

# Airborne irritant contact dermatitis due to synthetic fibres from an air-conditioning filter

JISKA A. PATIWAELE, MARJOLEIN WINTZEN, THOMAS RUSTEMEYER AND DERK P. BRUYNZEEL

Department of Dermato-Allergology and Occupational Dermatology, VU University Medical Center, Amsterdam, The Netherlands

We describe 8 cases of occupational airborne irritant contact dermatitis in intensive care unit (ICU) employees caused by synthetic (polypropylene and polyethylene) fibres from an air-conditioning filter. Not until a workplace investigation was conducted, was it possible to clarify the unusual sequence of events. High filter pressure in the intensive care air-conditioning system, maintained to establish an outward airflow and prevent microorganisms from entering the ward, probably caused fibres from the filter to become airborne. Upon contact with air-exposed skin, fibres subsequently provoked skin irritation. Test periods in the ICU with varying filter pressures, in an attempt to improve environmental conditions, led to even higher filter pressure levels and more complaints. The sometimes-very-low humidity might have contributed to development of skin irritation. The fact that most patients recovered quickly after treatment with emollients and changing the filters made it most likely that the airborne dermatitis was of an irritant nature.

**Key words:** airborne irritant contact dermatitis; air-conditioning; dust; hospital; mineral fibres; occupational dermatoses; polyethylene fibres; polypropylene fibres; synthetic fibres. © Blackwell Munksgaard, 2005.

*Accepted for publication 15 November 2004*

Airborne dermatoses often cause diagnostic problems and therefore not only confuse the patient, but also the doctor. Airborne irritant contact dermatitis is particularly difficult to prove because of lack of diagnostic tests. We describe 8 patients with periocular eczema. All patients were employees of the intensive care unit (ICU) at the VU University Medical Center (VUmc) and attended our clinic within a period of 3 months. Patients were treated by various physicians before visiting our Clinic for Dermato-Allergology and Occupational Dermatology. Through collaboration with the Occupational Health Service (OHS) of the hospital, a connection between these cases was suspected. We discuss the environmental conditions that most probably caused the eczematous reactions.

## Case Reports

Within a period of 3 months, from September 2001 to November 2001, 8 employees of the same unit of the ICU at the VUmc visited our Outpatients Clinic with complaints of periocular dermatitis. Initially, 2 patients (A and B) presented with these complaints. After contacting

the OHS, it turned out that 3 other patients (C, D and E) had consulted this department with comparable symptoms. Additionally, 3 more patients reported similar skin lesions to the OHS, but no further medical information about these patients was on file.

Patient A, a 36-year-old woman without a history of atopy, presented with periocular dermatitis for 6 months. She was working as a physiotherapist in the ICU. During working days, she noted exacerbation of these complaints. Her dermatologist prescribed prednisolone eye cream, which gave only temporary relief. She was therefore referred to our department. As an allergic contact dermatitis from her eye cosmetics was suspected, she was patch tested with the European standard series, an additional standard series, a hospital disinfectant and her own cosmetics. As no contact allergies were identified at day 2 (D2), D3 and D7, and prick tests with natural rubber latex were negative, she was diagnosed with seborrheic dermatitis and treated accordingly with ketoconazole cream. However, this treatment did not result in the expected improvement.

Patient B, a 25-year-old woman, also employed as a physiotherapist in the ICU, presented with

itchy periocular dermatitis. These symptoms had persisted for 6 months and worsened on working days. Aside from a personal history of atopic dermatitis, she developed an itchy feeling when using particular shower products. Patch tests with the European standard series, an additional standard series and her own cosmetics showed a positive (+) reaction to Myroxylon pereirae (balsam of Peru), at D3 and D7. Prick tests with natural rubber latex were negative. She was therefore diagnosed as having an allergic contact dermatitis from balsam of Peru, which could be relevant as she practised aromatherapy at home. Hence, she was advised on which products to avoid. Nonetheless, the dermatitis persisted.

She noticed that there were more employees of the ICU with similar complaints. Therefore, the OHS was consulted and this resulted in 6 more cases, of which 3 were investigated.

Patient C, a 54-year-old non-atopic ICU nurse, was referred by the OHS, with a 1-month history of work-related periocular pruritic and papular erythema. Patch tests with the European and an additional standard series, hospital disinfectants and her cosmetics were performed, but no positive reactions were detected at D2, D3 and D7. Prick tests with natural rubber latex were negative. She was diagnosed with periocular dermatitis, probably of irritant nature and consequently treated with emollients. Thereafter, the dermatitis partially improved.

Patient D, a 27-year-old woman, also referred by the OHS, had complaints of red, itchy eyes for 1 month. Although there was a personal history of allergic rhinoconjunctivitis, this time, symptoms were accompanied by erythema and oedema of her eyelids. She was working as a nurse in different hospital wards, including the ICU. Eye symptoms worsened while working in the ICU, but not during her work in other departments. Patch tests with the European and an additional standard series, disinfectants and her own cosmetics showed only a non-relevant reaction for thimerosal at D2, D3 and D7. Prick tests with natural rubber latex were negative. An irritant contact dermatitis was considered most likely, and the patient was prescribed an emollient cream. Skin lesions partially subsided after regularly using this cream.

Patient E, a 55-year-old woman with no history of atopy, was referred by the OHS as well. For 6 months, she had experienced periocular itchy erythema spreading to neck, cheeks and ears. The patient was a member of the administrative staff of the ICU; her activities were partly carried out in the ICU and partly outside. Symptoms increased only on days when working in the IC

department itself. Topical treatment with corticosteroids prescribed by her family doctor resulted in temporary improvement. Notably, complaints disappeared after thorough cleaning of the ICU, but gradually returned over the following months. Extensive patch testing with relevant series and materials proved negative. An irritant contact dermatitis was diagnosed and she was treated with emollients, which resulted in partial clearance of the lesions.

None of the patients we saw showed a positive demography.

Considering that the development of symptoms seemed work-related and patients were employees of the same ICU, an occupational dermatitis was presumed in all cases. Because of facial distribution, an airborne component was suspected. A workplace investigation in the ICU was therefore conducted at request of the OHS.

#### *Workplace investigation*

Initially, the investigation of the ICU revealed nothing extraordinary. However, closer examination disclosed unusual white dust that appeared different from common grey dust. Remarkably, this unusual dust was mainly found on surfaces close to the ceiling such as lamps, curtain rails and closets. Large quantities of this white dust on the air-conditioning ventilation grids pointed towards the source of this dust. Microscopic examination of the dust showed fibrous, partially splitting material of various lengths (Figs 1 and 2). This suggested that fibres were blown into the ICU rooms by the air-conditioning system, where these fibres settled as white dust.

The supposition that skin lesions were air-conditioning related was further supported by the fact that complaints of the ICU employees started after changes had been made to the

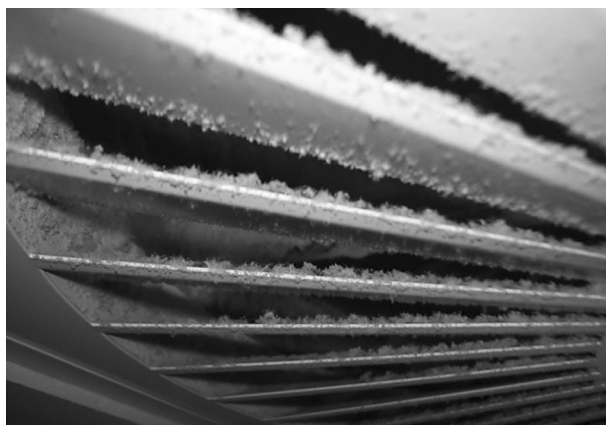


Fig. 1. Large quantities of white dust on an air-conditioning ventilation grid in the intensive care unit.

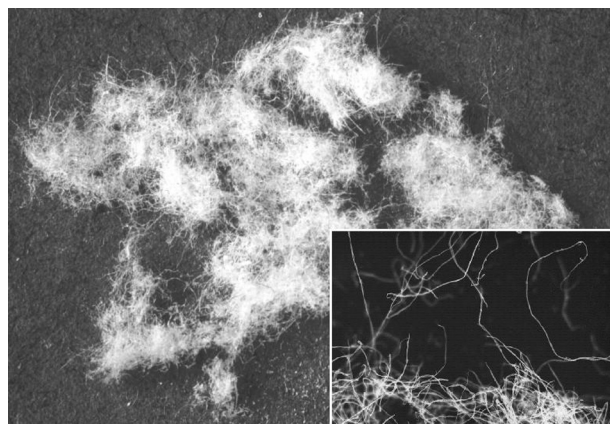


Fig. 2. Microscopic photograph of the polypropylene and polyethylene fibres.

air-conditioning system. Filters in parts of the hospital air-conditioning system, providing multiple hospital departments including the ICU, had been replaced.

A few months afterwards, experimental tests with varying, higher filter pressures were carried out only in the ICU, in order to improve its environmental conditions. As a result of these test periods, temperature and relative humidity also fluctuated: temperature mainly varied from 20° celsius to 25° celsius and the relative humidity mostly oscillated between 30% and 50%, fluctuations comparable to the situation in the other ICUs. First facial symptoms developed 1 month after the experiments had started. Moreover, skin lesions deteriorated 7 months later after vacuuming the ventilation grids, allowing more loose fibres already in the system to become airborne in the room. Unfortunately, it was impossible to detect fibres on the facial skin of the ICU employees, by means of tape stripping.

The new air-conditioning filters were analysed and shown to have the same structure as the unusual white dust in the ICU. Filters consisted of synthetic fibres composed of polypropylene (PP) and polyethylene (PE). No information was provided by the manufacturers about diameter, length and possible coating of these fibres. Although filters that had been replaced supplied several departments, only employees of the ICU developed complaints. This was essentially attributed to higher filter pressure in the ICU maintained to establish an outward airflow and prevent microorganisms from entering the ward. Probably due to this high filter pressure, and especially due to aforementioned test periods with even higher filter pressure levels, fibres became airborne and were preferentially transported to the ICU along with the high-pressure flow. Upon contact with air-exposed skin, fibres

subsequently provoked skin irritation when rubbing the face. The sometimes-very-low humidity, caused by these experiments, might have contributed to the development of this irritant dermatitis.

Irritant contact dermatitis was considered more likely than an allergic reaction, as fibres are known to induce a similar clinical picture of irritant dermatitis. Patch testing of 1 patient (E) with the fibrous material showed no allergic reactions at D2, D3 and D7. Patients were hence diagnosed as having an airborne irritant contact dermatitis due to PP and PE fibres from the air-conditioning filter. After replacement of filters, the skin complaints resolved completely. Only patient B had a recurrence of her complaints, but this was considered to be of another origin.

To resolve the problem, the filters were replaced by the original ones.

### Discussion

In hospital-related eczema, airborne contact dermatitis should be considered. Possible airborne agents, such as propacetamol and various antibiotics, for example cefazolin, have been reported to cause contact dermatitis in health workers in hospital environments (1, 2). In our cases, PP and PE fibres from an air-conditioning filter were suspected to be the causative agents of airborne irritant contact dermatitis in ICU employees with periocular dermatitis. There were no complaints in other exposed areas like the neck and the arms. A reason for this could be that the facial skin is more vulnerable.

The air-conditioning was an unexpected source of skin problems in aforementioned patients. This system provided several hospital departments, whereas skin lesions developed only in ICU employees. The ICU was the only ward with high air-conditioning filter pressure, and on top of this, experimental tests with the filter pressure were carried out. This high pressure caused release of fibres from the filter into the ICU room itself. Usually, skin irritation due to air-conditioning is caused by low environmental humidity (3). In our cases, however, the high filter pressure in the ICU was especially the cause of skin problems: low humidity led to dry skin and high filter pressure caused accidental circulation of synthetic fibres.

PP and PE synthetic fibres are an infrequent source of airborne irritant contact dermatitis. To our knowledge, there is only one report of airborne irritant dermatitis caused by PP fibres and no reports of irritant dermatitis from PE fibres (4). Synthetic fibres can be divided into mineral and non-mineral fibres, such as PE/PP fibres.

Synthetic fibres that are known causes of irritant dermatitis are synthetic mineral fibres and include glass filaments, mineral wool (glass wool, rock wool and slag wool), refractory ceramic fibres and fibres for special purposes (5–7). These mineral fibres are a well-known and common source of work-related skin irritation, though not often reported. According to the Finnish Register of Occupational Diseases, irritant contact dermatitis due to synthetic mineral fibres was diagnosed in only 0.05% of all occupational dermatoses in Finland over a period of 10 years (8). Fibreglass, a classic example of a mineral fibre, comes in 2 main types, i.e. wool and textile fibreglass. The former is used in thermal and acoustic insulation, whereas the latter is used for reinforcement of plastic and rubber. Fibreglass is likely to cause skin irritation when the diameter is more than 4.5 µm (9, 10). Unfortunately, this information on fibres from the filters used in our hospital is lacking.

Upon contact with the skin, external irritants such as fibres, especially when rubbed into the skin, can bring on skin barrier impairment and induction of an inflammatory signal, resulting in skin irritation and leading to clinical dermatitis (11). Unfortunately, there is no diagnostic test for irritant contact dermatitis, and allergic reactions have to be excluded first by patch testing. Importantly, the fact that most patients recovered quickly after treatment with emollients and changing back to the original filters made it most likely that the airborne contact dermatitis was of irritant nature. Probably due to high filter pressure in the air-conditioning system, PP and PE fibres from the filter became airborne agents in the ICU and caused an airborne irritant dermatitis in ICU employees.

## References

1. Mathelier-Fusade P, Mansouri S, Aïssaoui M, Chabane M H, Aractingi S, Leynadier F. Airborne contact dermatitis from propacetamol. *Contact Dermatitis* 1997; 36: 267–268.
2. Straube M D, Freitag M, Altmeyer P, Szliska C. Occupational airborne contact dermatitis from cefazolin. *Contact Dermatitis* 2000; 42: 44–45.
3. Morris-Jones R, Robertson S J, Ross J S, White I R, McFadden J P, Rycroft R J. Dermatitis caused by physical irritants. *Br J Dermatol* 2002; 147: 270–275.
4. Lachapelle J M. Recent data on airborne contact dermatitis. *J Eur Acad Dermatol Venereol* 1997; 9: S39.
5. Lachapelle J M. Industrial airborne irritant or allergic contact dermatitis. *Contact Dermatitis* 1986; 14: 137–145.
6. Angelini G, Vena G A. Airborne contact dermatitis. *Clin Dermatol* 1992; 10: 123–131.
7. Bjorkner B E. Industrial airborne dermatoses. *Dermatol Clin* 1994; 12: 501–509.
8. Jolanki R, Makinen I, Suuronen K, Alanko K, Estlander T. Occupational irritant contact dermatitis from synthetic mineral fibres according to Finnish statistics. *Contact Dermatitis* 2002; 47: 329–333.
9. Sertoli A, Francalanci S, Giorgini S. Fiberglass dermatitis. In: *Handbook of Occupational Dermatology*, Kanerva L, Elsner P, Whalberg J E, Maibach H I (eds), Berlin, Springer, 2000: 122–134.
10. Konzen J L. Fiberglass and the skin. In: *Occupational and Industrial Dermatology*, 2nd edition, Maibach, H I (ed.), Chicago: Yearbook Medical Publishers, 1987: 283–285.
11. Lisby S, Baadsgaard O. Mechanisms of irritant contact dermatitis. In: *Textbook of Contact Dermatitis*, 3rd edition, Rycroft R J G, Menné T, Frosch P J, Lepoittevin J-P (eds), Berlin, Springer, 2001.

## Address:

Derk P. Bruynzeel  
 Department of Dermato-Allergology and  
 Occupational Dermatology  
 VU University Medical Center  
 PO Box 7057  
 1007 MB Amsterdam, The Netherlands  
 Tel: +31 20 4440145  
 Fax: +31 20 4440148  
 e-mail: dp.bruynzeel@vumc.nl