An Overview of Airborne Contact Dermatitis

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Abstract

Airborne-contact dermatitis (ABCD) is a unique form of contact dermatitis originating from dust, sprays, pollens or volatile chemicals by airborne fumes or particles without directly touching the allergen. ABCD has been reported worldwide due to various types of plant and non-plant allergens, most commonly secondary to plant antigens especially to compositae family. This review focuses on common airborne contactants, clinical manifestations, diagnosis and therapeutic issues in However, in recent years increased reports of ABCD due to non-plant sources especially in developing countries are seen airborne contact dermatitis.

Key Words: Airborne, Allergen, Antigen

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INTRODUCTION

Airborne contact dermatitis is an acute or chronic dermatoses predominantly affecting exposed parts of the body and caused by allergens/irritants present in the atmosphere. Allergens can be present in the form of dust, sprays, pollen, volatile chemicals by airborne fumes or droplets, which settle on the exposed skin of the body.¹ Airborne dermatitis commonly affects face, neck, ‘V’ area of chest, eyelids, axillae and forearms. This form of dermatitis can also involve non-exposed skin like major body folds and sometimes may be generalized in distribution.², ³, ⁴ Airborne contact dermatitis can be of both plant and non-plant origin. Most common airborne dermatitis is due to compositae plant Parthenium hysterophorus. However cases of non-plant and industrial origin are on increasing trend especially in the developing countries.², ⁵ The diagnosis can be made on the basis of the history and follow up of the patient, existence of the dust, droplets or volatile substances in the environment, the distribution & morphology of the lesions and the results of the epicutaneous tests.⁶ The incidence of the airborne contact dermatitis is rising considerably in recent years.⁷ In this article, an overview of the nature of airborne contactants, clinical manifestations of airborne dermatitis, diagnosis, differential diagnosis and various preventive and treatment modalities will be provided.

NATURE OF AIRBORNE CONTACTANTS

Airborne dermatitis can be due to occupational as well as non-occupational, plant as well as non-plant airborne allergens and irritants. The harmful agents can enter the environment in many different ways: vapors, droplets or solid particles. The most common allergens and irritants causing airborne dermatitis has been listed in Table 1. Causes of airborne contact dermatitis (modified from Santos et al ⁶, Huygens et al ⁷ and Handa et al³) ABCD may be caused by: a) pollens or dust containing particles from plants such as Parthenium
hysterophorus, ragweed or certain types of woods or medicaments by the process of delayed hypersensitivity. b) fibrous materials like grain dust, glass fiber and rock wool causing mechanical dermatitis c) aerosols of mineral oils inducing irritant reaction d) cement and wood dust, causing irritant as well as sensitization reactions.  The most important allergens in *Parthenium hysterophorus* responsible for causing allergic contact dermatitis are Sesquiterpene lactones (SQL). They are lipophilic, and are present mainly in the oleoresin fraction of the plant. Among the SQL’s, Parthenin is the major allergen. Parthenin belongs to pseudoguinolide class of SQL’s and has an alpha methylene group exocyclic to gamma lactone, which is probably essential for the induction of allergy. Apart from parthenin, other important allergens are coronopilin, hymenin, tetraneurin A etc. The other components, namely, thiopenes, monoterpenes and acetylenes, are known to cause phytotphotodermatitis. These SLs are found in other compositae plants such as genera, namely, liverwort (*Frullania*), tulip tree (*Liriodendron Magnoliacea*) and sweetbay (*Lauraceae, Laurus nobilis*), which may show cross sensitivity with parthenin and vice versa.

**CLINICAL MANIFESTATIONS**

Sensitization of a person to airborne contactants can occur by direct or indirect contact, ingestion of

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<th>Table. 1 Allergic airborne contact dermatitis</th>
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<td>Plants, natural resins, vegetable and wood allergens</td>
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<tr>
<td>Ambrosia deltoidea</td>
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<td>Acacia melanoxylon</td>
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<td>Cedar pollen7</td>
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<td>Citrus fruits</td>
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<td>Parthenium hysterophorus</td>
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<tr>
<td>Soybean</td>
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<td>Tropical and domestic woods</td>
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<th>Non-allergic airborne contact dermatitis</th>
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<td>Irritant contact dermatitis</td>
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<td>Phosphates26</td>
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<td>Synthetic fibers</td>
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<tr>
<td>Chlorothalonil</td>
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<td>Mustard gas</td>
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<td>Metal dust</td>
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<td>Carbon fiber</td>
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<td>Ethylene oxide</td>
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allergens in herbal teas or exposure to herbal cosmetics. According to a classification by Dooms- Goossens, airborne contact dermatitis can be divided into five different types.

1. Airborne irritant contact dermatitis
2. Airborne allergic contact dermatitis
3. Airborne phototoxic reactions
4. Airborne photoallergic reactions
5. Airborne contact urticaria

Other rare airborne skin reactions include acne like, exfoliative dermatitis, lichenoid eruptions, fixed drug eruptions, hyper- and depigmentation, telangiectases, paresthesias, purpura, erythema multiforme like eruption, pellagra like dermatitis and lymphomatomatoid contact dermatitis. One particular product can cause more than one type of reaction like *P. hysterophorus* can produce allergic contact dermatitis, photocontact dermatitis and a lichenoid eruption. Similarly, formaldehyde and phosphorus sesquisulfide can lead to an airborne irritant or allergic CD and contact urticaria. Sometimes one dermatitis may mask another one, for example, in case of rosacea and air-borne dermatitis in a farmer. Classical airborne contact dermatitis presents as involvement of exposed areas of the face, nasolabial folds ‘V’ of neck, hands and forearms. The skin symptoms can also occur on those parts of the body not exposed to the air. Volatile substances can be captured in the clothing, and dust particles can accumulate on occluded sites such as the genital area and particularly in the major body folds. This can be seen when wood sawdust is deposited on the clothes of sensitized subjects, and, together with sweat retention, causes dermatitis in occluded area. Occasionally, though rarely there can be generalized involvement with the picture of an erythoderma, for example, erythoderma due to compositae dermatitis, mercury exanthema (generalization of the dermatitis caused by volatile substances such as mercury vapors). In addition to airborne factor, penetration through clothing and inhalation may play role in generalization.

Severity of the ABCD can be assessed by a Clinical Severity Score (CSS) put forward by Verma *et al* which takes into consideration both subjective features like pruritus and objective features like the morphology of the lesions and the area involved. ABCD can also be subclassified as plant or non-plant origin. Plants and wood are a rich source of airborne allergens and irritants. The most commonly seen plant originated dermatitis is Parthenium dermatitis. Recently, cases of ABCD of non-plant origin have been reported from urban and semiarboreal areas of developing countries. Organic compounds that are use or generated anthropogenically in large quantities in the cities can be source of human contact allergens. Non plant allergens include potassium dichromate, epoxy resins, colophony, formaldehyde, perfumes/deodorants, volatile paints etc. Ghosh *et al* studied 64 patients and the pattern of allergens contributory to ABCD detected in the study was as follows: potassium dichromate 39.7% (*n*=25), fragrance mix 28.1% (*n*=18), epoxy resin 26.6% (*n*=17), colophony 17.8% (*n*=12), formaldehyde 13.2% (*n*=7) and parthenium 9.4% (*n*=6).

Cement, perfumes or deodorants, volatile paints and synthetic glues have become commonest allergens contributing to ABCD in urban and semiarboreal areas. Contact dermatitis due to cement dust usually presents as a dry, lichenified dermatitis rather than oozey even in cases of allergic cases of contact dermatitis to chromium or cobalt content in cement. Dermatitis due to vapors is mainly occupational in origin.

**DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS**

Airborne allergens or irritants are suggested if the symptomatic complaints occur on particular parts of the body especially the face, and if the symptoms clear with the change of environment by the patient. The allergen in the environment may sometimes be isolated by means of chemical analysis or direct microscopic studies of the air or the materials in the air whereupon, if the reaction is allergic, an allergen may be demonstrated by patch testing. This is, of course, more difficult for airborne irritants. Light tests and photopatch tests can help in exclusion of a light factor in the pathogenesis of the lesions. Differentiating an airborne dermatitis from a photodermatitis may sometimes pose problems. However, involvement of covered parts of the body such as major body folds, the genital region, lower leg, as material may be trapped under clothing and anatomically shadowed portions of the body like both eyelids, “Wilkinson's triangle,” the scalp that is covered by hair and area under the chin suggest airborne contact dermatitis. Upper eyelids are particularly susceptible to airborne allergens and irritants because they can readily collect there, and sometimes may be the only sites affected. Other
differential diagnosis of facial airborne irritant or allergic contact dermatitis include dermatitis caused by directly applied agents, dermatitis caused by occasional contacts with an allergen or an allergen-contaminated surface (e.g., a pillow), connubial or consort dermatitis, an id type spread of a dermatitis elsewhere on the body, systemic eczematous contact type dermatitis reactions limited to the face (e.g., due to thiomersal in an injection), and photoinduced reactions.\(^8\) Another important cause of irritant and particularly allergic contact dermatitis of the face is the transfer of allergenic particles by other parts of the body, as occurs with nail polish. This is the classic example of an "ectopic dermatitis," a term coined by Fisher to indicate a dermatitis displaced from its usual site.\(^{[41]}\) However, in this case an airborne factor cannot be excluded absolutely, particularly if the facial eruption is symmetric. Another example of ectopic dermatitis in males is genital lesions caused by ‘hand transportation’ of the allergens. Finally, other eczematous skin diseases, for example, atopic eczema having predominant flexural and skin crease involvement must be taken into consideration as a differential diagnosis. Initially, there is an acute flare of the dermatitis during the plant growing season but, with repeated exposure, the flare becomes prolonged and produces a chronic lichenified eczema associated with secondary infection, fissuring and hypo or hyperpigmentation.

**MANAGEMENT**

**General measures**

Severity of contact dermatitis depends upon, degree of contact hypersensitivity and quantity of antigen to which the patient is exposed. For effective control of airborne dermatitis, these two factors should be reduced. In cases of ABCD due to parthenium, one should avoid going outdoors on days especially in summers and in the months from September to November when concentration of pollens is high in the air. Causative plant should be removed from the immediate environment. Air conditioning also decreases indoor pollen counts. Patient should cover as much of the skin as possible by clothing. Uncovered areas should be washed with soap and water as frequently as possible to wash off the antigen before it penetrates the skin. Barrier creams can be used after every wash on the exposed areas in order to slow down penetration of antigen into skin. Other measures which can be used are photoprotection, sunscreens, change of job, change of residence, antihistamines, drying agents in cases of weeping eruptions, aluminum sulfate and calcium acetate and emollients for lichenified areas, etc.\(^8\)

**TREATMENT**

Corticosteroids are the mainstay of therapy. Topical corticosteroids can be used for mild to moderate disease. They decrease the number of HLA DR+ Langerhans cells and inhibit the production and function of IL-1, IL-2 and interferon (IFN)-\(\gamma\). But when there is more than 25% body surface area involvement and when dermatitis is suspected to be caused by allergens which persist in the skin for weeks after exposure (Toxicodendron oleoresins), systemic steroids may be required. Systemic steroids decrease T cell proliferation and are usually prescribed at a starting dose of 0.5-1 mg/kg/ day of prednisolone or 2-3 mg/day of betamethasone. Complete remission generally occurs within 3 months. In order to avoid side effects related to high dose of corticosteroids, effort should be made to taper the steroids accordingly and add adjuvant if required.\(^{[22]}\) Most commonly used immunosuppressive is azathioprine which blocks the DNA replication by incorporating 6-thioguanine into DNA and preventing lymphocytic proliferation after antigenic stimulation;\(^{[42]}\) it is effective in the treatment of the parthenium dermatitis at the dose of 1-2 mg/kg/day. Treatment may need to be continued for 6-12 months after subsidence of disease.\(^{[43]}\) But azathioprine takes 4-6 weeks to exert its action, so is preferred for the treatment of chronic cases and it should be supplemented with corticosteroids in the beginning during the management of acute stage. Weekly azathioprine therapy 300mg /week was found to be as effective as daily treatment with azathioprine with better compliance and reduced cost of therapy.\(^{[44]}\) Major side effects with azathioprine are gastrointestinal intolerance, hepatic dysfunction and bone marrow suppression. Cyclosporine has also been reported to be effective in the acute phase of airborne contact dermatitis due to parthenium. It produces a quicker response and also overcomes the side effects of systemic corticosteroid usage. It suppresses the delayed hypersensitivity reaction as well as the late phase reaction. Side effects of cyclosporine include hypertension and nephrotoxicity. Methotrexate has also been reported to effective steroid sparing immunosuppressive. Sharma et al., treated 16 patients with parthenium dermatitis, unresponsive to topical treatment, with oral methotrexate (15 mg/week). Clinical response was monitored using
a dermatitis area and severity index. Seven patients completed 6 months or more of follow-up, and their mean DASI fell to 5, 2.7 and 2.1 at the end of 1, 3 and 6 months, respectively, from a baseline score of 10. Only 3/7 patients required oral prednisolone in the initial 2–4 weeks. Combination and sequential therapies has also been suggested. Oral hyposensitization was demonstrated to be effective in the 1950s for ragweed dermatitis, but has not been widely accepted because it carries considerable risk of provoking and worsening eczema. In this therapeutic modality, an antigen is introduced into the body by a route different from natural one to induce such a change in the immune system that the body does not develop clinical manifestations when antigen is introduced into the body through normal route. It is thought to act by causing depletion of memory T-cells. Handa et al evaluated the effect of oral hyposensitization in 24 patients of parthenium dermatitis. In 70% of those patients who completed the study, there was a gradual improvement in their clinical status and 30% of patients had an exacerbation during the course of the study. Patients tolerated therapy well and no significant side-effects were seen, except for abdominal pain, 'heartburn,' and cheilitis. Immunootherapy with recombinant protein can be a new emerging option in cases where patients are co-sensitized with several unrelated pollen allergens. Based on frequent co-sensitization patterns, some of the hybrid proteins have been evolved with the polymerase chain reaction. These hybrids contain all the epitopes from the different allergen in a single protein. These have been used for vaccination against pollen allergy. It has been reported useful in hay fever and allergic rhinitis and is under trial for use in ABCD.

CONCLUSION

Despite treatment and change of job in occupational airborne contact dermatitis patients tend to have active symptoms many years after diagnosis. Upto 50% patients with ABCD whether occupational or non-occupational, experience adverse effects on quality of life, daily function and personal relationship, and take time off work on sick leave and may lose or change job because of their skin disease. Emphasis should be laid on avoidance of further exposure. Parthenium dermatitis being most common form of ABCD, continuing attempts to control the spread of the weed through biological measures like introduction of exotic arthropods and opportunistic pathogens, use of antagonistic plants and bioherbicides as well as use of selective chemical herbicides are being taken.

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