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CANINE ATOPIC DERMATITIS:
CLINICAL DISEASE AND DIAGNOSIS

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INTRODUCTION

Canine atopic dermatitis (AD) is thought to be a genetically inherited, Type I hypersensitivity to environmental allergens. Common allergens include grass, tree, and weed pollens, molds, house dust and house dust mites.

The incidence of AD in the canine population is unknown. Previous studies have estimated a 3-15% incidence in the canine population. However, reliable studies are difficult to complete. It is generally thought that in those geographic areas that support flea development, the incidence of AD is second to flea allergy dermatitis. In some areas of the country, however, the incidence of AD may exceed that of flea allergy. Studies in human medicine indicate an increasing incidence of AD in the human population. Anecdotally, there appears to be a similar increase in canine AD.

As stated above, AD is thought to be a genetically inherited condition. Although progress is being made, the mode of inheritance has not been definitively determined. The idea of heritability is supported by the increased incidence within certain breeds and breeding lines, but the clinical manifestations of AD are most likely multifactorial.

PATHOGENESIS OF CANINE ATOPIC DERMATITIS

The pathophysiology of canine AD is still being investigated. However, it is generally thought that the pathogenesis for development towards AD consists of two stages: sensitization and elicitation. In the sensitization stage, there is percutaneous absorption of an allergen, such as weed pollen. The Langerhans’ cell (LC) plays a very important role in AD, and is located in the suprabasilar layer of the epidermis. Initially, there is binding of the antigen to the LC. The LC engulfs the allergen and processes it so it can be presented to the immune system. The immature LC migrates from the skin to a regional lymph node, where it interacts with naive T cells (CD4+) of the T-helper 2 subtype. The LC matures during its migration and stimulates proliferation and differentiation of both effector and memory T-cells, resulting in clonal expansion. These T-cells in turn interact with B-cells. The end result of the sensitization stage is (1) memory and effector T- and B-cells, and (2) circulating and bound, antigen-specific IgE. During this first stage, the individual does not produce clinical signs of AD.

The second stage is the elicitation stage. Once again, there is exposure to a specific allergen and percutaneous absorption of the allergen. In the sensitized individual, there are two events that lead to the manifestation of clinical signs. The first event is that mature LC’s in the skin are primed with IgE and will bind the allergen, process it and present it to the immune system. This time, the LC will present to allergen-specific, memory CD4+ T-helper cells. This stimulates the Th2 CD4+ cells to secrete cytokines IL-4, 5, 10, and 13. These cytokines activate eosinophils (IL-5) and other inflammatory cells that contribute to pruritus. In addition, the allergen will bind to allergen-specific IgE on mast cells and basophils. Cross-linking bound IgE molecules causes degranulation of the preformed mediators found within these cells. These mediators include histamine, serotonin, tryptase, chymase, and heparin. Histamine release causes contraction of vascular endothelial cells and plasma leakage into extracellular space. Additional mediators, such as leukotrienes and prostaglandins are released. All of these inflammatory mediators play a role in the severity of the clinical signs. The role of each has not been completely defined, however.

In human AD, a late-phase reaction has been defined within the skin, and there is good evidence that this reaction also occurs in canine skin following allergen challenge. The late-phase reaction occurs because of chemotraction and infiltration of inflammatory cells into lesional skin. It is thought that this reaction contributes to the clinical signs of AD, but the exact role has not been defined.

CLINICAL SIGNS AND DIAGNOSIS OF CANINE ATOPIC DERMATITIS

The clinical signs of canine atopic dermatitis have been characterized over the past several decades. Pruritus and erythema are considered to be hallmark signs of atopy. Usually, dogs develop clinical signs between 6 months and 3 years of age. Most dogs will initially have distinct seasonality. With time, however, the majority will go on to develop nonseasonal symptoms. In certain geographic locations, a lack of seasonality from the beginning is common. The more common areas of pruritus in the atopic dog include the face, ears, paws, extremities, and/or ventrum. While many dogs will exhibit pruritus in most or all of these locations, some will have fairly localized symptoms. AD has historically been considered the “itch that rashes,” meaning that there is no primary lesion associated with initial symptoms. Recently, some dermatologists have suggested that AD may be associated with a primary eruption such as an erythematous macule, papule or plaque. With chronicity, secondary lesions such as excoriation, lichenification, and hyperpigmentation may occur. Secondary bacterial pyoderma and Malassezia dermatitis are also common. Other symptoms associated with canine AD include changes to the quality of the hair coat, conjunctivitis, rhinitis, urticaria, pyotraumatic dermatitis, acral lick dermatitis, seborrhea, and hyperhidrosis. Aural pruritus, followed by bacterial or yeast infection, can be the sole symptom of atopy in the dog.

Differentials for pruritus in the dog include ectoparasites (scabies, demodicosis), infection (bacterial, fungal), allergies (flea allergy dermatitis, food allergy, contact allergies), metabolic causes (hepatocutaneous syndrome), and neoplasia (epitheliotropic lymphoma). Differentials can be narrowed based on history, concurrent clinical signs, and basic diagnostics. A strong suspicion for atopic dermatitis should be made based on history, physical exam findings, and ruling out other pruritic diseases before “allergy testing” is performed.

ALLERGY TESTING

If the dog’s history and clinical signs are consistent with other pruritic diseases, I attempt to rule them out prior to doing allergy testing. In addition, I recommend allergy testing only for the development of allergen-specific immunotherapy (ASIT). There is no benefit to knowing which specific allergens the dog is reacting to if the owners have no interest in pursuing ASIT.
Many labs offer serum allergy testing, and an entire lecture could be given regarding this subject. Current available in vitro methods include RAST or ELISA tests. Most labs use polyclonal anti-IgE for the detection of allergen-specific IgE in patients’ serum. One lab utilizes a unique recombinant fragment of the extracellular portion of the human high-affinity IgE receptor (FceRIα) for detection of IgE. Because each lab’s techniques are different, comparison between labs is difficult. Traditionally, a lack of specificity (false positive reactions) has plagued serum allergy testing procedures. Newer technology has helped to diminish (but not eliminate) false positives.

Intradermal testing involves injection of small quantities of allergen within the dermis and observation for immediate wheal and flare reaction. While most dermatologists have considered this to be the “gold standard” for allergy testing in the dog, it is not infallible. The disadvantage of skin testing is lower sensitivity (false negative reactions). This can be limited by appropriate withdrawal of medications that suppress wheal and flare reactions (systemic and topical corticosteroids and antihistamines).

Results of serum and intradermal allergy tests on the same patient rarely correlate. In the past, many have used this lack of correlation as evidence that serum-based tests are inaccurate. This may not be the case, however. In my practice, I recommend intradermal testing as the most reliable method for developing allergen-specific immunotherapy. However, for those dogs in which sedation, clipping, and/or withdrawal of medications is not possible, we utilize serum allergy testing.

Once the allergy test results are available, it is your responsibility to choose the relevant allergens that should be included in the ASIT. This may have more influence on your success than the allergy test you choose. First of all, only those allergens that are found in the patient’s environment should be considered. The seasonality of the positive reactions needs to be compared to the dog’s symptoms. Lastly, avoidance should be practiced whenever possible. For example, in a flea allergic dog, strict flea control should be practiced. ASIT with flea antigen has typically been unrewarding.