

Case report

Occupational asthma in a hairdresser caused by persulphate salts

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Persulphate salts, which are common constituents of hair bleaches, have occasionally been reported to cause occupational asthma in hairdressers. We describe the clinical and immunological studies carried out in a hairdresser who developed cutaneous and respiratory symptoms, about 1 year after being employed in a hairdressing salon. Skin prick tests with 1:5 w/v potassium and sodium persulphate extracts were positive in our patient at 15 min and negative in control subjects. The European standard contactants (ECDRG) and a battery of hairdressing agents were patch tested with positive result to KATHON CG (isothiazolinone). The methacholine-inhalation test showed airway hyperresponsiveness. Bronchial provocation test with a 1:50 w/v potassium persulphate extract elicited a nonimmediate asthmatic reaction, followed by recurrent nocturnal fall in FEV₁ lasting up to 3 days after the test. Plethysmography results revealed air trapping caused by a marked increase of airway resistance 3 h after the specific bronchial challenge. Histamine release test was not conclusive, and the determinations of specific IgA, IgM, IgG and IgG subclasses by EIA and IgE by RAST against persulphate salts were negative.

Persulphate salts are widely used in hair bleach formulas because they have a strong oxidizing action that accelerates the hair-bleaching process. These salts also make the hair more receptive to dyes or toners and enable very light shades to be obtained. These highly reactive chemical compounds are also used in industrial processes (chemical, pharmaceutical, textile, photography, food preservation).

The salts are extensively used by hairdressers, who are particularly predisposed to their harmful effect. In 1963, Calnan & Shuster (3) first reported that hair bleaches containing persulphate salts can produce local urticarial reactions. Since then, several cutaneous adverse reactions have been reported, including irritant and allergic contact dermatitis and urticaria (7, 9). There are also a few reports of occupational asthma caused by these salts in hairdressers (1, 2, 7, 8, 13), but the mechanism underlying the symptoms produced by these low-molecular-weight chemicals remains unclear.

We describe the clinical and immunological studies carried out on a hairdresser who developed rhinitis and asthmatic attacks when using these products.

Case report

A 16-year-old girl, non-smoker, with a family history but no personal history of atopy or other serious illnesses, was studied at our clinic.

She had been employed in a hairdressing salon for about 1 year before she began to notice a non-productive cough, sneezing, nasal itching and hydroporrhea, which was diagnosed as recurrent sinusitis. A few months later she developed chest tightness, wheezing and dyspnea. She also developed dermatitis on her hands; this motivated the use of gloves while working. The patient associated these respiratory and cutaneous symptoms with the handling of hair bleaches and hair bleaches containing hair dyes. Respiratory symptoms began within a few hours after she left for work and became worse later in the afternoon and early in the morning. She improved during weekends and was free of symptoms during vacation periods. She required emergency treatment on three occasions due to asthma attacks.

At the time of consultation, having been absent from work for 1 month, the patient was asymptomatic and was admitted to the study. Physical exam-

ination, full blood count and chest radiograph were normal. A cytological examination of nasal smear revealed eosinophilia. Sinus radiograph detected maxillary mucosal thickening. A spirometry revealed an FEV₁ of 89%, FVC of 98% and FEV₁/FVC of 90% predicted. Total serum IgE was increased (419 IU/ml) and other serum immunoglobulins were in the normal range.

Material and methods

Potassium and sodium persulphate extracts for in vivo and in vitro tests

Extracts of potassium persulphate and sodium persulphate were prepared by mixing 2 g of the substance in 10 ml of PBS (phosphate-buffered saline) (pH 7.3) at laboratory temperature. After stirring for 30 min and passing through filter paper, the solutions were passed through a 0.22- μ m Millipore filter (Millipore Corp., Bedford, MA, USA) for sterilization. Part of each extract was also dialysed against PBS for 24 h. The four extracts obtained had a final concentration of 1:5 w/v.

Skin tests

Skin prick tests were done with the dialysed and nondialysed potassium and sodium persulphate extracts. Five normal persons and five nonexposed atopic individuals served as controls. All skin tests were examined at 15 min, 6 h and 24 h. A battery of commercially available common inhalants (Bencard, Worthing, UK) was also tested. We measured the largest wheal diameter, and considered a reaction to be positive if the wheal size induced by the allergen was ≥ 3 mm over negative control (PBS). Histamine phosphate 1 mg/ml was used as positive control.

The European standard contactants (ECDRG) (True test, Pharmacia, Uppsala, Sweden), and a battery of hairdressing agents (*p*-aminodiphenylamine 0.25%, *o*-nitro-*p*-phenylenediamine 2%, resorcinol 2%, *p*-toluenediamine 1%, hydrogen peroxide, glyceryl-monothioglycolate 2.5%, pyrogallol 1%, and potassium and sodium persulphates 2%) were patch tested, with readings at 15 min, 48 h and 96 h.

Bronchial provocation tests (BPT)

BPT with methacholine. Methacholine inhalation test was performed according to the method of Cockcroft (4), with some modifications. The aerosolized particles were generated by a continuous pressurized nebulizer, model De Villbis 646, with a fitted output of 0.28 ml/min.

Specific BPT with potassium persulphate extract. A simple blind specific BPT was performed with po-

tassium persulphate nondialysed extract. The patient was first skin tested with 10-fold dilutions of the extract in order to determine a safe starting dose for the challenge. Base-line pulmonary function measures of FEV₁ and FVC were performed, and a control challenge with PBS was carried out before antigen provocation. The patient inhaled the aerosolized allergen for 2 min at tidal volume, and pulmonary function measures of FEV₁, and FVC were performed at 5, 10, 15, 20, 25, 30, 60 and 120 min after challenge; readings were then repeated at 2-h intervals for 24 h. A positive response was defined as $\geq 20\%$ fall in FEV₁. A differential white blood cell count was performed before and after bronchial challenge.

Two nonexposed asthmatic patients with similar nonspecific reactivity were also challenged with the 1:50 w/v potassium persulphate extract, after obtaining their informed consent.

Pulmonary mechanics studies. Recordings of airway resistance and lung volumes were measured by plethysmography technique (Micro-S-200, C. Chatzmann, Spain) before challenge and 3 h later. A single breath carbon monoxide transfer test (TLCO) was performed before bronchial challenge (Alveo-Diffusionstest, Jaeger, Würzburg, Germany).

Immunological tests

Persulphate salt protein conjugates with human serum albumin (HSA) were performed according to the method of Levine & Ovary (10).

Histamine release test was performed by an automated fluorometric method according to Siraganian (15) with the modifications previously described (11), with six different 5-fold dilutions (1:5, 1:25, 1:125, 1:625, 1:3125 and 1:15625) of the extracts.

Specific IgE determination against persulphate salts was measured by modification of the RAST assay. Potassium and sodium persulphate protein conjugates were coupled to nitrocellulose paper discs (Hoeffer Scientific Instrument, UK) by a 48-h incubation period at 4°C. Results were expressed as percent binding and calculated by dividing cpm of the test sera by cpm of total added radioactivity. The RAST was performed in duplicate for each persulphate salt. The control group consisted of sera from five normal individuals. A positive result was considered to be a value higher than the mean of the control sera + 2 SD.

Specific IgM, IgA, IgG and IgG subclasses against persulphate salts by enzyme-immunoassay (EIA). For this purpose, we used the same discs described above for the RAST. The discs were incubated with 0.1 ml of the patient's serum for 24 h at 4°C, followed by washing in PBS-Tween.

For detection of specific IgM and IgA, 0.2 ml of

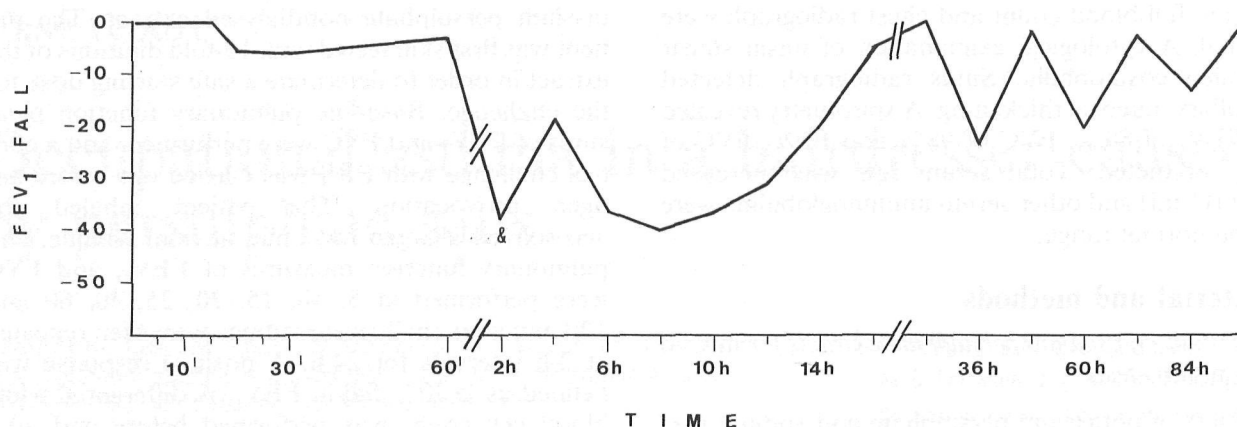


Fig. 1. Bronchial provocation test with the 1:50 w/v potassium persulphate extract. Two hours after the challenge, inhaled beta-mimetic treatment was required.

a 1:2000 dilution of peroxidase labelled antihuman-IgM and antihuman-IgA, respectively, were added to the discs and incubated for 3 h at room temperature. The same procedure was performed for detection of specific IgG or IgG₁ by the addition of a 1:4000 peroxidase labelled antihuman-IgG or IgG₁; and for detection of specific IgG₂, IgG₃ or IgG₄, by the addition of a 1:1000 peroxidase labelled antihuman-IgG₂, IgG₃ or IgG₄.

The discs were then washed in PBS-Tween, and 0.2 ml of substrate was added (*o*-phenylenediamine (2 mg/ml) in enzyme buffer (0.1 mol citrate, pH 5) containing 0.03% hydrogen peroxide), at room temperature. After 15 min the colour reaction was arrested by the addition of 4 NH₂SO₄. Optical density at 492 nm was measured with a Titertek-Multiscan (Flow Lab., Irvine, UK). The EIA was performed in duplicate for each persulphate salt and specific immunoglobulin. The control group consisted of sera from five normal individuals. A positive result was considered as a value higher than the mean of the control sera + 2 SD.

Results

Fifteen minutes after skin prick testing with the 1:5 w/v potassium and sodium persulphate dialysed extracts, the patient exhibited a 4-mm and a 3-mm wheal, respectively, and erythema response. The same response was obtained with the nondialysed extracts (PBS control negative, histamine control 4 mm). No late cutaneous responses were observed. Control subjects did not react to these extracts. The skin prick tests with a battery of common inhalants were negative.

Patch testing with the European standard of epicutaneous tests showed a positive result with KATHON CG (isothiazolinone) (+++/+++), and hairdressing agents patch tests were negative. There

was no evidence of positivity to persulphate salts at 2% in an open test, in either the immediate reading or after 48 h and 96 h.

Bronchial challenge with methacholine revealed bronchial hyperreactivity, with a PC₂₀ value of 1.33 mg/ml. Two hours after bronchial challenge with potassium persulphate extract (1:50 w/v), a 39% fall in FEV₁ was recorded (Fig. 1). At that time, the patient presented headache, shortness of breath and a nonproductive cough, and physical examination revealed widespread expiratory wheezing which required beta-adrenergic treatment. Afterwards, besides the nonimmediate asthmatic reaction, the patient began to show a recurrent nocturnal fall in airflow that only resolved 3 days after the test. White blood cell count showed leukocytosis with neutrophilia 8 h after challenge, and eosinophilia 48 h later (Fig. 2). Plethysmography results 3 h after bronchial challenge, revealed air trapping caused by a marked increase of airway resistance

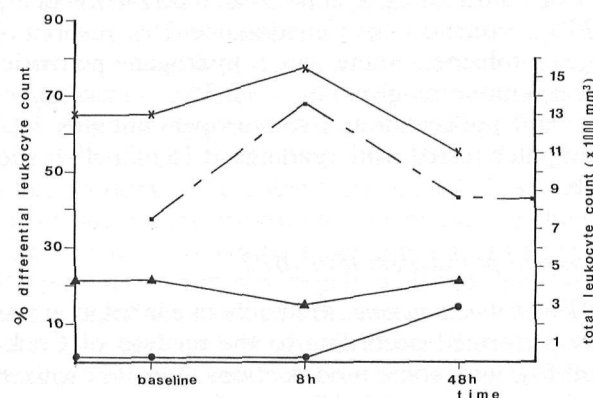


Fig. 2. Leukocyte count in peripheral blood before and after bronchial challenge with the potassium persulphate extract. Left: differential leukocyte count (neutrophils X—X, lymphocytes —●—, eosinophils —▲—). Right: total leukocyte count (— — —).

Table 1. Plethysmography results

	Base line	3 h after challenge	% change
TLC (ml)	4820	4590	-4.8
VC (ml)	3150	2000	-36.5
FRC (ml)	1150	750	-34.8
TGV (ml)	2820	3340	+18.4
RV (ml)	1670	2590	+55.1
RV/TLC %	35	56	+60.0
Raw (cm H ₂ O X s/l)	2.75	5.79	+110.5
SGaw (1/KPa X s)	0.129	0.052	-59.7

(Table 1). TLCO test before bronchial challenge was within normal range. Control subjects showed no reaction to bronchial challenge with the potassium persulphate extract.

Histamine release test was not conclusive in the patient, and the determinations of specific IgA, IgM, IgG and IgG subclass antibodies by EIA and of specific IgE antibodies by RAST against potassium and sodium persulphates were negative.

Discussion

Hairdressers are exposed to several substances which can produce respiratory and cutaneous symptoms. Some of them exert an irritant action (1, 17), while others, such as persulphate salts, may act as sensitizers and produce allergic contact dermatitis, urticaria, rhinitis and bronchial asthma.

The mechanism underlying asthma caused by these low-molecular-weight chemical compounds is unknown. Certain features suggest an allergic pathogenesis: only a proportion of exposed subjects develop respiratory symptoms; there is a latent period between first exposure and the onset of symptoms (2); and there is the finding of immediate positive skin prick tests, as previously reported (1, 2, 7). In our patient, the immediate skin prick tests with these salts were positive, but the specific bronchial provocation test elicited a late-phase asthmatic response as plethysmography results indicated. Further, a recurrent nocturnal pattern of fall in FEV₁ was observed after the specific bronchial challenge. This pattern of recurrence has been described after a severe late asthmatic response, which was associated with increased airway responsiveness (5), and also in occupational asthma caused by low-molecular-weight chemicals such as isocyanates (14) and by Western Red Cedar sawdust (5).

Obtaining a differential white cell count in peripheral blood after specific bronchial challenge might be a good approach to the airway inflammatory response, because of their close relationship with bronchoalveolar lavage results, as documented by several authors during the late-phase asthmatic reaction (6).

In this case we have not been able to demonstrate

specific IgE, nor any other specific serum immunoglobulins against persulphate salts. The best available evidence suggests that the late asthmatic response is dependent on the presence of allergen-specific IgE, although it is sometimes difficult to detect, as with occupational chemical sensitizers (12).

Bronchial response after specific challenge in our patient indicates that persulphate salts are the causative factor for the symptoms that she described, and the fact that control subjects did not react to these salts supports the specificity of this test.

The principal occupational cutaneous hazard for hairdressers is contact dermatitis, which often has an irritant basis (9). However, our patient developed an allergic contact dermatitis caused by isothiazolinone, which is commonly found as a preservative in shampoos and cosmetics.

Until skin testing reactivities and immunological assays of proved efficacy are developed for the diagnosis of persulphate salts hypersensitivity, the specific bronchial provocation test could be useful for establishing a definite diagnosis in suspected cases.

References

- BAUR X, FRUHMANN G, LIEBE VV. Persulphat-Asthma und Persulphat-Dermatitis bei zwei Industriearbeitern. *Respiration* 1979; 38: 144-50.
- BLAINEY AD, OLLIER S, CUNDELL D, SMITH RE, DAVIES RJ. Occupational asthma in a hairdressing salon. *Thorax* 1986; 41: 42-50.
- CALNAN CD, SHUSTER S. Reactions to ammonium persulphate. *Arch Dermatol* 1963; 88: 812-5.
- COCKCROFT DW, KILLIAN DN, MELLON JJ, HARGREAVE FE. Bronchial reactivity to inhaled histamine: a method and clinical survey. *Clin Allergy* 1977; 7: 233-43.
- COCKCROFT DW, HOEPPNER VH, WERNER GD. Recurrent nocturnal asthma after bronchoprovocation with Western Red Cedar sawdust: association with acute increase in non-allergic bronchial responsiveness. *Clin Allergy* 1984; 14: 61-8.
- DE MONCHY JGR, KAUFFMAN HF, VENGE P, KOETER GH, JANSEN HM, SLUITER HJ, DE VRIES K. Bronchoalveolar eosinophilia during allergen-induced late asthmatic reactions. *Am Rev Respir Dis* 1985; 131: 373-6.
- FISHER AA, DOOMS-GOOSSENS A. Persulphate hair bleach reactions. *Arch Dermatol* 1976; 112: 1407-9.
- GAMBOA PM, DE LA CUESTA CG, GARCIA BE, CASTILLO JG, OEHLING A. Late asthmatic reaction in a hairdresser, due to the inhalation of ammonium persulphate salts. *Allergol Immunopathol* 1989; 17: 2: 109-11.
- HOLNESS DL, NETHERCOTT JR. Dermatitis in hairdressers. *Dermatol Clin* 1990; 8: 1: 119-26.
- LEVINE BB, OVARY Z. Studies on the mechanism of formation of the penicillin antigen III. The *N*-(D-benzylpenicilloyl) group as an antigen determinant responsible for hypersensitivity to penicillin G. *J Exp Med* 1961; 114: 875-84.
- MONEO I, BOTELLO A, HINOJOSA M, ALCOVER R. Nuevos avances en la determinación de histamina automatizada. *Allergol Immunopathol* 1980; Suppl. 7: 311-15.
- O'BYRNE PM, DOLOVICH J, HARGREAVE FE. Late asthmatic responses. State of the art. *Am Rev Respir Dis* 1987; 136: 740-51.

13. PEPYS J, HUTCHCROFT B, BRESLIN ABX. Asthma due to inhaled chemical agents – persulphate salts and henna in hairdressers. *Clin Allergy* 1976; 6: 399–404.
14. SIRACUSA A, CURRADI F, ABBRITTI G. Recurrent nocturnal asthma due to tolylene di-isocyanate: a case report. *Clin Allergy* 1978; 8: 195–201.
15. SIRAGANIAN RP. An automated continuous flow system for the extraction and fluorometric analysis of histamine. *Anal Biochem* 1974; 57: 383–94.
16. STARR JC, YUNGINGER J, BRAHSEER GW. Immediate type asthmatic response to henna following occupational exposure in hairdressers. *Ann Allergy* 1982; 48: 98–9.
17. ZUSKIN E, BOUJUYS A. Acute airway responses to hair-spray preparations. *N Engl J Med* 1974; 290: 660–3.

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