ORIGINAL ARTICLE

Annals of Agricultural and Environmental Medicine 2014, Vol 21, No 3, 531–533
www.aaem.pl

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

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Obtulowicz A, Antoszczyk G, Wojas-Pelc A. Air-borne dermatitis from *Chrysanthemum* – case report with a discussion of diagnostic procedures and therapy. Ann Agric Environ Med. 2014; 21(3): 531–533. doi: 10.5604/12321966.1120596

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 erythemato-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.

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Received: 24 October 2012; accepted: 14 May 2013

On admission to hospital, diffuse erythemato-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.

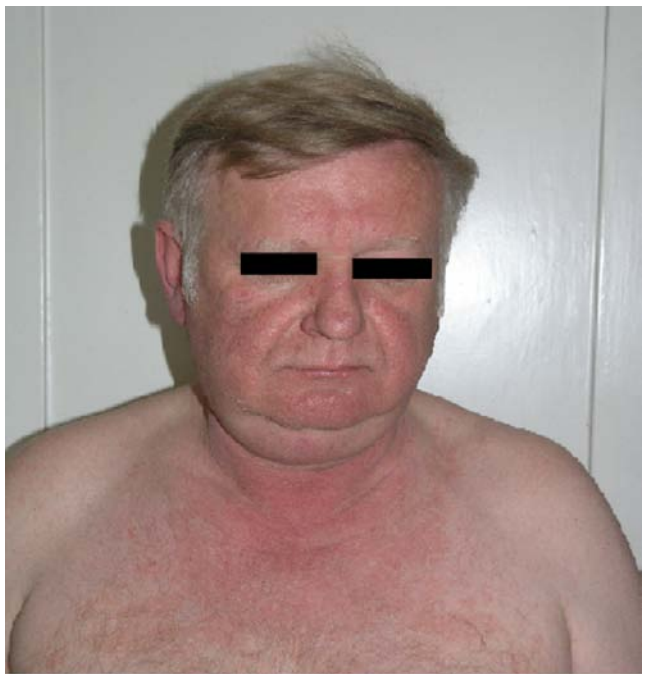

Photo 1. Erythematous maculopopular eruption on the face and upper part of the chest. Skin lesion remains active, although last exposure to chrysanthemum was noted 10 years ago

RESULTS

Positive patch test with lactones, positive patch test with peru of balm and fragrance mix were detected. Delayed strongly positive infiltrated erythemato – a popular contact

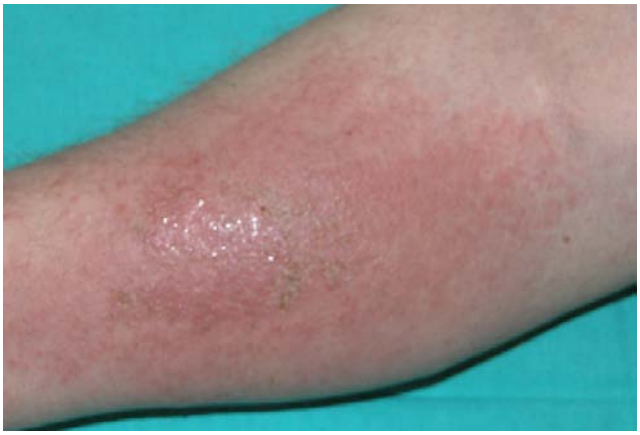

Photo 2. Positive rub test 24 hrs after an application of a chrysanthemum leaf on forearm of the patient

test with the leaf of chrysanthemum was noted (Photo. 2). During the testing, aggravation of preexisting lesions on the face, chest and hands, as a result of secondary allergization, were observed. Results of basal biochemical tests and total IgE serum level were normal. Prick tests with aeroallergens and photo test were negative.

DISCUSSION

Air-borne dermatitis, known as Composite dermatitis, is a disease with characteristic erythemato-squamous and popular lesions initially localized on uncovered skin [5, 13, 15]. Currently, five different types of airborne dermatitis can be distinguished: airborne irritant contact dermatitis, airborne allergic contact dermatitis, airborne phototoxic reactions, airborne photoallergic reactions and airborne contact urticaria [2, 16, 17]. The diagnosis is based on present acute or chronic dermatoses predominantly of exposed parts of body. It can be induced by occupational or non-occupational plant substances which are released into the air and affect exposed skin [2,17, 18, 19]. Patch tests are used to find the causative sensitizer. The most important sensitizers responsible for airborne contact dermatitis are lactones ring combined with a sesquiterpen. They are lipophilic and are present mainly in the oleoresin fraction of the plants [3,20, 21, 22]. Sesquiterpen lactones represent the main plant allergens of the Composite family [21]. According to the literature, in Europe the most common causes of this disease, chrysanthemum potential sensitizers were found in fractions of monoterpens isolated from *Tanacetum partenium* (in 88% of all received airborne substances) [12, 13]. They do not confirm the theory of a unique role of lactones in the pathogenesis of composite dermatitis, even though their importance in this disease is still under consideration [9, 23, 24]. Because of the variability of clinical presentation, frequent co-existence of atopic dermatitis and contact allergy, the most important in diagnosis seem to be tests with lactones and their extracts [10].

The positive patch test and lack of allergospecific IgE are proof of a delayed form of allergy. Studies performed on animals showed increased levels of pro-inflammatory cytokines typical for Th1 lymphocytes: IL 6,8,17 and tumour necrosis factor (TNF), IFN–gamma, and decreased levels of anti-inflammatory cytokines, such as IL-4 and IL-10. The above data indicate that not only Th1 lymphocyte, 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 terpens.

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 dexapolcort spray and Advantan (0.1% methylprednisolone aceponate emulsio), 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].

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