Atopic dermatitis is a common diagnosis in veterinary dermatology affecting as much as 10% of the canine population. A key factor in the pathogenesis of the clinical manifestations of atopy is the presence of high levels of allergen specific IgE with cross linking of this IgE on the surface of mast cells resulting in mast cell degranulation. Release of inflammatory cytokines from de-granulating mast cells is responsible for many of the clinical signs associated with atopic dermatitis. However, it should be appreciated that canine atopic dermatitis is a complex and multifactorial disease involving immune dysregulation, allergic sensitization, skin barrier defects, microbial colonization and environmental factors.

Atopy is an inherited predisposition to develop antibodies against aerogenous, contact (epicutaneous) and ingested environmental allergens. Distribution of lesions is not dependant on the route of allergen exposure and the same environmental allergen is capable of inducing atopic skin disease via epicutaneous absorption, ingestion or inhalation. All routes of exposure have an additive effect, but the epicutaneous route appears to be the most important. Recent studies have shown that many atopic dogs exhibit signs of atopic dermatitis when challenged with food items to which they may become spontaneously allergic.

Although numerous treatments exist for atopic dermatitis, many have significant side effects and drawbacks and not all are universally effective. Allergen specific immunotherapy (ASIT) is the only proven treatment for atopic dermatitis that works through reversing the underlying immunopathogenesis of the disease with the added advantages of being virtually free of serious adverse effects, even with prolonged use, offering substantial, long-lasting relief in many patients.

**Diagnostic Principles for Atopic Dermatitis**

In 2010 Favorot et al published a robust set of historical and clinical criteria consistent with a diagnosis of atopic dermatitis.

- Onset of signs under 3 years of age
- Dog living mostly indoors
- Glucocorticoid-responsive pruritis
- Pruritis before skin lesions
- Affected front feet
- Affected ear pinnae
- Non-affected ear margins
- Non-affected dorso-lumbar area
Attaining a good history and recognizing the primary clinical signs should be the initial starting point of the clinical workup of the case. Perform a dermatological examination to characterise the lesions and their distribution to create a differential list to work from. Perform some basic in-house diagnostic tests (superficial and deep skin scrapes, surface cytology and otic cytology) and collect lab samples for histopathology and culture (bacterial and fungal). Food trials are also essential to perform prior to confirming CAD.

Distribution of lesions and pruritis is quite characteristic – face, ears, paws, limbs, ventral aspects of the body and perineum. Not all sites are affected in all dogs with CAD. Distribution can vary with complicated CAD (ectoparasites, bacterial / yeast infection, food allergy)

There are also significant breed variations in the history and clinical presentation of canine atopic dermatitis. However, it is important to remember that none of these criteria are pathognomonic. Therefore, diagnosis of atopic dermatitis still remains a diagnosis of exclusion and it is critical to eliminate ectoparasites and evaluate the role of food.

**Therapy for Atopic Dermatitis in Dogs**

In 2005 the then American Task Force introduced the Canine Atopic Dermatitis Extent and Severity Index (CADESI) as a uniform scoring system in clinical trials. This system has been further developed and refined by the International Committee for Allergic Diseases to develop the current CADESI-03 system which has been rigorously validated for use in canine atopy clinical trials. In 2012 a more practical system Canine Atopic Dermatitis Lesion Index (CADLI) was developed, whereby only scoring of frequently affected sites was performed. This CADLI has applications in both clinical trials as well as in practice.

With the CEDESI scoring system being widely employed in most evidence based research into canine atopic dermatitis the International Committee for Allergic Diseases has been able to rate the efficacy of various treatment modalities employed for the treatment and management of CAD.

Currently medications with good evidence of high efficacy include
- Topical and oral glucocorticoids and calcineurin inhibitors such as oral ciclosporin and topical tacrolimus.
- Mastnib a selective tyrosine kinase inhibitor capable of down regulating mast cell functions, has proven to be an effective and mostly well tolerated treatment for canine AD, including severe and refractory cases.
- Oclactinib a novel selective anti-pruritic Janus kinase inhibitor is registered in the USA and EU for the control/treatment of pruritis associated with allergic dermatitis and control/treatment of dogs with atopic dermatitis. Oclactinib provides rapid, effective and safe control of canine AD by eliminating pruritis and the associated inflammatory cascade which follows.
- Allergen Specific Immunotherapy is recommended in all cases of CAD, unless clinical signs are very mild and controlled very easily with non-steroidal therapy.

The International Committee for Allergic Diseases (International Task Force for Canine Atopic Dermatitis) recommends a multifaceted approach to the treatment of dogs with AD.
Avoidance
- Keep grass short in the garden.
- Use of wipes (dairy teat wipes) to wipe dogs down after outdoor activity.
- Cover mattresses with impermeable covers, clean and remove dust from home frequently.
- Dogs bedding should be made of synthetic materials.

Topical Measures
- Frequent bathing (1 x weekly) to remove allergens and reduce bacterial and yeast colonization. Hypoallergenic shampoo’s with anti-bacterial and anti-fungal effects (chlorhexidine based products) are best suited here.
- Topical steroids spray / leave on rinses – can assist in putting the “fire out”.
- Barrier Function restorers (phytosphingosines, ceramides, fatty acids).
- All dogs with CAD should be on some form of topical therapy.

Control of Secondary Factors
- Immune dysregulation and epidermal barrier dysfunction are central in the pathogenesis of atopy resulting in a low irritant threshold and poor ability to resist colonisation by micro-organisms (bacteria / yeasts / ectoparasites)
- Empiric therapy for control of Sarcoptic mange and fleas must be implemented early. Many flea allergic patients have concurrent CAD and atopic dogs show increased susceptibility to other forms of allergic dermatitis.
- Control bacterial and yeast infections with topical and systemic therapy. For best results shampoo’s should be left on for contact times of 10-15 minutes. Warn owners that many of these medicated shampoo’s do not lather well.

Nutritional Measures
- Essential Fatty Acids – fish based oils high in omega-3 have shown far greater improvements in CADESJI scores compared to non-fish based oils (linseed, soya, canicola, hemp seed). Mechanism of action is through restoration of epidermal barrier function.
- There are no over-the-counter hypoallergenic diets.
- Only therapeutic, veterinary distributed, hypoallergenic diets should be used. These diets can be used in treating and testing food allergy.

Acute flares should be treated with a combination of non-irritating baths and topical glucocorticoids, once an attempt has been made to remove the suspected causes of the flare. Oral glucocorticoids and antimicrobial therapy must be added when needed. Currently recognized flare factors include food, flea and environmental allergens, Staphylococcus bacteria and Malassezia yeasts. Skin and coat hygiene and care must be improved by bathing with non-irritating shampoos and dietary supplementation with essential fatty acids. Strict ectoparasitic control is mandatory. The severity of the pruritis and skin lesions can be reduced with a combination of anti-inflammatory drugs.

For the treatment of chronic atopy consider avoidance of factors that trigger AD, improving skin barrier function by bathing with non-irritating shampoos; dietary supplementation with essential fatty acids, Allergen Specific Immunotherapy (ASIT), administration of anti-inflammatory medications with evidence of good efficacy including topical and oral glucocorticoids, oral ciclosporin / topical tacrolimus and anti-pruritic agents such as the Janus
kinase inhibitor oclacitinib.

The evidence for efficacy of anti-histamines in the treatment of CAD is poor.

However, many of these modalities have significant side effects and drawbacks and not all are universally effective. **Allergen specific immunotherapy** (ASIT) is the only proven treatment for atopic dermatitis that works through reversing the underlying immunopathogenesis of the disease with the added advantages of being virtually free of serious adverse effects, even with prolonged use, offering substantial, long-lasting relief in many patients.

**REFERENCES**