doi.org/10.51891/rease.v9i11.12246

+CANINE ATOPIC DERMATITIS: DETAILED GUIDELINES FOR DIAGNOSIS AND ALLERGEN IDENTIFICATION

Diogenes José Gusmão Coutinho
Jaqueline Saraiva de Souza
Jaqueline Moura da Silva
Ana Paula Teixeira

ABSTRACT: Canine atopic dermatitis (AD) is a common, genetically predisposed, inflammatory and pruritic skin disease. The variation in clinical presentations, due to genetic factors, extent of the lesions, stage of the disease, secondary infections, as well as resemblance to other non-atopic related skin diseases, can complicate a diagnosis of canine AD. A sub-group of the International Committee for Allergic Diseases in Animals (ICADA) was tasked with the development of a set of practical guidelines that can be used to assist practitioners and researchers in the diagnosis of canine AD. Online citation databases and abstracts from international meetings were searched for publications related to the topic, and combined with expert opinion where necessary. The final set of guidelines was approved by the entire committee.

Keywords: Animals. Dogs. Dermatitis.

RESUMO: A dermatite atópica canina (DA) é uma doença cutânea comum, geneticamente predisposta, inflamatória e pruriginosa. A variação nas apresentações clínicas, devido a fatores genéticos, extensão das lesões, estágio da doença, infecções secundárias, bem como semelhança com outras doenças cutâneas não atópicas, pode complicar o diagnóstico de DA canina. Um subgrupo do Comité Internacional para Doenças Alérgicas em Animais (ICADA) foi encarregado de desenvolver um conjunto de directrizes práticas que possam ser utilizadas para auxiliar profissionais e investigadores no diagnóstico da DA canina. Bancos de dados de citações on-line e resumos de reuniões internacionais foram pesquisados em busca de publicações relacionadas ao tema e combinados com opiniões de especialistas quando necessário. O conjunto final de diretrizes foi aprovado por todo o comitê.

Palavras-chave: Animais. Cães. Dermatites.

INTRODUCCION

A new tool to assist with the interpretation of the clinical findings when confronted with a pruritic dog is application of clinical criteria known as “Favrot’s criteria” (Table 4) [5]. These include a set of criteria that have been developed from a large case series of confirmed cases of canine AD. The use of complex statistical analysis allowed a set of clinical features to be identified that had maximum

association with canine AD. The analysis revealed two sets of criteria, which yield varying levels of sensitivity and specificity for the condition. Clinicians can use whichever set best serves their needs. For example, use of a set of criteria that yields the highest specificity is more likely to ensure that a particular case actually has canine AD. However, this set would exclude some pruritic dogs that were suffering from the disease. A set yielding the highest sensitivity is more likely to capture cases of canine AD, but it could allow some dogs with other conditions to be classified as atopic when in fact they were not. Further guidance about application of these criteria sets is shown in Table 4.

It is crucial to remember that these criteria should not be used in isolation as a "diagnostic test" for canine AD. They should be applied alongside the other guidelines outlined in this review. In other words, the accuracy of using these criteria will be greatly enhanced if the dog has been subjected to a careful work-up as described in the previous section.

Allergy testing

Once a clinical diagnosis of canine AD has been made several factors may play a role in the decision-making whether an allergy test is necessary or not. Severe clinical signs, duration of clinical signs for more than 3 months per year, and insufficient management with symptomatic therapy, due to side effects to the drugs used and/or poor owner compliance, justify in most cases allergy testing. These can be performed by IDT and ASIS. Both tests are not recommended as screening tests and should only be used to confirm the clinical diagnosis of canine AD. The results of these tests are also used to identify the offending allergen(s) in order to formulate an allergen-specific immunotherapy (ASIT). Although IDT is considered the preferred diagnostic method among dermatologists, ASIS has several advantages over IDT, such as: no patient risk (no sedation required), less traumatic (no repeated injection required), more convenient (no clipping needed, less time consuming), and lower risk of drugs interfering with test results (concurrent anti-inflammatory/antipruritic therapy) [45, 46]. However, ASIS only measures circulating allergen-specific IgE, does not take into account other allergic pathways and often shows positive reactions in non-allergic dogs [47, 48].

IDT and ASIS are still lacking standardization and it is suspected that false positive and false negative results do occur. It is estimated that between 10 and 30 % of dogs with a clinically confirmed canine AD may show a negative IDT [49, 50]. This high percentage of false negative results can be due to several factors including improper technique, too low test concentration of allergens [51, 52], drug interference [46], intrinsic host factors, incorrect selection of allergens, IDT performed too long after (>60 days) or during the peak allergy season, and presence of a condition called atopic-like dermatitis [49].

Canine atopic-like disease is clinically identical to canine AD, but IgE response to environmental or other allergens cannot be documented [1]. However, in a recent study the condition has been associated with a lymphocyte-mediated reaction to food [53]. Although it is well known that in people age and season may influence ASIS [54], this information has not been well established in dogs.

Results

A total of 81 publications relevant for this review were identified. The guidelines generated focus on three aspects of the diagnostic approach:

1. 1.

Ruling out of other skin conditions with clinical signs resembling, or overlapping with canine AD.

2. 2.

Detailed interpretation of the historical and clinical features of patients affected by canine AD.

3. 3.

Allergy testing by intradermal versus allergen-specific IgE serum testing.

Conclusions

The diagnosis of canine AD is based on meeting clinical criteria and ruling out other possible causes with similar clinical signs. Flea combing, skin scraping and cytology should be performed, where necessary, as part of a thorough work-up. Elimination diet trials are required for patients with perennial pruritus and/or concurrent gastrointestinal signs. Once a clinical diagnosis of canine AD is made,

allergy testing can be performed to identify potential causative allergens for allergen-specific immunotherapy.

Background

Canine Atopic Dermatitis (AD) has been defined as a genetically predisposed inflammatory and pruritic allergic skin disease with characteristic clinical features. It is associated most commonly with IgE antibodies to environmental allergens [1]. Although this definition encompasses many aspects of the pathogenesis and clinical aspects of the condition, it is important to remember that this disease has no pathognomonic clinical signs that permit a definitive diagnosis to be made upon initial owner interview and clinical examination [2]. This is due to the diversity of the clinical presentation, which may depend on genetic factors (breed-associated phenotypes) [3, 4], extent of the lesions (localised versus generalised), stage of the disease (acute versus chronic), and the presence of secondary microbial infections or other flare factors. Furthermore, some aspects of the disease can resemble other skin conditions that are not related to canine AD. For the above-mentioned reasons, the definitive diagnosis of canine AD can be difficult.

A sub-group of the International Committee for Allergic Diseases in Animals (ICADA) developed, based on extensive searches in online citation databases and abstracts from international meetings, a set of practical guidelines that can be used to assist practitioners and researchers in the diagnosis of canine AD.

These guidelines provide an overview of the diagnosis of canine AD that involves three distinct, but complementary, approaches. These are:

1. 1.

Ruling out of other skin conditions with clinical signs that can resemble, or overlap with canine AD. This is traditionally referred to as “the work-up”.

2. 2.

Detailed interpretation of the historical and clinical features of the condition. A new tool to assist with interpretation of these findings is the application of clinical criteria known as “Favrot’s criteria” [5].

3. 3.

Assessment of skin reactivity by IntraDermal Testing (IDT) or detection of IgE by Allergen-Specific IgE Serology (ASIS) testing. This is traditionally referred to as "allergy testing".

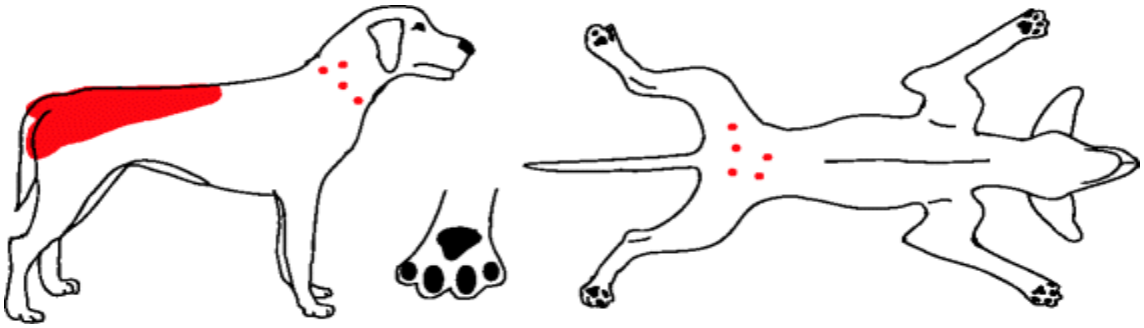
Use of any one of these approaches in isolation can result in misdiagnosis, so it is important not to rely on any of them as a sole diagnostic principle.

Ruling out of other skin conditions with clinical signs that can resemble, or overlap with, canine AD

The evaluation of a pruritic dog requires a step-by-step thought-process and approach that should lead to a definitive diagnosis. The differential diagnoses and role of complicating factors (Table 1) need to be narrowed down using information derived from the history, the findings on physical examination, diagnostic tests (where necessary), and response to treatment. Basic sampling methods and diagnostic tests, which may be required to rule out most of the common differentials are flea combing, skin scraping, hair plucking and cytological examination of skin and ear samples. Depending on the complexity of the case, the following steps may be performed over a series of visits, or all at once.

While the clinical signs in a dog with flea infestation are variable, the location of skin lesions and pruritus associated with flea allergy dermatitis (FAD) are most commonly found at the lumbosacral area, tail base and caudomedial thighs (Fig. 1) [6]. A flea infestation is associated with increased flea counts, whereas in dogs with FAD this may not be the case. In addition, clinicians must be aware that many atopic dogs may suffer from concurrent FAD, which may complicate the clinical diagnosis.

Fig. 1



Distribution of skin lesions and pruritus associated with FAD. Acute lesions: Erythematous macules, papules, crusted papules, hot spots. Chronic lesions: Self-induced alopecia, lichenification, and hyperpigmentation

To exclude FAD or flea infestation as a possible cause of pruritus in a particular case, clinicians should apply the following guidelines:

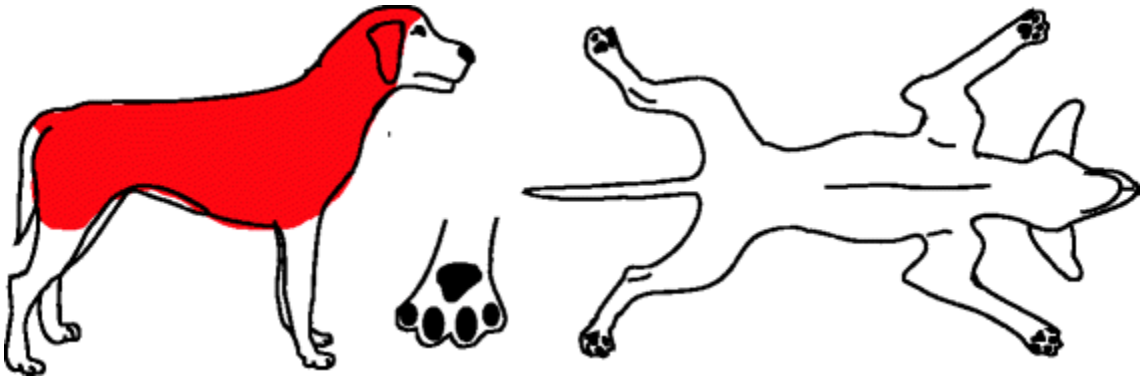
- The prevalence of fleas and associated hypersensitivities depends on the geographical area in which the animal lives. Fleas can be a perennial problem in subtropical and tropical climate zones, seasonal in more tempered climate zones and practically non-existent in arid, high elevation, or cold climates [7, 8]. Even if fleas are considered to be absent from a particular area, clinicians should consider any recent travel history to flea endemic areas or contact with animals from such areas.
- In dogs with pruritus and/or lesions in areas of the body that are not primarily affected by fleas (e.g., the paws or ear canals), FAD may not be the sole cause of pruritus.
- Clinicians should check all pruritic dogs for fleas or flea faeces on direct examination or brushing the hair coat (flea combing). To exclude FAD when fleas or flea faeces cannot be found, an effective flea control program should be initiated. Clinicians should be aware that none of the current flea preventatives have an effective repellent effect, and that the fleas in the pupal stage can survive up to 174 days [9]. Based on duration of survival it is recommended to maintain consistent flea prevention in flea endemic areas. It is also advised that fast-acting systemic adulticides are used as these may be more effective at reducing pruritus quickly compared to other topically applied flea preventatives [10].
- Cases that are being entered into a study of canine AD should undergo effective flea control prior to study enrollment. Because the duration of flea control, prior to study inclusion, may influence the outcome of such trials, a recent study suggests that dogs should be on flea prevention for at least 3 months prior to study enrollment [11]. In addition, all other dogs and cats in the household need to be on effective flea control as well.

Step 2 – Consider the possibility of other ectoparasites

Besides fleas, other ectoparasites may be associated with pruritus (e.g., sarcoptic mange, cheyletiellosis, pediculosis, trombiculiasis, otoacariasis) or can be found as a concurrent disease (e.g., demodicosis). Although the majority of these

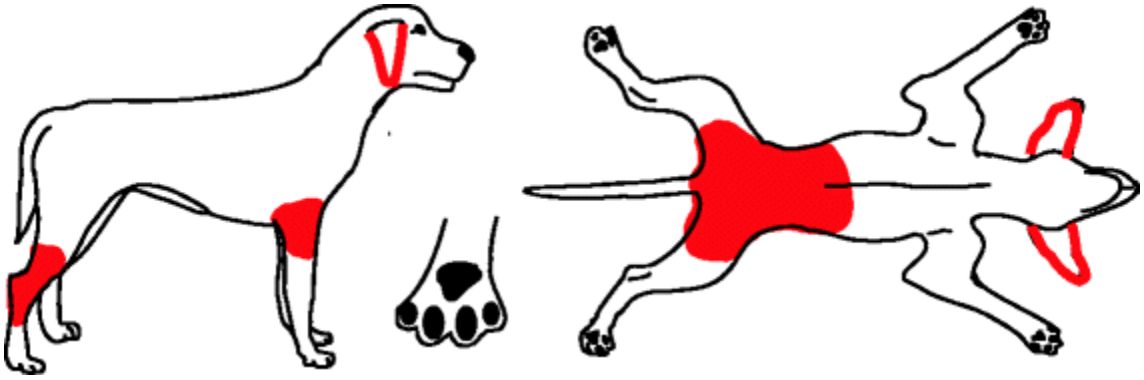
parasites favour specific body areas (Figs. 2, 3, 4, 5 and 6), they can be difficult to distinguish clinically.

Fig. 2



Distribution of skin lesions and pruritus associated with Lice/Cheyletiella. Lice: No visible lesions, or mild scaling and excoriation. Cheyletiella: Marked dorsal seborrhea

Fig. 3

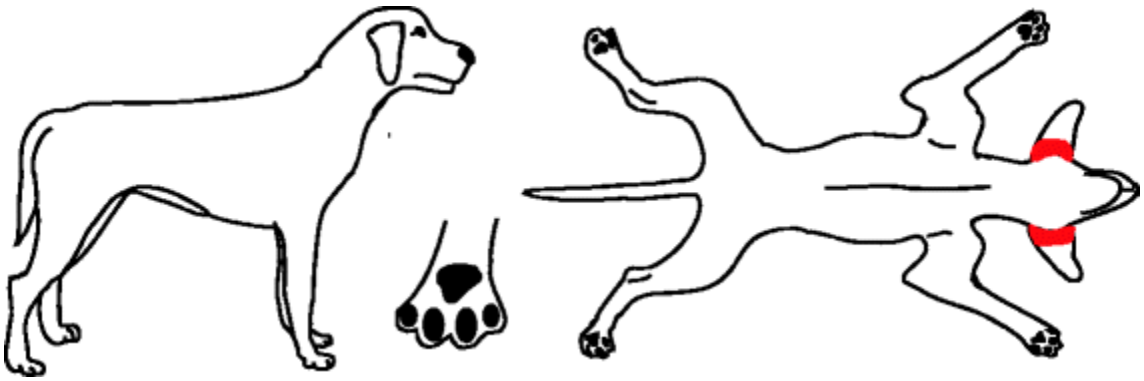


Distribution of skin lesions and pruritus associated with sarcoptic mange. Lesions include papular eruption, erythema, scaling, excoriations

Fig. 4

Distribution of skin lesions and pruritus associated with trombiculiasis. Lesions usually manifest as eruption

Fig. 5



Distribution of skin lesions and pruritus associated with otoacariasis. Lesions include erythema, dark-brown, coffee-ground like discharge

Fig. 6

Distribution of skin lesions and pruritus associated with demodicosis. Lesions include focal, multi-focal or generalised alopecia, scaling, erythema, follicular casts, comedones, Furunculosis

Prior to an allergy investigation, every attempt should be made to rule out potential ectoparasitic skin diseases. Various sampling methods such as skin scraping, hair combing, hair plucking, ear swabbing, and acetate tape impressions can be used to collect specimens. For the identification of these parasites a microscopic examination with a low-power objective (4× or 10×) and low light intensity should be used [12]. The following list indicates which sampling methods are effectively used for various ectoparasites:

- *Sarcoptes scabiei var. canis*: Microscopic examination of multiple superficial skin scrapings, and, where available, blood serum for serology testing (indirect Enzyme-Linked ImmunoSorbent Assay (ELISA) [13, 14]. Sarcoptes mites can occasionally be found on skin biopsies and fecal flotation [15].

- *Demodex spp.*: Microscopic examination of multiple deep skin scrapings and acetate tape impressions of "squeezed" skin, and hair pluckings [16, 17]. Usually *Demodex* mites are easy to find if multiple affected body areas are sampled. However, sampling infected feet or in breeds with thick skin (e.g., shar peis) may not always be effective and skin biopsies may sometimes be required [18].
- *Cheyletiella spp.*, *Trombicula spp.* (chiggers), and lice: Microscopic examination of coat brushings, acetate tape impressions and superficial skin scrapings [15]. *Cheyletiella spp.* and lice also produce eggs, which are attached to hair shafts and can be identified by trichography.
- *Otodectes cynotis*: Microscopic examination of aural discharge. The discharge often appears dark brown-black and crumbly (coffee ground-like) and the mites are white, very mobile and light shy. Occasionally ear mites can be found on superficial skin scrapings at other body sites [19].

Sarcoptes scabiei var. canis and *Cheyletiella spp.* can be difficult to find [15, 20]. For this reason a response to an antiparasitic trial treatment (e.g., selamectin, moxidectin, ivermectin, amitraz, lime sulfur) may be necessary to rule out these parasites. A positive pinnal pedal reflex has been associated with Sarcoptes and justifies trial therapy [21]. Especially in the light that Sarcoptic mites are able to cross-react with house dust mites (HDM) in allergy testing, a trial treatment in very pruritic patients is strongly recommended [22, 23].

Step 3 – Consider the possibility of Staphylococcal infection and *Malassezia* overgrowth

Pyoderma

Bacterial skin infections caused by *Staphylococcus pseudintermedius* (SP) are common in dogs with AD. The typical lesions of superficial pyoderma, such as papulo-pustular eruption and epidermal collarettes, are often distinctive enough to make a clinical diagnosis on gross appearance alone. However, the initial diagnosis should be confirmed by examining cytological samples, stained with Diff-Quik®, taken from the skin by impression smears or acetate tape impressions [12, 24]. Samples from pricked pustules will most likely yield definitive results, while samples from papules and epidermal collarettes may be less rewarding. Aerobic bacterial culture and

sensitivity testing is not indicated in every case, but if particular conditions are fulfilled (e.g., previous history of antibiotic treatment, initial appropriate antibacterial treatment has not been effective, high prevalence of meticillin-resistant SP in the area, etc.), a bacterial culture with antibiogram should be performed [25]. Bacterial cultures can be performed while the dog is currently being treated with systemic antibiotics [26].

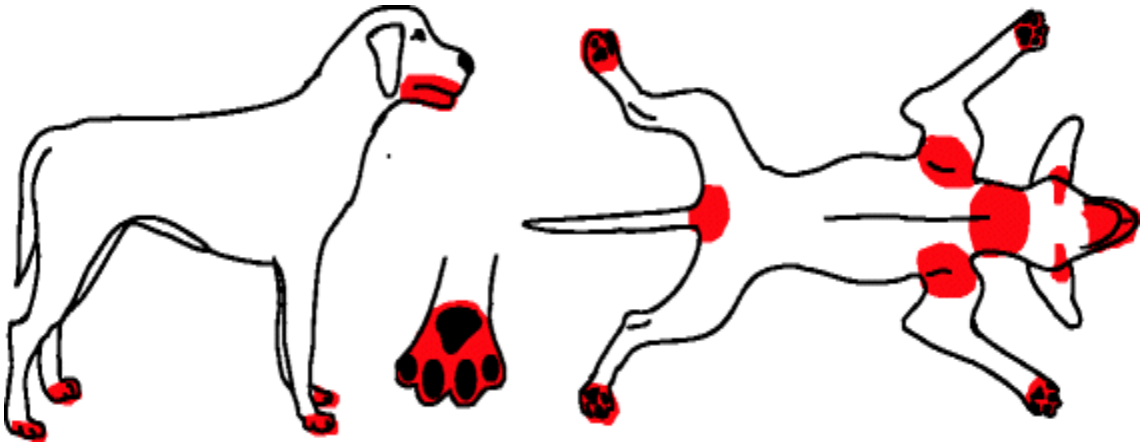
Staphylococcal pyoderma is in most cases a secondary problem associated with underlying pruritic and non-pruritic diseases such as canine AD, but also other allergies as well as endocrinopathies. The pyoderma often causes a change in the overall level or distribution pattern of the pruritus. In these cases, eliminating the pyoderma will determine if the primary disease is itself pruritic, and what its severity and distribution pattern may be. In addition to typical pyoderma lesions, dogs with AD can develop bacterial overgrowth that can complicate other lesion types. Hence, it is wise to sample a variety of lesions to characterise the extent of bacterial involvement and manage the infection appropriately. This should certainly be done whenever cases are poorly responsive to "anti-allergy" therapies, or if studies on canine AD are being performed.

Malassezia dermatitis

The most effective diagnostic test for the identification of *Malassezia* organisms is skin cytology from affected areas such as skin folds, areas with lichenification and oily seborrhea (Fig. 7) [12, 24]. *Malassezia pachydermatis* is a budding yeast organism (3–5 μm in diameter) with a characteristic oval, peanut or "Russian doll" shape, allowing easy identification. In general, clinical signs associated with the cytological presence of yeasts reflect a yeast overgrowth or infection. However, in dogs with *Malassezia* hypersensitivity, few organisms may elicit pruritus and associated skin lesions. For this reason a diagnosis of *Malassezia* dermatitis should be based on the clinical and cytological findings and confirmed by a response to antifungal therapy [27]. Fungal culturing can be performed as well, but is not used routinely for the diagnosis of *Malassezia* dermatitis, because false negative culture results have been reported [28, 29]. Therefore, in studies of canine AD, the presence

of any number of *Malassezia* organisms should warrant a trial therapy to determine what role, if any, low numbers of *Malassezia* are playing in causing the dog's pruritus.

Fig. 7



Distribution of skin lesions and pruritus associated with *Malassezia* dermatitis. Lesions include erythema, yellowish or brownish greasy scale, hyperpigmentation

Step 4 – Consider the role of cutaneous adverse food reaction (CAFR)

Food related pruritus can be caused by two different mechanisms, one a non-immune mediated reaction (food intolerance), the other immune mediated which includes IgE-mediated hypersensitivity (food allergy) [30]. Because reactions to food components can present clinically as canine AD, or serve as a flare factor in canine AD, dogs with CAFR may be indistinguishable clinically from canine AD [31–33]. The presence of gastrointestinal signs, such as diarrhoea, vomiting, tenesmus, soft stools, flatulence, and increased number of bowel movements is more typically seen with food-induced canine AD [5, 33]. In any canine AD case that has year-round clinical signs, CAFR can only be ruled out by effective strict elimination diet trials, since accurate diagnostic commercial tests are not currently available. This is especially important in trials evaluating drugs for the treatment of canine AD since food-induced AD may not respond well to those drugs, as shown for corticosteroids [5]. Unfortunately, there are no diets that have been shown to be effective in all cases of CAFR. Therefore in some cases, especially when gastrointestinal signs are present, multiple different diet trials may be needed until a sufficient control of the clinical signs has been achieved.

Ideally an elimination diet trial should be performed with a diet to which ingredients the dog has never been exposed before. Unfortunately, most commercially available diets contain a wide range of ingredients and by-products, making the selection of an appropriate diet difficult. Most over the counter diets as well as some prescription elimination diets may be contaminated with traces of other food components [34, 35]. Although hydrolysed diets are offered as an alternative option, the protein source is based on either chicken or soy. For this reason some dogs allergic to chicken and/or soy may not respond to such diets [36]. The most common food allergens in dogs are: beef, dairy, chicken products and wheat, and to a lower degree soy, lamb, pork, fish, and corn [37].

A diet trial is performed by instituting a strict trial with a diet containing commercial or home-cooked novel (e.g., rabbit, kangaroo, venison, horse, etc.) or hydrolysed protein ingredients. The use of these novel proteins is becoming more problematic because several of these novel proteins are now available in over the counter commercial diets. A study in humans has also shown that venison does cross-react in vitro with bovine IgG [38], while another study reported that up to 85 % of food allergic dogs may adversely react to venison [39]. Any strict elimination diet trial should be fed exclusively for a minimum of 8 weeks to achieve complete clinical remission in most cases [40]. If the condition improves, the diet should be continued to determine if there is complete or only partial control of the clinical signs. If a dog is not responding to a commercial elimination diet a second attempt with a home-cooked diet should be performed [34]. Home-cooked diets are considered the most limited ingredient diets if done properly. All diet trials should be continued until the veterinarian examines the dog. This is important as some owners may not recognize a partial response or be aware of lesions still present when a dog appears to have improved. Dietary involvement is confirmed if there is a relapse of clinical disease when the original diet is re-introduced. Clinicians should be aware that poor owner/patient compliance is a common problem. Typical pitfalls during a diet trial are: feeding table food, raw hides, treats, “hiding” medication in food, using flavoured tooth paste, giving medication in gelatine capsules, using flavoured drugs (e.g., NSAIDs, antibiotics, chewable heartworm or flea preventative), and dogs eating other animals’ faeces. Clients need to realize that very small amounts of other foods or food

additives ingested, even intermittently, can prevent a favourable response [41]. Crumbs on the floor and even licking another pet's empty bowl may result in a poor outcome. The client's job is to make sure the dog ingests nothing but the prescribed diet and water.

Once steps 1–4 of the diagnostic work-up has been completed, a clinical diagnosis of canine AD should be considered if the pruritus is still present.

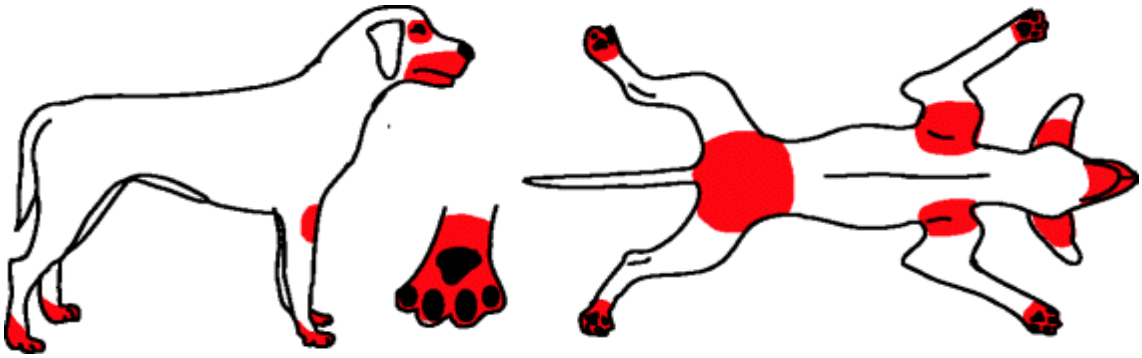
Detailed interpretation of the historical and clinical features of canine AD

The initial clinical feature of canine AD is pruritus, which can include scratching, rubbing, chewing, excessive grooming or licking, scooting, and/or head shaking. Depending on the allergens involved, the pruritus may be seasonal (e.g., pollen) or non-seasonal (e.g., dust mites, food) [42]. At the beginning the pruritus may be alesional or associated with primary skin lesions such as erythema and occasionally papules (Table 2) [43, 44]. The face, concave aspect of the ear pinnae, ventrum, axillae, inguinal area, perineal area and distal extremities are most commonly affected in canine AD (Fig. 8) [43], but breed-associated variations of body sites affected by canine AD have been identified (Table 3, Fig. 9) [3]. In more chronic stages secondary skin lesions (Table 2) will occur due to self-trauma, chronic inflammation and secondary infections. Typical secondary skin lesions are excoriations, alopecia, lichenification, hyperpigmentation, crusting, and seborrhea (Fig. 10a-c).

Table 2 Key dermatologic features for canine pruritic skin diseases

Full size table

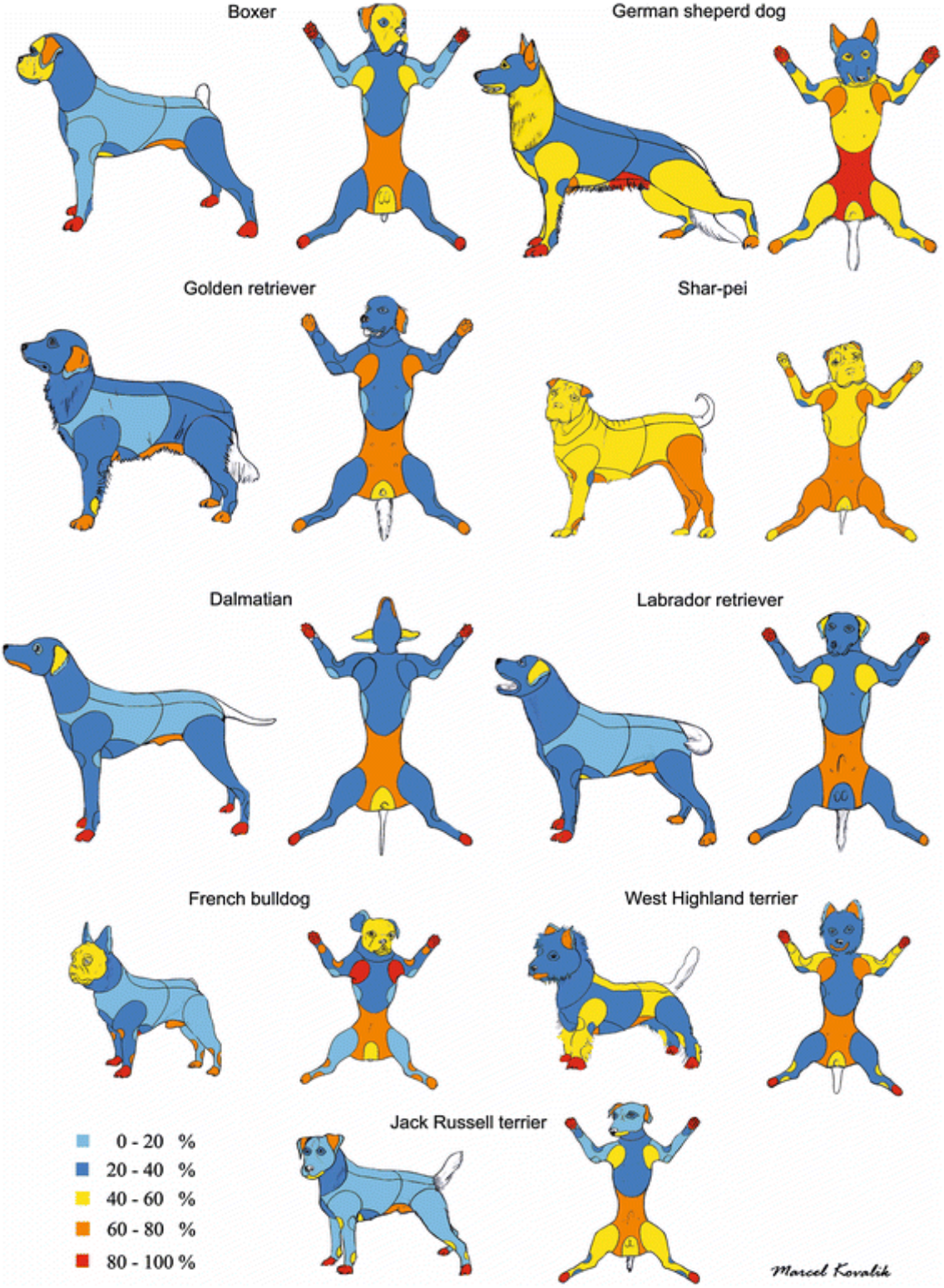
Fig. 8



Common distribution of clinical lesions and pruritus associated with canine AD and food allergy

Table 3 Additional body sites involved in canine AD in certain breeds [3]

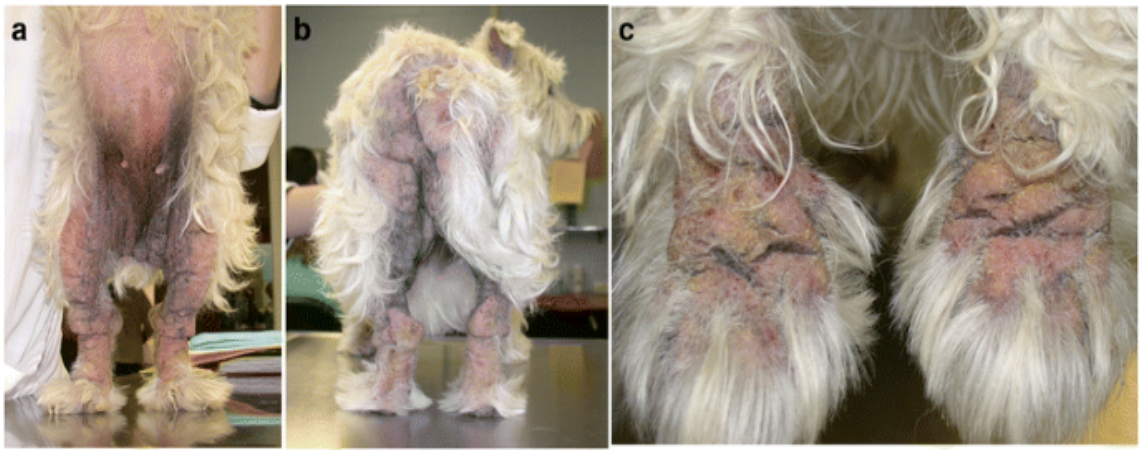
Fig. 9



Silhouettes of atopic boxers, German shepherd dog, golden retrievers, shar peis, Dalmations, Labradors retriever, French bulldogs, West Highland white terriers and Jack Russell terriers (in this order). Each

colour corresponds to the percentage of affected animals (Reproduced with permission from Veterinary Dermatology)

Fig. 10



a, **b**, **c** Typical distribution of secondary skin lesions in a West Highland white terrier

CONCLUSION

Both testing methods are very different and not standardized, which inevitably results in poor correlation between both tests [55]. Nonetheless the success rate of ASIT based on ASIS vs. IDT is not significantly different [56]. Finally, it is important to remember that, although little information is available, cross-reactions between related allergens, e.g., house dust and storage mites, have been reported [57–59]. Based on this problem it is important to determine if a dog is really exposed to the allergen(s) it reacted too. The proper interpretation of these test results, in conjunction with the clinical history and clinical presentation, can be complex and time-consuming. For this reason a referral to a veterinary dermatologist is recommended.

1. Foster AP, Littlewood JD, Webb P, Wood JL, Rogers K, Shaw SE. Comparison of intradermal and serum testing for allergen-specific IgE using a Fcepsilon RIalpha-based assay in atopic dogs in the UK. Vet Immunol Immunopathol. 2003;93(1-2):51–60.

Article CAS PubMed Google Scholar

2. Park S, Ohya F, Yamashita K, Nishifuji K, Iwasaki T. Comparison of response toimmunotherapy by intradermal skin test and antigen-specific IgE in canine atopy. J Vet Med Sci. 2000;62(9):983–8.

Article CAS PubMed Google Scholar

3. Buckley L, Schmidt V, McEwan N, Nuttall T. Cross-reaction and co-sensitizationamong related and unrelated allergens in canine intradermal tests. Vet Dermatol. 2013;24(4):422–7. e491-422.

Article PubMed Google Scholar

4. Marsella R, Saridomichelakis MN. Environmental and oral challenge with storagemites in beagles experimentally sensitized to Dermatophagoides farinae. Veterinarydermatology. 2010;21(1):105–11.

Google Scholar

5. Saridomichelakis MN, Marsella R, Lee KW, Esch RE, Farmaki R, Koutinas AF. Assessment of cross-reactivity among five species of house dust and storage mites. Vet Dermatol. 2008;19(2):67–76.

Article PubMed Google Scholar

6. Bauer CL, Hensel P, Austel M, Keys D. Determination of irritant threshold concentrations of six house dust and storage mites through serial dilutions in intradermal testing on healthy clinically non-allergic dogs. Vet Dermatol. 2009;20:227.

Google Scholar

7. Hensel P. Differences in allergy skin testing among dermatologists within the samegeographical region in the USA. Vet Dermatol. 2012;23 Suppl 1:60.

Google Scholar

8. Hubbard TL, White PD. Comparison of subjective and objective intradermal allergy test scoring methods in dogs with atopic dermatitis. J Am Anim Hosp Assoc. 2011;47(6):399–405.

Article PubMed Google Scholar

9. Cunha VE, Faccini JL. Use of histamine phosphate for the interpretation ofintradermal skin tests in dogs. Vet Rec. 2009;165(24):723–4.

CAS PubMed Google Scholar

10. DeBoer DJ, Hillier A. The ACVD task force on canine atopic dermatitis (XVI): laboratory evaluation of dogs with atopic dermatitis with serum-based “allergy”tests. Vet Immunol Immunopathol. 2001;81(3-4):277–87.

Article CAS PubMed Google Scholar

11. Saevik BK, Ulstein TL, Larsen HJ. Evaluation of a commercially available enzyme-linked immunosorbent assay for the detection of allergen-specific IgE antibodies indogs. Res Vet Sci. 2003;74(1):37–45.

Article CAS PubMed Google Scholar

12. Wassom DL, Grieve RB. In vitro measurement of canine and feline IgE: a review of FceR1a-based assays for detection of allergen-reactive IgE. Vet Dermatol. 1998;9:173–8.

Article Google Scholar

13. Stedman K, Lee K, Hunter S, Rivoire B, McCall C, Wassom D. Measurement of canineIgE using the alpha chain of the human high affinity IgE receptor. Vet Immunol Immunopathol. 2001;78(3-4):349–55.

Article CAS PubMed Google Scholar

14. Olivry T, Jackson HA, Murphy KM, Tater KC, Roberts M. Evaluation of a point-of-care immunodot assay for predicting results of allergen-specific intradermal andimmunoglobulin E serological tests. Vet Dermatol. 2005;16(2):117–20.

Article PubMed Google Scholar

15. Olivry T, Paps J. Evaluation of the agreement between allergen-specific intradermalor IgE serological tests and a point-of-care immunodot assay in dogs with atopicdermatitis. Vet Dermatol. 2011;22(3):284–5.

Article PubMed Google Scholar

16. Plant JD, Neradelik MB, Polissar NL, Fadok VA, Scott BA. Agreement betweenallergen-specific IgE assays and ensuing immunotherapy recommendations fromfour commercial laboratories in the USA. Vet Dermatol. 2014;25(1):15–e16.

Article PubMed Central PubMed Google Scholar

17. Jeffers JG, Shanley KJ, Meyer EK. Diagnostic testing of dogs for foodhypersensitivity. J Am Vet Med Assoc. 1991;198(2):245–50.

CAS PubMed Google Scholar

18. Mueller R, Tsohalis J. Evaluation of serum allergen-specific IgE for the diagnosis offood adverse reactions in dogs. Vet Dermatol. 1998;9(3):167–71.

Article Google Scholar

19. Jackson HA, Jackson MW, Coblentz L, Hammerberg B. Evaluation of the clinical andallergen specific serum immunoglobulin E responses to oral challenge withcornstarch, corn, soy and a soy hydrolysate diet in dogs with spontaneous foodallergy. Vet Dermatol. 2003;14(4):181–7.

Article CAS PubMed Google Scholar

20. Johansen C, Mariani C, Mueller RS. Evaluation of patch testing with proteins, carbohydrates and commercial foods for diagnosis of canine adverse food reactions. Vet Dermatol. 2013;24:385.

Google Scholar

21. Marsella R, Kunkle GA, Vaughn DM, MacDonald JM. Double-blind pilot study on theeffects of ketoconazole on intradermal skin test and leukotriene C4 concentration inthe skin of atopic dogs. Vet Dermatol. 1997;8:3–10.

Article Google Scholar

22. Bond R, Lloyd DH, Craig M. The effects of essential fatty acid supplementation onintradermal test reactivity in atopic dogs: a preliminary study. Veterinarydermatology. 1993;4:191–7.

Google Scholar

23. Goldman C, Rosser Jr E, Petersen A, Hauptman J. Investigation on the effects ofciclosporin (Atopica) on intradermal test reactivity and allergen-specific immunoglobulin (IgE) serology in atopic dogs. Vet Dermatol. 2010;21(4):393–9.

PubMed Google Scholar

24. Aleo MM, Galvan EA, Fleck TJ, Humphrey WR, Coscarelli EM, Mahabir SP, et al. Effects of oclacitinib and prednisolone on skin test sensitivity. Vet Dermatol. 2013;24(3):297.

Google Scholar

25. Martin DD, Martin AL. Pain management and anesthesia in veterinary dermatology. Vet Clin North Am Small Anim Pract. 2006;36(1):1–14.

Article PubMed Google Scholar

26. Kennis RA, Robertson SA, Rosser Jr EJ, Hauptman JG. Effects of propofol anesthesiaon intradermally injected histamine phosphate in clinically normal dogs. Am J Vet Res. 1998;59(1):7–9.

CAS PubMed Google Scholar

27. Graham LF, Torres SM, Jessen CR, Horne KL, Hendrix PK. Effects of propofol-induced sedation on intradermal test reactions in dogs with atopic dermatitis. Vet Dermatol. 2003;14(3):167–76.