

CLINICAL KNOWLEDGE INSIGHTS

ALLERGIC DERMATOSES

ATOPIC DERMATITIS – CANINE

Clinical Knowledge Insight created by Candace Sousa, DVM, DABVP, DACVD

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AT A GLANCE

- A genetically predisposed inflammatory and pruritic allergic skin disease with characteristic clinical features commonly associated with IgE antibodies directed against environmental allergens.
- Mode of inheritance is unknown and likely polygenetic; environmental factors may be important in the development of the disease.
- Defects in the skin barrier may play an important role in allergen exposure. Some dogs with AD have decreased expression of filaggrin in their keratinocytes. Others have decreased quantities of ceramides and structural defects in the intercellular lipid lamellae of their stratum corneum.
- Allergens are absorbed through the skin and captured by cutaneous Langerhans cells which present them to T-helper 2 lymphocytes in the dermis. This results in release of inflammatory mediators including cytokines that increase allergen-specific IgE as well as cytokines that cause direct neuronal stimulation and itch.

WHAT DOES IT LOOK LIKE?

- Can occur in any breed of dog but is more commonly diagnosed in the terrier breeds (West Highland White, Cairn, fox terrier, etc), golden retriever, Dalmatian, bulldogs and setters
- Mean age of onset is 1-3 years but may begin as early as 6 months of age
- May be seasonal or year round
- Estimate that 3-15% of dogs have AD ^{1,2}
- The major clinic feature is pruritic behavior – scratching, rubbing, licking, chewing
- The pruritus usually responds to treatment with glucocorticoids
- Primary lesions are rare and most of the clinical signs are secondary to self trauma
- Most commonly affected areas are the periorcular, perioral, caudal carpus and tarsus, inner pinnae and axillae
- Secondary skin infections with *Staphylococcus* and *Malassezia* are common

PATHOLOGIC IMAGE LIBRARY : ATOPIC DERMATITIS



Classic signs of AD



Perioral and periorcular dermatitis



Inner pinnae, erythema



Caudal carpus

WHAT ELSE LOOKS LIKE THIS?

- Other allergic and pruritic dermatoses
 - Food allergy (cutaneous adverse reactions to food)
 - Flea bite hypersensitivity
 - Sarcoptic mange
 - Pruritic pyoderma
 - *Malassezia* infections
- Demodicosis

HOW DO I DIAGNOSE IT?

- The diagnosis of AD is made by exclusion of other causes of pruritic dermatitis
- Intradermal testing or measurement of serum allergen-specific IgE is used to select allergens used for hyposensitization (see below), not to make the diagnosis

INTRADERMAL TESTING VIDEOS: [ExcellenceInDermatology.com](https://www.excellencein dermatology.com) → [Education Library](#) → [Videos](#)

HOW DO I TREAT IT?

ENVIRONMENTAL CONTROL (ALLERGEN AVOIDANCE)

- Use HEPA vacuums and replace filters frequently
- Clean air ducts, use HEPA filters
- Keep animals indoors when mowing grass
- Use micro-pore covers on mattresses and pillows and wash bedding frequently
- Use air conditioners and dehumidifiers indoors
- Benzyl benzoate can be used to kill housedust mites

CORTICOSTEROIDS

- Usually provide rapid relief from itching and control of inflammation
- Prednisone or prednisolone - 0.25-0.5 mg/kg PO SID for 3-7 days to start and then tapered to the lowest effective dose given every other day
- Methylprednisolone given orally has a lower incidence of causing polyuria and polydipsia
- Avoid repeated injections of long acting or repository corticosteroids
- Safe annual dose of prednisone: Body Weight (kg) X 30 = mg prednisone / year

ANTI-HISTAMINES

- None are approved for use in veterinary medicine, however are usually well-tolerated
- Evidence-based medicine fails to show that they provide benefit in the treatment of AD, however individual animals may show response
- May be synergistic with other medications (e.g., Temaril-P® contains trimeprazine with prednisolone)

CYCLOSPORINE

- 5mg/kg PO SID x 30 then decrease frequency
 - Response may take 4-8 weeks
 - Most common side effects are vomiting, diarrhea and loose stools (~30%)³
- Give with food to minimize GI side effects
- Can premedicate with metoclopramide HCl or maropitant citrate
 - Gingival hyperplasia may occur, reduce dose or use azithromycin oral paste
 - Control skin infections before starting cyclosporine
 - Administration with a cytochrome P-450 inhibitor will decrease dose required (e.g., ketoconazole 2.5-5 mg/kg PO daily + cyclosporine 2.5 PO mg/kg daily)⁴

FATTY ACIDS

- Oral and topical supplementation with ω -3 FAs may help in moderating inflammation and improving the skin barrier

TOPICAL THERAPY

- Frequent bathing to remove allergens and control colonization by *Staphylococcus* and *Malassezia*
- Improve barrier function through topical application of fatty acids, ceramides and phytosphingosines

IMMUNOTHERAPY (HYPOSENSITIZATION)

- Allergens selected based on the results of intradermal testing or allergen-specific IgE serology
- About 2/3 dogs show some response to therapy⁵
- May take up to a year of therapy before improvement is seen

FURTHER READING

PUBLICATIONS: [ExcellenceInDermatology.com](#) → [Education Library](#) → [Publications](#)

COMMENTS

- Best practices for control of AD are early diagnosis and establishment of a management program
- Controlling flare factors such as skin infections and fleas is essential
- Client education about the pathogenesis and ongoing nature of AD is key to successful management.
- Consider referral to a local dermatologist

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