Air-borne dermatitis from *Chrysanthemum* – case report with a discussion of diagnostic procedures and therapy

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**Abstract**

Airborne dermatitis belongs to a heterogeneous group of dermatoses of various etiopathology and clinical characteristics. This disease is characterized by acute or chronic inflammation of the uncovered skin exposed to irritants or allergens. Initially skin lesions are transient. The paper presents a description of chrysanthemum growers diagnosed with air-borne dermatitis from chrysanthemum. Etiology, pathomechanism, clinical course, diagnostics and therapeutical methods are described.

**Key words**

air-borne contact dermatitis, lactones, patch tests

**INTRODUCTION**

Airborne dermatitis is a heterogenous group of dermatoses of various clinical characteristics and etiopathology [1, 2]. It characterizes with acute or chronic inflammation of uncovered skin exposed to various airborne substances [3, 4, 5]. Although the epidemiology of airborne contact dermatitis is difficult to estimate, according to the literature, the incidence estimated in the European population is 0.9–5.9% [6–8]. The most important sensitizers are lactones of the Composite family, present in the oleoresin fraction of leaves, flowers, and probably pollen [9, 10, 11]. The first report of Chrysanthemum dermatitis was described in 1887 [12, 13].

At the beginning, the disease is seasonal, usually starting in summer and ending in autumn [10, 14]. In the classical form, airborne contact dermatitis, the rash involves the skin exposed to UV, e.g., face, neck, forearms and hands [2]. Initially, the skin lesions are transient and active only during the plant growing season because pollen grains may act as allergens – inducing IgE mediated reactions, or as haptens provoking delayed T lymphocytes dependent reactions, or as irritants. Repeated, long-term exposures to them may lead to prolonged and chronic disseminated skin lesions [2, 10].

**CASE REPORT**

The patient was a 55-year-old male, a grower and salesman of chrysanthemums, generally healthy, with negative familiar and personal history to atopy. The first lesions, a erythemat-oedematous rash localized on the face, neck, trunk and forearms, first appeared about 17 years ago. Initially, they were in a transient form and disappear during a period of isolation from his job. Later, they become persistent. According to the interview, the isolation of the patient from growing chrysanthemum led after a few years to complete remission of the disease.

On admission to hospital, diffuse erythemat-squamous lesions with lichenification were observed, mainly on the dorsal part of the hands and forearms (Photo 1). During hospitalization, patch tests with European Standard Allergens (Trolab, Hermal), prick skin tests with aeroallergens (Allergopharma), photo tests and patch tests with leaves of chrysanthemum were performed.

**RESULTS**

Positive patch test with lactones, positive patch test with peru of balm and fragrance mix were detected. Delayed strongly positive infiltrated erythematous – a popular contact...
The above data indicate that not only Th1 lymphocytes, e.g., TNF-alfa, INF-gamma, as well as Th2, play a significant role in the immunopathogenesis of airborne contact dermatitis [5, 25]. Persistence of skin lesions with seasonal aggravation may inform about cross-reactivity reactions in the group of terpenes.

The treatment of airborne contact dermatitis is difficult. For effective control of the dermatitis, it is necessary to detect and reduce exposure to the causal allergens or irritants. In the beginning, the patient was treated many times by the local steroid creams dexaportal spray and Advantan (0.1% methylprednisolone aceponate emulsion), moisturizing cream and ceterizine in a dose of 10 mg, twice a day. Because of the considerable tightening of the skin lesions the patient was hospitalized. He received methylprednisolon iv. in a dose 0.5 mg/kg and Ceterizine a 10 mg twice a day. Local treatment was continued. Improvement of skin lesions appeared after 2–3 days. The patient was discharged in a well state of health after 10 days hospitalisation. The administration of methylprednisolone was stopped but Ceterizine was continued for a few more weeks.

In severe cases, in which more than 25% of the body surface is affected, or when the exposure to the sensitizer is longer than few weeks, PUVA or UVB-311 therapy may be considered [2, 26].

REFERENCES

17. Spiewak R. The substantial differences between photoallergic and phototoxic reactions