Accepted for publication 26 May 2007
Allergy 2007: 62:1345–1346
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DOI: 10.1111/j.1398-9995.2007.01468.x

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Airborne occupational allergic contact dermatitis from coal dust

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Key words: airborne contact dermatitis; coal dust; occupational; patch test.

Airborne contact dermatitis is a common problem in occupational dermatology (1). Various kinds of dust particles, vapours and gasses present in the work environment can be implicated. Coal is a fossil fuel extracted from the ground, composed primarily of carbon along with assorted other elements and is primarily used a solid fuel to produce electricity and heat through combustion. According to the literature, the incidence of allergic contact dermatitis to coal or its products is small.

Coal dust, a particle implicated in occupational contact dermatitis.

A 52-year-old man employed in a power station for more than 10 years, presented a 6-month history of a symmetrical eczematous eruption in his thighs, backside, trunk and arms, being more intense on flexural areas. He had no personal and familial history of atopy. Control measures taken in the factory were respiratory protection and protective clothing (gloves and safety glasses with side shields). He also used long cotton underwear, but he referred the coal dust passed under protective clothes because of his skin being black when he was taking them off. There was nobody else in his job with his symptoms and the lesions reduced on vacations and worsened after returning to work.

Because of the clinical features, the distribution of the lesions and the work exposure, we suspected an occupational airborne allergic contact dermatitis. Skin biopsy was taken showing light surface dermatitis and lymphocytic perivascular infiltrate with orthokeratosis compatible with contact dermatitis. He started to be on sick leave and he was successfully treated with systemic and topical corticosteroids and oral antihistamines for 3 weeks.

Patch tests were carried out with the GEIDC standard series (http://www.aedv.es/; accessed 4 July 2007) and coal dust (1% petrolatum), becoming positive for coal dust at 96 h (+++). Coal dust was patch tested in 20 controls with negative results.

Coal tar has been described as a contact allergen in topical preparations in the treatment of psoriasis, after a long-standing skin contact (2) and only after a short time on a small skin area (3). We have found a report of contact dermatitis on hands because of newsprint ink (4) whose composition was carbon black. However, airborne occupational allergic contact dermatitis by coal dust is not frequent. There is one case in the literature (5) in a chimney sweep and in certain occupations; soot should not be considered as an irritant. The importance of anamnesis data, analysis of symptoms and signs are needed to reach a correct conclusion (6) and patch testing is invaluable for the investigation of most forms of occupational contact dermatitis (7).

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Accepted for publication 4 June 2007
Allergy 2007: 62:1346
© 2007 The Authors
Journal compilation © 2007 Blackwell Munksgaard
DOI: 10.1111/j.1398-9995.2007.01478.x

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Angioedema after administration of methylprednisolone to treat drug allergy

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Key words: corticosteroids; drug allergy.

Despite the wide use of corticosteroids (CSs), allergic reactions to these drugs

remain exceptional, their incidence being reported to be around 0.3% for systemic CSs (1). Immediate reactions to CS are often provoked by excipients (e.g. carboxymethylcellulose, bisulphites, succinate, Tween 80; 2). Clinical features include anaphylaxis, angioedema, urticaria, generalized cutaneous eruption and severe respiratory distress. Both immunological and nonimmunological mechanisms have been suggested to be involved in adverse reactions to CSs (3). On the other hand, delayed reactions are usually sustained by a T-cell-mediated response. Delayed hypersensitivity to one CS can be followed by cross-reaction with other molecules belonging to the same structural group (4). Cross-reactivity has also been observed among topical corticosteroids (Table 1), where cross-reactions occur between group A and group D2. Nonetheless, the classification of these cross-reactivities is not fully applicable to adverse reactions to systemic corticosteroids (2). Of note, CSs (in association with antihistamines) are often recommended as the first choice premedication to prevent severe drug adverse reactions (5).

When corticosteroids unexpectedly worsen the condition they are used for.

Table 1. Grouping of corticosteroids (topical) according to structure

Group	Molecule
A	Hydrocortisone acetate 1%
	Prednisone 5%
	Tixocortol pivalate 1%
B	Amcinonide 1%
	Fluocinolone acetonide 1%
	Fluocinonide 1%
	Triamcinolone acetonide 1%
	Budesonide 0.1%
C	Desamethasone phosphate 1%
	Desoximetasone 0.05%
D	Betamethasone valerate 1%
	Aclomethasone dipropionate 1%
D1	Clobetasol propionate 1%
	Betamethasone dipropionate 0.05%
	Mometasone furoate 0.1%
D2	Hydrocortisone butyrate 1%

Modified from Davis et al. *J Am Acad Dermatol* 2007;**56**:921.

We report the case of a 39-year-old woman suffering from autoimmune thyroiditis and contact sensitization to nickel sulphate, tixocortol-21-pivalate, cobalt chloride and gold thiosulphate. She also reported immediate allergic reactions to some nonsteroidal anti-inflammatory drugs (ketorolac, nimesulide, tenoxicam), and antibiotics (clarithromycine, cefatrizine, amoxicillin). All these reactions were clinically characterized by severe facial angioedema. When she had to undergo dental surgery involving the administration of local anaesthetics, a premedication with oral prednisone 50 mg (13 and 7 h before) and clorpheniramine 30 mg (1 h before) was prescribed according to international protocols. One hour after the dental intervention, the patient experienced facial angioedema that was interpreted as reaction to mepivacaine. Thus, i.v. methylprednisolone 80 mg was given, which immediately provoked worsening of angioedema with the onset of laryngeal oedema. The patient was then treated with intramuscular adrenaline and antihistamines, and the symptoms gradually disappeared. The facial angioedema completely resolved in a few days. Based on this history, the patient was studied for CS allergy. The patch tests, performed with Standard Series, confirmed the already known sensitizations. Skin tests were carried out on the volar side of the forearm with methylprednisolone succinate, betametasone phosphate and triamcinolone acetonide. All the above reagents were initially tested by the prick method, and a wheal of 3 mm or more in diameter was considered positive. When the prick test was negative, 0.02–0.05 ml of the CS solution was injected intradermally and results were considered positive if a wheal >5 mm appeared. Positive (histamine 0 mg/ml) and negative (saline) controls were also used. The patient displayed an immediate positive reaction at the intradermal test only methylprednisolone, whereas the remaining steroids proved negative. Further premedications were then successfully performed with triamcinolone acetonide and chlorpheniramine.

As CSs are poorly soluble in water, they are usually coupled with esters in C21 position to increase their solubility for intravenous use. Different esters such as succinate or phosphate are used (6), so that adverse reactions to these components cannot be excluded. In our case, the patient reacted to both prednisone and methylprednisolone. The pharmaceutical preparations of the two drugs differ only for the presence of succinate in the second one, therefore an effect of this additive can be reasonably excluded. Moreover, the excipients included in the tested preparations (i.e. benzylic alcohol, lactose, polisorbate, sodium phosphate and sulphate, carboxymethylcellulose, magnesium stearate, stearic acid, phenol) are all contained in drugs that are tolerated by the patient. Finally, prednisone (and methylprednisolone) belong to group A corticosteroids (Table 1) that include tixocortol-21-pivalate, to which the patient was sensitized. This fact makes the possibility of a cross-reaction very likely.

In conclusion, adverse reactions to drugs occurring despite an appropriate premedication with systemic CSs should be investigated for a possible allergic response to CS. The use of patch tests and skin tests may provide useful support to confirm diagnosis and identify alternative tolerated molecules.

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Accepted for publication 8 June 2007
Allergy 2007: 62:1346–1348
© 2007 The Authors
Journal compilation © 2007 Blackwell Munksgaard
DOI: 10.1111/j.1398-9995.2007.01481.x

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