Advisory Guidelines for the Avoidance of Exacerbating Factors of Atopic Dermatitis in Daily-Life

JMAJ 45(11): 466–471, 2002

Ichiro KATAYAMA*, Motoi TAKENAKA** and Kenshi YAMAMOTO***

Professor*, Lecturer**, and Senior Lecturer***,
Department of Dermatology, Nagasaki University School of Medicine

Abstract: This article describes current information on factors exacerbating atopic dermatitis (AD) and guidelines for daily-life routines that may be adopted to avoid such exacerbating factors. The prevalence of atopic dermatitis and its refractoriness to treatment have now become matters of public concern. This phenomenon may be explained by multiple factors, such as increase in the prevalence of the responsible allergens attributable to changes in housing conditions, weakening of the skin barrier resulting from alterations of life-style and obsession with cleanliness, inappropriate use of steroid ointments, a flood of folklore medicines, and emotional factors, including stress. To cope with refractory AD, the exacerbating factors should first be identified in individual cases. A partnership between the patient and physician is of great importance in precluding the identified exacerbating factors from the living environment. It is essential in the treatment of AD, to focus on daily-life guidance to eliminate exacerbating factors, adapted for each patient, combined with a skin care routine, appropriate use of topical steroid preparations based on patient education, and drug therapy for pruritus control.

Key words: Atopic dermatitis; Skin barrier function; Exacerbating factors; Daily-life guidance

Introduction

The prevalence of atopic dermatitis (AD) and its refractoriness to treatment have now become matters of public concern. An upsurge of AD in adults since 1980, alterations in the disease pattern of AD, and an increasing number of severe cases with refractory AD have often been pointed out. Although the cause of the increase in number of adults with AD and severe AD remains unclear, increase in the prevalence in the environment of the respon-
possible allergens, attributable to housing conditions, weakening of the skin barrier function resulting from alterations of life-style, inappropriate use of steroid ointments, and a flood of folklore medicines have been proposed. 2) A sharp rise in the prevalence of AD has generated widespread concern in Japan. The Japanese Dermatological Association has developed diagnostic criteria for AD, 3) a scoring system to determine the severity, and guidelines for the treatment of AD. 4) A national epidemiological study on AD has also been initiated by the Ministry of Health, Labor and Welfare. These efforts may be rewarded by the establishment of more effective treatment modalities for AD and better patient education, directed at radical therapy of the allergic disease. From these standpoints, careful treatment of AD and daily-life guidance should be provided in clinical practice to the patients after identification of the exacerbating factors in individual patients.

Pathogenesis of Atopic Dermatitis

Since it was first proposed by Besnier et al. that AD was a familial or constitutional skin disease, many different hypotheses on the etiology of AD have been published, reflecting the new scientific dogma and discovery of the day (Table 1). Dermatologically, physiological disorders, such as skin and airway hypersensitivity, are considered to greatly influence the
development and progression of AD in people with an inherited allergic disease trait, characterized by overproduction of immunoglobulin E (IgE).

As shown in Fig. 1, the skin of a patient with AD is often very susceptible to allergy and is called atopic skin. Such vulnerability of the skin may result from a combination of genetic and environmental factors. Environmental allergens involved in atopic disease, such as house dust mites, referred to as atopens, trigger the overproduction of IgE antibody when they come in contact with sensitive, atopic skin, while irritants induce epidermal keratinocytes and fibroblasts to release various types of cytokines and predispose to skin inflammation. External skin preparations, cosmetics, and shampoos can also cause allergic contact dermatitis in some people.

Thus, when we review the pathogenesis of AD in terms of atopens and irritants, factors further triggering the already overactive immune system producing IgE antibody should be analyzed in allergic inflammation. At the same time, a detailed investigation of non-allergic factors impairing the physiological functions in the skin is required.5–6) Inappropriate treatment of AD mentioned above also needs reviewing.

No national epidemiological study on the cause of AD has been conducted in Japan. There are several conceivable reasons. Firstly, no diagnostic criteria have been clearly established for AD in Japan until 1994.3) Secondly, data integration is difficult, since medical treatment of AD is conducted at several departments of a hospital, including the departments of dermatology, pediatrics, and internal medicine. Furthermore, the disposition of the patient population seen from university hospitals to local clinics varies. At present, we have to speculate the epidemiology of AD in our country from the results of small-scale surveys.

A recent study by Ueda et al. demonstrated that the morbidity rate of allergic diseases, including AD, in children aged 3 to 15 years who underwent medical checkups in Aichi prefecture, was 132/1,512 (8.7%) in city areas, 52/983 (5.3%) in suburban areas, and 46/994 (4.6%) in rural areas, the variations in the rate among the different areas being statistically significant.7) Factors involved in the increase in AD morbidity rate in city inhabitants and adults are complex. The results of a thorough epidemiological study on AD are awaited.

Living Environment and Atopic Dermatitis

In examining how the living environment influences the development, progression, and refractoriness of AD in each patient, the following considerations may be necessary:

1. Factors contributing to overproduction of IgE antibodies

Ever since the suggestion by Sulzberger that AD might be associated with the overproduction of IgE antibodies, IgE has been considered to play an important role in the pathogenesis of AD. However, the details of the involvement of IgE antibodies in the onset of AD remain unknown. Clinical observations have revealed elevation of IgE levels in aggravated AD and elevated IgE titers in proportion to the disease duration in AD. Experimental studies have shown that FcεR1 (+) Langerhans cells in the skin of patients of AD are more active in presenting antigens inducing the production of IgE to T cells. These findings indicate that IgE contributes to the development and progression of AD in many ways.

Airborne allergens, such as house dust mites, molds, pollens, and animal dander, food allergens, microorganisms, metals, and chemicals are commonly known to induce AD. Immunological studies have indicated that the allergens might more strongly induce the proliferation of Type 2 helper T cell (Th2), which stimulates the production of IgE. Frequent exposure to allergens in the living environment leads to overproduction of IgE.

An increase in the prevalence of house dust mites and molds among these allergens could
be attributed to insufficient natural ventilation due to the adoption of air sealing, aluminum-mesh doors/windows, and carpeting, which have rapidly become popular since the 1960’s. Hot humid air emitted from open-type heaters, including oil heaters, also contributes to allergen-filled indoor environments. Condominiums and pre-fabricated houses are the clearest examples of air-sealing and heat insulation.8) Besides, carpets, curtains, sofas, beds, pillows, and stuffed toys that are rarely washed and left around in rooms are good places to find mites. Widespread use of air conditioners, cooking and washing in ill-ventilated homes, and indoor cultivation of tropical fish and foliage also provide favorable environments for the propagation of mites and molds. Pet animals themselves, including dogs and cats, can be a source of allergens, when they are reared indoors. Therefore, patient education is important for the avoidance of these allergens.

Screening tests for atopy conducted yearly on new students at Nagasaki University reveals that the AD morbidity rate is 7 to 8%, while the positivity rate for IgE antibodies specific to dust mites, cedar pollen, and other allergens has been increasing, now at 50%. Metal allergy attributable to the use of various kinds of metal ornaments, and drug allergy owing to the overuse of antibiotics are also evident.

Concerning pollens, Japanese cedar pollen has always drawn much attention as a source of allergens in our country. In fact, dermatitis in the face and other body sites periodically flare up in a significant number of Japanese people during the months of February and March every year. Many of these people have allergy to Japanese cedar pollen. Skin scratch and patch tests also support the contention that cedar pollen triggers the flare of dermatitis in these patients. However, pine pollen and ragweed pollen, which may also be the cause of atopic dermatitis, should not be ignored.

Although food allergy occurs in a limited number of cases of adult AD, proper monitoring is required if redness of the skin or other skin symptoms appear in infants and children after food intake. Parasitic infections, once common in children, are now rarely seen in Japan. The number of patients with tuberculosis has dropped greatly. With this background in mind, some researchers have proposed that a Th1 to Th2 shift in the immune response may be responsible for the increased morbidity rate of AD.

2. Impairment of physiological functions of the skin

Various types of skin dysfunction are observed in patients with AD. Recent studies have indicated that a impaired skin barrier function might be responsible for the skin to become dry and more vulnerable to such allergens as microorganisms and mites. The skin barrier function is conditioned by ceramide, a major component of corneous interstitial cell lipids, and natural moisturizing factor (NMF), which helps to keep the skin moist. There are also sweat-derived molecules involved in pH control and elimination of microorganisms, secretory IgA, and cytokines, including IL-1, in the skin. Abnormal production of these factors may lead to disruption of integrity of the skin barrier.9)

Daily morning shampooing, which has become a phenomenon among senior high school students, can exacerbate dermatitis in the face and hands.10) Since most houses now have a bathroom, daily baths are commonplace; daily baths can damage the natural skin barrier and worsen dermatitis. Further studies are required to clarify the involvement of these factors in AD.

The relationship between air pollution and AD has not been investigated in pilot studies, as in the case of asthma and rhinitis. The effect of nitric oxide and sulfides on the skin has been discussed in several studies. Ultraviolet rays have been reported to exacerbate AD in some cases.

3. Treating atopic dermatitis

In the past few years, impaired development in children resulting from strict food restriction, skin disorders attributable to the indiscrimi-
nate use of corticosteroid ointments, and the flood of folklore medicines for the skin with no scientifically proven efficacy have become problems in the treatment of AD in daily practice.

The harm done by extensive food restriction has already been clarified. Eliminating a proven allergenic food or desensitization by using minimal quantities of the food-borne allergens in the food may prove effective in the control of AD.

The use of topical steroid preparations is often beneficial if they are used according to the directions prescribed in the guidelines developed by the Japanese Dermatological Association. Abrupt cessation of steroid therapy or switch to other therapy without physician supervision, influenced by the propaganda in mass media, folklore medicines, or word of the mouth, have resulted in of great importance in the management of AD. It is therefore important to establish a good relationship between the patients and the physicians in the treatment of AD.

Factors causing Exacerbation of AD

To identify factors causing exacerbation of AD in individual cases in order to guide the treatment, we usually deliver a questionnaire to our patients. As is evident from the responses to the questionnaire shown in Fig. 2, many subjects believe that sweating, emotional stress, sunlight, and a deteriorated housing environment are exacerbating factors. The use of soaps and shampoos are reported to be detrimental in some patients. Since multiple exacerbating factors are found in some severe cases of AD, it is essential to make the effort to identify these factors and eliminate them from the environment for each patient, through taking a proper detailed medical history or other appropriate means.

Conclusion

It is essential in the treatment of AD, to focus on daily-life guidance to eliminate exacerbating factors, adapted for each patient, combined with a skin care routine, appropriate use of topical steroid preparations based on patient education, and drug therapy for pruritus control.

REFERENCES


