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Letter to the Editor

ATOPIC DERMATITIS AND ALOPECIA AREATA ARE TWO AUTOIMMUNE DISEASES THAT NEED ATTENTION

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INTRODUCTION

Atopic dermatitis (AD) and alopecia areata (AA) are both conditions that can be associated with autoimmune responses. AD is a chronic inflammatory skin condition characterized by itchy, red, swollen patches of skin. It is often part of the "atopic triad", which includes asthma and allergic rhinitis. AA is an autoimmune condition that causes hair loss, typically in round patches on the scalp, though it can affect other hair-bearing areas of the body. It is considered to be a tissue-specific autoimmune disorder that is cell-mediated, and it can complicate AD. AA is classified as a type 1 inflammatory disease, while AD is considered to be a type 2 inflammatory disease.

AA can complicate AD, and infections can sometimes trigger autoimmune responses or exacerbate existing autoimmune conditions. For example, certain viral or bacterial infections might precipitate or worsen AA or AD. People with AD often have a compromised skin barrier, making them more susceptible to skin infections, which can further complicate the condition.

DISCUSSION

AD, or atopic eczema, is a dermatological disorder that normally begins in childhood and is characterized by chronic inflammation of the skin. The disease presents with eczematous lesions and itching, and affected subjects have a greater risk of developing allergies, asthma, and other immune and inflammatory disorders (1,2). In AD, the immune system is altered with an increased response to Th2 cells and allergens, with exaggerated production of IgE antibodies (3). The dermis is altered and presents less protection and inflammatory patches. Corticosteroid therapy is still the most used treatment option and in severe cases, immunosuppressive drugs are also used, even if these are not specific. A better knowledge of the pathogenesis of AD, and of autoimmunity in general, could help in the development of new therapies.

AA is a disease where the immune system mistakenly attacks hair follicles, causing hair loss. Understanding the pathogenic mechanisms of this disease can certainly be useful in developing new therapeutic strategies. In this disease, the immune system targets hair follicles during the growth phase, an effect mediated by cytotoxic T lymphocytes (4). Hair follicle-associated antigens are presented by antigen-presenting cells (APCs) to CTLs that damage the follicles (4). There is a genetic predisposition to AA that involves multiple genes. Genes such as CTLA4, PTPN22 and IL2/IL21 have been associated with an increased risk of AA (5). Research studies have shown that Janus kinase (JAK)-signal transducer and activator of transcription (STAT) pathway are important in AA (6,7). These biochemical reactions are activated by

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the cytokines interferon-gamma (IFN-γ) and IL-15 which induce the transcription of genes that lead to inflammation and activate immune responses against hair follicles.

In immune-mediated diseases, such as erythema multiforme (Stevens-Johnson syndrome), toxic necrosis of the epidermis with vasculitis may occur. It has been observed that autoimmune phenomena in certain mammals, such as dogs, can predominantly affect certain parts of the body, including the muzzle and trunk of the animal, causing hair loss and inflammatory patches. Both the front and rear legs of the animal can also be affected. In animals with AA, therapy not only includes pharmacological treatment, but also mechanical stimulation utilizing a small roller with needles which, when slid over skin lacking hair, can reactivate the hair bulb and restore the presence of hair.

Immunological dysfunction can trigger pemphigus foliaceus with both histological and clinical pathological characteristics. Pemphigus foliaceus can present with pustules, erosions, crusts, ulcers and scars. At the level of the primary lesions, there may be flaccid vesicles that are easy to break, and the histological tissue may also present with soprobasal acantholysis with cleft formation, and basal cells remaining at the base (row of tombstones). In dogs, penphigus vulgaris can be treated with prednisolone or dexamethasone at a dose of 3-4 mg/kg of body weight.

AD can also affect other domestic animals such as cats with lesions that are predominantly located on the face and ears. The mechanism of the disease involves vascular cutaneous erythematosus, cutaneous mucus and inflammation, a phenomenon in which toll-like receptor 7 (TLR-7) participates. In fact, in autoimmune diseases, it has been seen that there is an aggravation of plaque in psoriasis when TLR-7 is blocked (8). The pathogenesis involves strong genetic predisposition and exposure to UV light and can appear a few years after birth.

CONCLUSIONS

AA is a complex disorder involving the interaction of genetic predisposition, immune system dysregulation, inflammation, and biochemical pathways like the JAK-STAT pathway. AD is a disorder characterized by chronic inflammation of the skin where the immune system shows an increased response to Th2 cells and allergens, with exaggerated production of IgE antibodies. Studies on these conditions are needed to develop targeted and effective therapies.

Conflict of interest

The author declares that they have no conflict of interest.

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