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Contact dermatitis to chlorprothixene

WE report the case of a 24-year-old nurse who complained of acute, intermittent skin reactions appearing periorbitally and on the cheek and hands. She was first seen in 1992, 1 year after starting work on a psychiatric ward and 6 months after the first symptoms. The symptoms improved during weekends and holidays. The previous year, the ward had been redecorated and so she was initially thought to have contact allergy to the paints used and their ingredients. She had no history of atopic disease. When the patient was first seen, she had already undergone treatment with local corticosteroids. We could see only folliculitis of the face, but the history was typical of airborne contact dermatitis.

A skin prick test panel which included the major airborne allergens and the ingredients of the paints used to redecorate the ward, and also patch testing with the European standard, the drug, and the disinfectant and preservative battery (all Fa.Hermal, Reinbeck, Germany) showed no positive skin reaction except to paraphenyldiamine, a reaction which was not clinically relevant. A high total IgE level with 1250 U/ml was shown without any history of airborne type I allergy or evidence of specific IgE.

Scratch test with neuroleptics belonging to the phenothiazines, the thioxanthenes, and the chemically unrelated butyrophenones, which had all been handled by the patient, showed no acute reactions,

Occupational allergy to a neuroleptic drug in a nurse working on a psychiatric ward.

but a strong reaction to chlorprothixene occurred after 24 h with additional reactions in the face and the neck, lasting 3 days. Patch testing was conducted with all the drugs the nurse handled (phenothiazines, thioxanthenes, butyrophenones, quinine sulfate, clonidine, and calcium dobesilate). No positive skin reaction was observed. Chlorprothixene was tested as syrup and coated tablets. Both showed strong reactions after 24 h, lasting 3 days. Control subjects did not show dermatitis after testing with chlorprothixene.

The nurse, after moving to another ward on which chlorprothixene was not used, was shown to be free of symptoms. After handling chlorprothixene again by mistake, she revealed skin reactions after a few minutes.

On the basis of clinical history and patch testing, the diagnosis of contact allergy due to occupational contact with chlorprothixene syrup was made.

Chlorprothixene belongs to the thioxanthenes, which are tricyclic antipsychotic drugs chemically related to the phenothiazines. This kind of neuroleptic has sedative, anxiolytic, antidepressant, antiemetic, antihistaminic, and anticholinergic effects. After widespread use of these antipsychotic agents in the mid-1950s, there were reports of frequent side-effects in patients, not only of the anticholinergic type but also of skin reactions (1, 2). Some authors estimate that skin reactions to psychotropic drugs are twice as frequent as those caused by most other drugs (3).

In particular, Chlorpromazine caused allergic dermatitis and even phototoxic and photoallergic skin reactions (1, 2, 4). In addition, eczematous reactions of the contact type were described, especially in health-care workers (1).

We report on a nurse who revealed contact dermatitis after 6 months of handling chlorprothixene, especially chlorprothixene syrup. She showed not only skin reaction at the contact site but also airborne contact allergy on the face and neck. To our knowledge, this has only once been previously reported with chlorprothixene (5). Cross-reactions with phenothiazines or group reactions with other thioxanthenes could not be shown in our patient, and there was also no reaction to the butyrophenones used. There was no indication of photosensitive dermatitis. After the cause of the patient's

dermatologic problem had been determined, she was employed on another ward, where she was no longer exposed to chlorprothixene. Accidentally re-exposed to chlorprothixene, she had a flare-up reaction. The cause of the repeated detection of an elevated total IgE level remained unclear. The sensitivity to *p*-phenyldiamine had no clinical relevance.

In conclusion, the well-known contact dermatitis due to neuroleptic drugs can occur not only in phenothiazines, particularly chlorpromazine, but also in thioxanthenes. As reports about this side-effect are less frequent than in the 1950s, it may be forgotten or thought to be unimportant, but the withdrawal of chlorpromazine merely reduced the problem and did not solve it. Therefore, health-care workers, allergists, and physicians specializing in occupational diseases should be aware of the possibility of contact allergies.

Key words: airborne contact dermatitis; chlorprothixene; neuroleptics; occupational allergy.

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Food allergy to concealed sunflower pollen

FOOD allergy reactions develop in some patients with pollinosis when they consume certain foods (1). It is well documented that such patients frequently show allergic symptoms of the immediate type after exposure to concealed food antigens.

In some cases of "food" allergy, the responses may result from far-related antigens. We report a case of systemic allergic reaction to sunflower pollen concealed in a commercial product of peeled sunflower (*Helianthus annuus* L.) seeds.

A 22-year-old woman was referred to our allergy clinic for a systemic allergic reaction after the ingestion of a commercially prepared package of peeled sunflower seeds. She was otherwise a healthy woman with insignificant medical history, and she had never had any reaction to ingestion of sunflower seeds when peeled by herself. She consumed such self-peeled seeds very often. A few minutes after first ingestion of the commercial product, she developed systemic allergic reactions comprising rhinitis, nasal congestion, tearing, and facial and generalized urticaria. The symptoms resolved spontaneously within a few hours. Skin prick tests revealed positive wheal and flare reaction to sunflower pollen and house-dust mites. Measurement of specific IgE concentrations in her serum by the Alastat diagnostic method (Alastat, Diagnostic Products Corporation, Los Angeles, CA, USA) showed elevated concentrations of IgE against pollen antigens of the Compositae family that were available for testing: mugwort (*Artemisia vulgaris*), oxeye-daisy (*Chrysanthemum leucanthemum*), and dandelion (*Taraxacum* species) (12.47, 10/78, and 4.5 IU/ml, respectively). The levels of specific IgE were elevated also against house-dust mites (*Dermatophagoides pteronyssinus* and *D. farinae*, 3.21 IU/ml and 5.63 IU/ml, respectively), cat epithelium (0.86 IU/ml), olive pollen (2.17 IU/ml), and eucalyptus (0.35 IU/ml). Negative results were obtained for several other pollen and food antigens. Since the patient did not suffer from any allergic symptoms before her recent exposure to sunflower seeds, no clinical relevance could be attributed to these latter results. Open food challenge with the exact commercial preparation of peeled sunflower seeds in minute quantity caused rhinitis and nasal congestion, which were treated promptly and resolved. Analysis of the commercial package of peeled seeds showed a high level of contamination by sunflower pollen. Such pollen had probably derived from a large number of unripe flower heads that had been mixed during harvest with the ripe ones.

Taken together, the medical history, skin tests, specific IgE, food challenge, and pollen analysis demonstrated IgE-mediated sensitization to sunflower pollen that had been concealed in the commercial product.

Sensitization to inhalant pollen allergens was found with significantly higher frequency in patients with sensitization to fruits, legumes, other

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