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REVIEW ARTICLE

Japanese Guideline for Atopic Dermatitis 2014

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ABSTRACT

Given the importance of appropriate diagnosis and appropriate assessment of cutaneous symptoms in treatment of atopic dermatitis, the basics of treatment in this guideline are composed of (1) investigation and countermeasures of causes and exacerbating factors, (2) correction of skin dysfunctions (skin care), and (3) pharmacotherapy, as three mainstays. These are based on the disease concept that atopic dermatitis is a inflammatory cutaneous disease with eczema by atopic diathesis, multi-factorial in onset and aggravation, and accompanied by skin dysfunctions. These three points are equally important and should be appropriately combined in accordance with the symptoms of each patient. In treatment, it is important to transmit the etiological, pathological, physiological, or therapeutic information to the patient to build a favorable partnership with the patient or his/her family so that they may fully understand the treatment. This guideline discusses chiefly the basic therapy in relation to the treatment of this disease. The goal of treatment is to enable patients to lead an uninterrupted social life and to control their cutaneous symptoms so that their quality of life (QOL) may meet a satisfactory level.

The basics of treatment discussed in this guideline are based on the "Guidelines for the Treatment of Atopic Dermatitis 2008" prepared by the Health and Labour Sciences Research and the "Guidelines for the Management of Atopic Dermatitis 2012 (ADGL2012)" prepared by the Atopic Dermatitis Guidelines Advisory Committee, Japanese Society of Allergology in principle. The guidelines for the treatment of atopic dermatitis are summarized in the "Japanese Guideline for the Diagnosis and Treatment of Allergic Disease 2013" together with those for other allergic diseases.

KEY WORDS

atopic dermatitis, exacerbating factors, guideline, pharmacotherapy, skin care

1. Definition/Disease Concept, Pathophysiology/Etiology of Atopic Dermatitis

1.1. Definition and Disease Concept

The guidelines adopt the definition (concept)[1] of the Japanese Dermatological Association on atopic dermatitis that states "atopic dermatitis is a disease with repeated exacerbation and remission, chiefly characterized by eczema with itch, mostly exhibited by patients with atopic diathesis."

Note: Atopic diathesis. (i) Personal or family history of bronchial asthma, allergic rhinitis and conjunctivitis, and/or atopic dermatitis and/or (ii) predisposition to overproduction of immunoglobulin E (IgE) antibodies. Patients with eczematous lesions that develop during infancy or childhood and persist

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without complete recovery or repeatedly recur even in adulthood.

1.2. Pathophysiology

1.2.1. Inflammatory mechanism

Atopic dermatitis is a disease included in the eczema/dermatitis group. The dominant mechanisms of atopic dermatitis in lesional skin are governed by Th2 cell-related cytokines such as IL-4 and IL-13, and chemokines such as TARC (thymus and activation-regulated chemokine) and eotaxin.[2] Among such chemokines, the so-called Th2 chemokines such as TARC/CCL17 and MDC/CCL22 deserve special attention. These chemokines are chemotactic for Th2 cells expressing the chemokine receptor CCR4. Accordingly, Th2 cells are usually observed at an eczematous site.[3]

This is, however, a pathology in the acute stage, and Th1 cells producing IFN-γ and IL-12 are reportedly dominant in the chronic stage.[4] Langerhans cells and mast cells are involved in the inflammatory response by expressing a high affinity IgE receptor (FcεRI) that causes antigen presenting cells and mast cells to release histamine, cytokines, etc.

The Th2 cytokines IL-4 and IL-13 stimulate fibroblasts to produce periostin, protein causing keratinocytes to produce TSLP,[5] which induces TARC/CCL17 production by dendritic cells.[6] Serum TARC/CCL17 levels are useful as a short-term disease marker for atopic dermatitis and the test is covered by health insurance. Research on Th17 as a new effector cell for allergic reactions[7] and on Treg (regulatory T cell)[8] that controls overreaction is also in progress.

In an eczematous lesion of atopic dermatitis, antimicrobial peptides (defensins, cathelicidins, etc.) are inhibited from being expressed by keratinocytes.[9]

1.2.2. Skin dysfunctions

Expression of ceramide[10] and filaggrin[11] decreases in skin with atopic dermatitis, particularly in lesions, and is considered as a primary cause of barrier dysfunctions. It is also considered as a secondary phenomenon associated with inflammation and as a cause of atopic dermatitis. Atopic dermatitis is accompanied by an acute itch allegedly due to a lowered threshold of itch. Involvement of IL-31 has been reported as a cause of the above.[12]

It is often experienced that itch due to atopic dermatitis cannot be well controlled with antihistamines. Histamine, substance P, and their receptors have been shown to play an important role in itch at the peripheral level. Recently, the role of endogenous opioids such as beta-endorphin and their receptors in itching at the central level has received attention. It has been reported that morphine induces itch via GRP receptors.[13]

1.3. Etiology

Atopic dermatitis is caused by combination of genetic and environmental factors.

1.3.1. Genetic factors

Regarding genetic factors, some etiological candidate genes associated with atopic dermatitis have been reported. Major candidate genes reported to date include CTLA4, IL18, TLR9, CD14, CARD4, PHF11, TLR2, SCCE, MCC, IL4R, GM-CSF, TIM1, CARD15, GSTT1, SPINK5, eotaxin, TGFβ1, IL13, RANTES, IL4, and FcεRIβ. In a recent GWAS of Japanese samples, "2q12 (IL1RL1/IL18R1/IL18RAP)," "3p21.33 (GLB1)," "3q13.2 (CCDC80)," "6p21.3 (MHC region)," "7p22 (CARD11)," "10q21.2 (ZNF365)," "11p 15.4 (OR10A3/NLRP10)," and "20q13 (CYP24A1/PFDN4) " have been reported as candidate genes.[14]

1.3.2. Etiological and exacerbating factors

A wide variety of etiological and exacerbating factors has been proposed, with the importance level of each varying among individual patients. In addition, inflammation associated with this disease will be elucidated by both allergic and non-allergic mechanisms. Etiological and exacerbating factors vary among age groups. While the dominant factors in the first half of childhood include foods, sweating, physical irritation (including scratching), environmental factors, microbes/fungi, the dominant factors in the second half of childhood to adulthood include environmental factors, sweating, physical irritation (including scratching), microbes/fungi, contact allergens, stress, and foods (Fig. 1).

It is commonly experienced that sweating induces itch leading to the aggravation of atopic dermatitis symptoms. Clinically, psychological stress is well known to exacerbate atopic dermatitis symptoms. Although the mechanism is mostly unknown, an increase in sensory nerve fibers containing substance P and CGRP is observed at inflammatory skin sites of patients with this disease.[15]

2. Epidemiology of Atopic Dermatitis

2.1. Global Prevalence of Atopic Dermatitis and Its Changes

An epidemiological survey (Phase I) was conducted from 1994 to 1996 by the International Study of Asthma and Allergies in Childhood (ISAAC).[16] The global prevalence in 6-7 year olds ranged from 1.1% in Iran to 18.4% in Sweden and was 7.3% on average. The global prevalence in 13-14 year olds ranged from 0.8% in Albania to 17.7% in Nigeria and was 7.4% on average. The highest prevalence was seen mostly in industrial nations including Sweden, Finland, UK, Japan, Australia, and New Zealand. In the epidemiological survey (Phase II) conducted from 2001 to 2003 by the ISAAC, few nations showed a significant decrease in the prevalence in 6-7 year olds compared with their

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