Canine Atopic Dermatitis: What’s New?

Rosanna Marsella, DVM, DACVD
University of Florida

Abstract: Much progress has been made in understanding canine atopic dermatitis (AD), its triggering factors, and the best approach to its diagnosis and management. This article discusses the clinical features of AD, the pitfalls of allergy testing, and how recent research developments can influence the clinical management of this condition. Of particular interest is the accumulating evidence of skin barrier dysfunction, which should prompt clinicians to consider a shift from “reactive” management, classically focused on controlling and suppressing the inflammation, to a more “proactive” management of young patients, aimed at improving skin barrier function and thus minimizing the risk of future allergic sensitization.

Canine atopic dermatitis (AD) is a common allergic skin disease. The term atopic dermatitis describes a set of clinical signs and lesion location. In most cases, AD has a chronic, progressive course and can significantly affect the quality of life of affected dogs and their owners. Because of its complexity, it is also a frustrating disease to properly diagnose and manage. Clinical signs of canine AD have been widely described, but because they are not unique to AD, diagnosis can be challenging. These challenges are compounded by the fact that allergy testing cannot be used as an initial diagnostic tool because AD is a clinical diagnosis, not an etiologic diagnosis. Allergy testing is only useful after the clinical diagnosis of AD has been established because patients suffering from other pruritic diseases may also have positive results on these tests, as may healthy dogs. Therefore, a systematic diagnostic approach is crucial.

Clinical Signs and Flare Factors

Typically, dogs with AD begin to show clinical signs between 1 and 3 years of age. These signs may initially be seasonal before becoming evident year round. In very mild cases, they may remain seasonal. Erythema and pruritus on the face, paws (FIGURE 1), ears, and inguinal area (FIGURE 2) are typical, as are recurrent skin and ear infections. Previously, it was accepted that primary lesions of AD did not exist; however, it is now known that some dogs with AD present with a primary papular eruption caused by the accumulation of eosinophils and lymphocytes in the area where the allergen is captured. This presentation seems to be more common in young dogs (e.g., younger than 1 year) and in dogs with house dust mite allergies. Clinical signs are triggered in most cases by environmental allergens (the traditional definition of allergy); they can also be caused by food allergens or be nonallergic (i.e., intrinsic disease).

Environmental allergens (e.g., pollen) have long been identified as flare factors for canine AD, which explains why seasonal amelioration of clinical signs can be observed. More recently, it was determined that multiple routes of allergen exposure (e.g., inhalation, ingestion) play a role in determining the severity of clinical signs and that the effect of all routes is additive. However, the most important route of allergen exposure is the epicutaneous route (FIGURE 3). Awareness of the importance of this route of exposure has two major clinical consequences: (1) it helps explain the distribution of clinical lesions, and (2) it highlights the paramount need for frequent application of topical therapy to remove the allergens and decrease their penetration of the skin because this practice could help reduce the sensitization and inflammation triggered by the allergen challenge. Thus, frequent bathing with mild hypoallergenic products can greatly alleviate clinical signs and should be part of the regular maintenance regimen for affected patients. The frequency of bathing depends on the individual patient and the severity of the clinical signs. Bathing two or three times a week is typically sufficient.

Secondary skin infections with bacteria and/or yeast are important complicating factors of AD in dogs. Dogs with AD are prone to recurrent skin and ear infections, which significantly contribute to pruritus. Proper diagnosis and management of such infections is therefore necessary. Hypersensitivity to bacteria and yeast plays an important role in AD in some patients; thus, identification of secondary infections is imperative to minimize the inflammatory process and control the pruritus.

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Food allergy can manifest in several ways, including AD. Therefore, the diagnostic workup of dogs with AD should rule out food as a flare factor. A food trial (e.g., using novel sources of carbohydrate and protein) may be necessary in these patients. If food is a factor, residual pruritus may be present after removal of the offending food from the diet; however, identification and correction of this flare factor may be sufficient to at least temporarily decrease the allergenic load below a threshold of clinical signs.

Although it has been known for a long time that AD is worsened by exposure to allergens, the realization that canine AD can occur even before allergic sensitization is relatively recent. Impairment of the skin barrier has been recognized as a factor in human AD for decades, but in veterinary medicine, evidence for the role of skin barrier dysfunction is recent. Human AD is becoming increasingly common in westernized countries, and it is proposed that excessive use of harsh soap at a young age may worsen an already compromised skin barrier, thereby facilitating the development of AD. In veterinary medicine, increasing evidence shows that the skin of dogs with AD is different from normal canine skin in terms of ultrastructure, lipid composition (e.g., amount of ceramides), and permeability. In people with AD, such changes are primary and precede the allergic component of the disease. Whether a similar situation applies to dogs is currently unknown. What is known is that atopic canine skin is most permeable in young dogs (2 to 3 years of age) and is therefore at higher risk for allergen absorption. The possible clinical implication of this knowledge is that a more proactive approach aimed at removing allergens, thereby minimizing their absorption, and potentially restoring skin barrier function in young dogs predisposed to AD may be the key to decreasing the risk of developing AD. Older dogs appear to have less permeable skin; thus, although sensitization may occur later in life, there seems to be a “window” for maximal allergenic sensitization. The sites predisposed to AD (e.g., paws, face, flexural surfaces, pinnae) are now known to be more permeable than other sites in dogs with AD as well as the same sites in dogs without AD. All these findings further emphasize the importance of topical therapy for the management and, possibly, prevention of AD. Although conclusive studies are lacking, it is speculated that topical application of products containing ceramides or other lipid formulations could be beneficial in improving the skin barrier function in atopic dogs.

In human medicine, early modulation of the immune system by bacteria and parasites has been postulated to play a role in determining the likelihood of development of allergies in predisposed individuals. The “hygiene theory” proposes that decreased exposure to endotoxin is at least partly responsible for the increased likelihood of children in westernized countries to develop atopic disease. Pilot studies in veterinary medicine using helminths for the treatment of canine AD have shown promising results, suggesting that a similar situation may apply to dogs. Early exposure to probiotics was also able to modulate the immune response in highly predisposed dogs in an experimental model of canine AD, further suggesting that modulation of the immune system in the early stages of life is important. All these considerations further highlight the fact that AD is the result of a complex interaction between genetic and
Once a clinical diagnosis of AD is established, allergy of a colony beagle used as an experimental model. This patient and the allergens are appropriately chosen, the success of immunotherapy based on intradermal skin testing is comparable to that based on serology testing.13 In terms of medical treatments to control inflammation and pruritus, evidence-based medicine has shown that very few treatments are very effective.17 Broad-spectrum antiinflammatory agents (e.g., prednisone, cyclosporine) are more effective than "targeted" therapies (e.g., antihistamines, pentoxifylline). The reason for this is that multiple mediators are involved in the inflammation and pruritus in canine AD, and targeted treatments may not be able to overcome alternative pathways and induce major clinical improvement. Combination therapy appears, clinically, to be the best approach. The efficacy of essential fatty acids is still under debate, and the best use of this treatment modality is in combination with other therapies to reduce the need for glucocorticoids. Frequent topical therapy with soothing, antipruritic agents and antibacterial agents is the mainstay in all cases of canine AD.

**Diagnosis Challenges**

Despite the availability of a variety of allergy tests, the diagnosis of AD remains clinical and is based on a compatible history, clinical signs, and the exclusion of other pruritic skin diseases. For this reason, it is important to rule out conditions that may look similar to AD. Deep skin scraping should be performed to rule out demodicosis, and appropriate treatment should be considered to rule out scabies. In nonseasonal cases, a food trial should be considered. Cytology is strongly recommended in all cases to look for evidence of secondary infections. In some cases, clinically relevant infections may be diagnosed based on clinical signs even when cytology is unremarkable; however, cytology should always be part of the global dermatologic evaluation of the patient.

Once a clinical diagnosis of AD is established, allergy testing can be considered to identify the allergens to be included in immunotherapy. Debate still exists about which allergy test is best. Intradermal skin testing and serology testing measure different concentrations of IgE (cutaneous and circulating, respectively), but ultimately, the most important aspect in the success of therapy seems to be proper patient selection for allergy testing (i.e., only after a clinical diagnosis of AD has been established) and proper allergen selection (i.e., correlation between the clinical history of the patient and the allergens to include in the vaccine). If the patient and the allergens are appropriately chosen, the success of immunotherapy based on intradermal skin testing is comparable to that based on serology testing.15

**Management**

Because allergies are chronic, long-term management of affected patients requires owner commitment and education as well as the ability to modify the treatment plan over time depending on the needs of the patient. Most successful treatment plans include a multimodal approach with a combination of oral and topical therapies as well as measures to control concurrent allergies and secondary infections. The age of the patient plays an important role in determining the best treatment regimen, as do the finances and time commitment of the owners. Because immunotherapy may alter the course of the disease (as has been shown in human medicine14,15), it should be presented as the best long-term option for young dogs with nonseasonal allergies that live in an environment with high exposure to environmental allergens. Immunotherapy is usually well tolerated. General guidelines on induction and maintenance schedules exist,16 and the individual patient’s reactions to immunotherapy should be used to modify these schedules to minimize adverse effects and increase success.

In terms of medical treatments to control inflammation and pruritus, evidence-based medicine has shown that very few treatments are very effective.17 Broad-spectrum antiinflammatory agents (e.g., prednisone, cyclosporine) are more effective than “targeted” therapies (e.g., antihistamines, pentoxifylline). The reason for this is that multiple mediators are involved in the inflammation and pruritus in canine AD, and targeted treatments may not be able to overcome alternative pathways and induce major clinical improvement. Combination therapy appears, clinically, to be the best approach. The efficacy of essential fatty acids is still under debate, and the best use of this treatment modality is in combination with other therapies to reduce the need for glucocorticoids. Frequent topical therapy with soothing, antipruritic agents and antibacterial agents is the mainstay in all cases of canine AD.

**References**