5% pet.	—	—
11. 1,3 butylene glycol	5% pet.	—	—
12. Duro-Tak 87-2196	as is	—	—
Evorel patch		+++	+++
(separate components not distributed by manufacturing firm)			
Estrifam tablet and separate components:			
1. Estrifam tablet		+++	++
2. estradiol	1% isoprop.	—	—
3. estradiol	2% isoprop.	—	++
4. estradiol	4% isoprop.	—	++
5. talcum	as is	—	—
6. colour	as is	—	—
7. lactose	as is	—	—
8. cornstarch	as is	—	—
9. magnesium stearate	as is	—	—
10. gelatine	as is	—	—
11. methylhydroxypropyl cellulose	1% isoprop.	—	—
12. titanium dioxide	0.1% pet.	—	—
Ovestin ovula and separate components*			
1. Ovestin ovula		—	—
2. estriol	1% eth.	—	—
3. estriol	2% eth.	—	—
4. estriol	4% eth.	—	—
(as separate components, the manufacturing firm distributed only estriol)			

titis from hydroxypropyl cellulose in a transdermal estradiol patch. *Contact Dermatitis* 1988; 18: 106–107.

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Delayed-type natural rubber latex allergy not accompanied by immediate-type

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Key words: allergic contact dermatitis; hand eczema; natural rubber latex; delayed-type; patch testing. © Munksgaard, 2000.

There were no reports concerning immediate-type natural rubber latex (NRL) allergy until the 1970s (1). From the 1980s, many such reports were published. The 1st Japanese case report of contact urticaria from NRL was in 1993, since when many further cases have been reported in Japan (3–15), mainly of the immediate-type.

Patients and Methods

Case no. 1

A 39-year-old housewife with a past history of allergic rhinitis and metal allergy had had hand dermatitis since 1990. That same year, she had used rubber gloves to protect her hands. She had been treated for her dermatitis with topical and systemic corticosteroids, but her symptoms had persisted.

Case no. 2

A 36-year-old male hairdresser with no relevant past history, had had hand dermatitis since June 1999. He had used rubber gloves to protect his hands while working since December 1999. He had treated his dermatitis with topical and systemic corticosteroids, but his symptoms remained.

Specific IgE

We used the method of Pharmacia CAP-RAST (Pharmacia & Upjohn, Tokyo, Japan) and Alastat (SRL, Inc., Tokyo, Japan). We examined specific IgE for latex, banana and avocado using Pharmacia CAP-RAST system and specific IgE for chestnut using the Alastat system.

Prick testing

For prick testing, we incubated 1 g of latex gloves supplied by the patients, 1 g Baxter Triflex (Tokyo, Japan) and 1 g ammoniated latex sheet (Pacific Dunlop Japan K.K., Tokyo, Japan) in 5 ml of saline. We also incubated 1 g of banana, 1 g of avocado and 1 g of chestnut in 10 ml of saline. After 30 min, latex sheet, latex gloves and foods were removed and filtered through sterilized papers. The solutions were stored in sterile bottles and used for scratch testing. We used saline (Otsuka Pharmaceutical Co, Ltd, Tokyo, Japan) as a negative control and 10 mg of histamine chloride (Wako Pure Chemical Industries, Ltd., Osaka, Japan) in 1 ml of saline as a positive control.

Using Prick-Lancetter (1 mm) with shoulders to prevent deeper penetration, we carried out prick tests with test solutions. Readings were made at 20 min after placing samples according to Lahti (16): – = not reacted; 2+ = 1/2 histamine solution's reaction-sized erythema

and 1/2 histamine solution's reaction-sized edema; 3+ = histamine solution's reaction-sized erythema and histamine solution's reaction-sized edema; 4+ = erythema and edema stronger than the histamine solution's reaction.

Patch testing

We carried out 2-day (D) closed patch testing with latex gloves supplied by the patients as is, ammoniated latex sheet as is, thiuram mix (Japanese Society for Contact Dermatitis; JSCD), PPD black rubber mix (JSCD), mercapto mix (JSCD), dithiocarbamate mix (JSCD) (17), saline (negative control) and petrolatum (negative control), according to ICDRG recommendations.

Results

The results of specific IgE, prick testing and patch testing are shown in Table 1.

IgE RAST

Case nos. 1 and 2 were both negative for specific IgE to latex, banana, avocado and chestnut.

Prick testing

Case nos. 1 and 2 were both negative to all materials on prick testing.

Table 1. Results of investigations in case nos. 1 and 2

Latex IgE RAST	Case no. 1		Case no. 2	
	<0.35 UA/ml		<0.35 UA/ml	
prick testing				
extract of patient's latex gloves	–		–	
Baxter Triflex extract	–		–	
ammoniated latex sheet extract	–		–	
patch testing	D2	D3	D2	D3
patient's latex gloves	+	+	+	+
ammoniated latex sheet	+	+	+	+

Table 2. Patch testing with ammoniated latex sheet in controls

	D2	D3
–	36	40
?+	3	0
+	1	0
++	0	0
total	40	40
%≥?+	10.0	0.0
%≥+	2.5	0.0

Patch testing

Case nos. 1 and 2 both reacted positively (D2/D3 +/+) to the latex gloves supplied by the patients and ammoniated latex sheet, and negatively to thiuram mix, PPD black rubber mix, mercapto mix and dithiocarbamate mix.

We conducted control patch testing in 40 subjects using ammoniated latex sheet as is. We read: - 36 cases; ?+ 3 cases; + 1 case at D2; - all 40 cases at D3 (Table 2).

Discussion

Several previous reports have described delayed-type latex allergy (18–23), though few unaccompanied by immediate-type, and none such from Japan (13–15). Ammoniated latex sheet proved to be a detector of delayed-type NRL allergy on patch testing, with no false-positive reactions by D3.

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