

ALLERGIC DISEASE

A therapeutic approach to allergic pruritus in the dog

Stephen Shaw

Allergic skin disease is a common and frustrating problem in dogs with a wide variety of differential diagnoses that must be excluded if appropriate treatment is to be implemented. There is a broad spectrum of possible clinical signs in allergic skin disease and the combination of these signs dictates the most appropriate treatment needed. This article discusses the agents available for treatment of canine atopic dermatitis.

When presented with a dog suffering with pruritus, obtaining an accurate diagnosis is key to successful treatment. Rushing to treat the irritation without due care can delay or even result in failure to effect a cure, leaving the clinician open to complaint or litigation. The most common cause of cutaneous allergy in the dog is canine atopic dermatitis (CAD). When a diagnosis of CAD has been made, further characterisation beyond the diagnosis is essential for the best patient care and, as the clinical signs may change over time, a process of regular re-evaluation is needed to alter the treatments and prevent iatrogenic disease. The owner should play an active part in this process and the informed client, who knows both the benefits and possible pitfalls of a given therapy, is a valuable asset. The minimum evaluation of a case should include a well-taken history and careful clinical examination, including otoscopy, combined with cytology and skin scrapings (discussed elsewhere in this supplement). The components of atopic dermatitis include a defective barrier function, inflammation, pruritus and pyoderma, each of which is interrelated. Considering all of these and noting their importance during diagnosis will ensure an individualised approach in each case (Fig 1).

There are a large number of possible interventions for CAD, these include allergen avoidance, treatments for secondary infection, glucocorticoids, ciclosporin, allergen-specific immunotherapy, antihistamines and essential fatty acids. During the diagnostic process you can gauge which of these are most likely to be appropriate.

Pyoderma

Dogs with pyoderma may be recognised by clinical examination, showing typical pustules, crusts and epidermal collarettes, but, whenever possible, the clinical impression should be substantiated by cytology, and in recurrent pyoderma, with culture and susceptibility testing. Superficial pyoderma can be highly pruritic regardless of the cause, whereas deep pyoderma is often associated with pain and gentle licking. Hypersensitivity to staphylococcal proteins is likely to augment the effects of bacterial toxins and superantigens and may explain the marked increase in pruritus with relatively mild pyoderma (Bexley and others 2013). Treatment with chlorhexidine shampoos (eg, Malaseb; Dechra or Microbex; Virbac) may be sufficient in mild cases, whereas more extensive superficial and deep pyoderma require antimicrobial therapy (see the article by Nuttall on pp 12-17 of this supplement). In CAD, in which recurrent pyoderma is a feature, the aim should be to treat the underlying disease consistently to prevent infection. In such situations the use of topical shampoo or

antibacterial wipes for prophylaxis is acceptable, but there is no place for pulse antimicrobial therapy or the intermittent use of otic antimicrobial ointments.

When pruritus is present with severe pyoderma (Fig 2), it is tempting to use concurrent anti-pruritic drugs. Glucocorticoids may reduce the irritation, but with the additional complications of reduced antimicrobial immune-responses. Thus in cases of severe superficial pyoderma and deep pyoderma caused by CAD steroids should be stopped, whereas the continuation of ciclosporin appears to be acceptable. It can be helpful to treat the pruritus in areas where pyoderma is absent using a topical agent such as hydrocortisone aceponate.

Malassezia dermatitis

Malassezia overgrowth and dermatitis is commonly seen in allergic skin disease and is a significant cause of pruritus. In some cases there is severe episodic facial pruritus with *Malassezia* dermatitis, which proves highly resistant to most antipruritic therapies. *Malassezia* is common in intertriginous areas such as the facial folds, perivulval skin, neck folds and interdigital spaces. Diagnosis is based on a combination of clinical signs and cytology that will readily identify this organism. Treatment with topical shampoos containing chlorhexidine (sometimes also including miconazole) as for pyoderma, as well as wipes containing either boric and acetic acid (Malacetic wipes; Dechra) or chlorhexidine (CLX wipes; Vetruus) is usually effective. In rare cases systemic antifungals are needed, but such drugs are expensive, unlicensed for this use in the UK, and have marked interactions with other agents (Negre and others 2009).

Hypersensitivity reactions to *Malassezia* are common in atopic dermatitis. In some dogs, the presence of *Malassezia* is the key feature causing pruritus and when this is identified, dogs that

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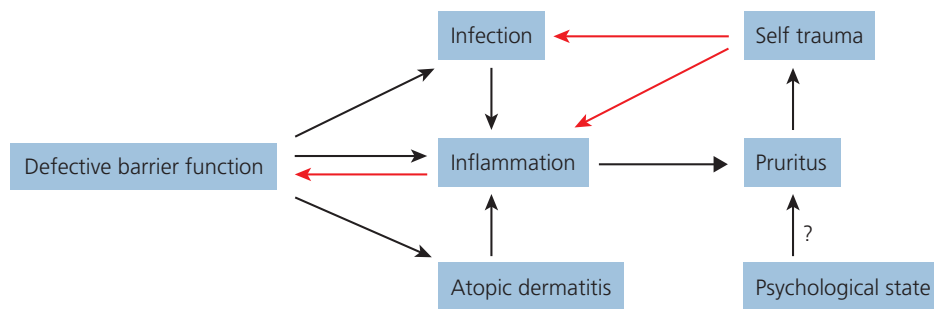


Fig 1: Interrelationship between the components of atopic dermatitis in the dog. In canine atopic dermatitis, a defective barrier contributes to the ingress of allergen, which causes infection and inflammation. These lead to pruritus that results in further infection and inflammation. As in people, dogs living in stressful situations may demonstrate more pruritus

previously relied on ciclosporin or prednisolone to provide comfort may be treated with comprehensive anti-yeast treatment alone, although this is labour intensive.

Other common secondary diseases

Other problems, which need to be addressed to develop a lasting therapeutic regime, include ear disease and recurrent anal sac impaction. Both are common in atopic dermatitis in my experience, and can be caused by dramatic increases in generalised pruritus, which may be confused with seasonal or other causes of flares. As previously stated, the chronic use of antibacterial agents to control such flares should be avoided and in all cases the underlying allergic inflammation controlled.

Treating primary disease

After secondary infections have been addressed, the remaining pruritus can be assessed more accurately. In some dogs, especially the golden retriever, irritation may be so much reduced after antimicrobials that the client will consider the dog to be normal when the pyoderma has been resolved.

There are many approaches to the treatment of pruritus in atopic dogs. These can be divided into avoiding allergens and flare factors, improving and managing barrier function, chemical manipulation of the inflammatory process and immunomodulation through immunotherapy.

Allergen avoidance

Domestic mites (house dust and storage mites) are the most commonly identified cause of sensitisation in dogs with atopic dermatitis in the UK. In house dust mite allergy, excluding the dog from bedrooms, bathrooms and from sitting on furniture is

ideal, but can be difficult to achieve. Washing animal bedding at over 60°C once a week, placing beds in washable hard plastic baskets and vacuuming with 'mite-busting' cleaners have all been recommended. Kennelling is a viable option for some dogs, but owners need to ensure that the kennel is regularly cleaned to avoid the house dust mite population from mirroring that in the house and that the area is included in a flea control plan. A carefully constructed study in the Netherlands showed that vigorous house dust mite control resulted in a significant improvement in the clinical scores of atopic dogs (Swinnen and Vroom 2004). However, for many owners, extensive mite control procedures are too onerous, too expensive and may exclude the pet from family life, so a compromise has to be sought. Often, frequent bathing of the dog, which removes the allergen as well as having other therapeutic advantages, is a better

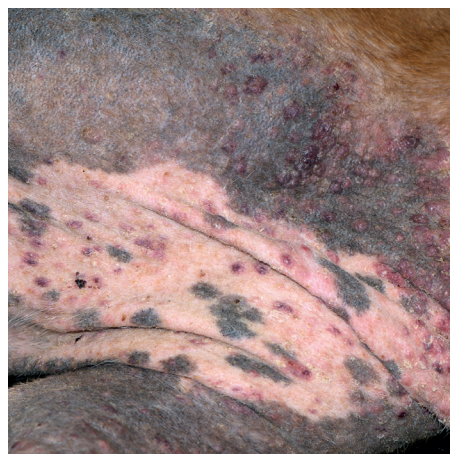


Fig 2: Severe deep pyoderma in a boxer dog with demodicosis, severe alopecia, comedones and large pustules and furuncles affecting the chest and lower neck can be seen. The dog was treated with glucocorticoids to counter the severe pruritus present. Treatments for atopic dermatitis need an accurate diagnosis in all cases. Even when the clinical picture appears to indicate allergic skin disease, clinicians must repeat the diagnostic tests when there is an unexpected response to treatment

use of time and money. Clinicians need to ensure that clients are aware that allergen avoidance is sometimes helpful, but rarely a complete answer.

Barrier dysfunction

Epidermal barrier dysfunction has recently received considerable attention in the veterinary literature. Renewed interest in this area developed when it was shown that filaggrin abnormalities were strongly associated with atopic dermatitis in people (Palmer and others 2006). Although genetic studies for changes in this protein in dogs, which is found in the stratum corneum (Barros Roque and others 2009), have been unrewarding, atopic dermatitis has been associated with changes in filaggrin in dogs (eg, Kanda and others 2013). There is some debate about whether these changes are primary or secondary (Marsella 2013). Regardless of the underlying mechanism, measurement of transepidermal water loss (TEWL) shows that barrier function is deranged in atopic dermatitis and that increased TEWL is noted after allergen challenge (Hightower and others 2010).

There are several ways that the clinician can treat barrier function defects; shampoos containing linoleic acid (eg, Allermyl; Virbac) may be used to improve the skin barrier, but may not be appropriate as the sole shampoo in the face of bacterial or *Malassezia* overgrowth. I have found that a minimum of twice-weekly bathing with a linoleic acid shampoo is needed for good effect, but can be very helpful in some dogs. When bathing with other types of shampoos is necessary, for example, to reduce *Malassezia* numbers, there is a risk that the epidermal barrier will suffer. This can be countered by using a humectant after each bath (eg, Humilac; Virbac). This agent uses urea to trap water and restore barrier function. In the future, we may see shampoos in the UK, such as those available in other countries in Europe, that combine potent antimicrobial properties and that also repair the epidermal barrier, although shampoos with higher concentrations of chlorhexidine require a medicines licence in the UK.

Other topical and systemic therapies may also be used to improve barrier function. Topical lipids (eg, Allerderm; Virbac) and essential oils (eg, Dermoscent; LDCA) have both been shown to be useful in the treatment of CAD (Piekutowska and others 2008, Tretter and Mueller 2011). Alternatively, essential fatty acids (EFAs) may be given orally as supplements or in

Fig 3: Severe pruritus without significant secondary infections in an atopic dog, showing alopecia and marked erythema affecting the caudal and lateral carpus. In cases in which pruritus is the predominant sign without there being significant infection, acute pruritus can be treated with hydrocortisone aceponate when the disease is localised or with glucocorticoids when there is more generalised pruritus. Long-term management using ciclosporin, immunotherapy and shampoo to improve the epidermal barrier is appropriate



Fig 4: Recurrent pyoderma in a golden retriever due to atopic dermatitis. Clinical signs seen are mild alopecia, erythema and crusted papules and macular hyperpigmentation affecting the ventral abdomen. The presence of recurrent pyoderma in canine atopic dermatitis demands prompt recognition by the owner to allow rapid treatment that in the early stages may only require topical shampoos. Once the pyoderma is cured, the underlying treatment for the allergic inflammation may need to be modified to prevent further infections. Dogs with pyoderma may respond poorly to anti-inflammatory treatment alone



diets designed for use in CAD. These supplements help maintain the epidermal barrier and may have an anti-inflammatory effect in the skin through changes in eicosanoid production. Many studies using EFAs fail to account for the EFAs in the diet being fed, but despite these limitations there is good evidence of their efficacy in some well-conducted trials (eg, Saevik and others 2004). I have had success with using an EFA-enriched diet (Dermatosis FP; Eukanuba), but a study comparing three EFA-enriched diets, including this diet, with a supermarket brand was less clear-cut.

There is a wide variation in the composition of EFA supplements, which may contain evening primrose oil, cod liver oil and other sources of EFAs, with widely varying dose rates recommended. Using veterinary products at the recommended doses seems satisfactory. One study has suggested that treatment in early atopic disease may be more useful than later intervention (Abba and others 2005). EFAs have a slow onset of action and few side effects, although softness of faeces and exacerbation of pancreatitis are possible.

Anti-inflammatory drug therapy

Glucocorticoids

Glucocorticoids are often appropriate at some stage of treatment in atopic dermatitis. In dogs, prednisolone is used for either a short course of around five days (in which case a tapered reduction is not needed) or for longer periods with reduction to alternate day therapy and then a further graduated

dose reduction. Atopic pruritus should resolve in 10 days when using 0.75 mg/kg daily in the majority of cases. Side effects include polydipsia, polyuria and polyphagia (possibly leading to obesity) with muscle loss, fat redistribution, increased susceptibility to infection (pyoderma, *Malassezia* dermatitis and demodicosis), depression and behavioural changes, urinary tract infection and intermittent digestive upsets. In trials considering the efficacy of oral steroids in CAD, satisfactory control of clinical signs was seen in 57 to 100 per cent of cases with better than a 50 per cent reduction in clinical signs in 58 to 86 per cent of cases. Adverse events were reported in 30 to 80 per cent of subjects (eg, Steffan and others 2003).

Topical steroid creams and ointments can be used to treat local disease, although these products may cause stickiness of the fur, be ingested through licking, and may cause adrenal suppression and other steroid side effects. Such products are usually combined with antimicrobials and are thus not suitable for use in chronic cases. In contrast, hydrocortisone aceponate (Cortavance; Virbac) is a spray-on product where the carrier allows relatively good penetration of haired areas and, as the drug is metabolised in the dermis, there is reduced potential for adrenal suppression. Although potent, hydrocortisone aceponate is not suitable as a sole therapy in severely affected animals, but when used carefully can prevent the use of more potent agents (Nuttall and others 2009). I do not advocate long-term therapy of the abdominal skin with hydrocortisone aceponate.

Ciclosporin

Ciclosporin (Atopica; Novartis) offers good control of the signs of atopic dermatitis in

both dogs and cats. Ciclosporin acts upon calcineurin-dependent transcription factors to reduce the production of proinflammatory cytokines such as interleukin (IL)-2, IL-3 and IL-4, resulting in a reduced T lymphocyte response and mast cell activity, explaining the improvement in clinical signs. It is possible that ciclosporin may improve epidermal innate immunity (Hendricks and others 2012) and at a low dose increase the numbers of T regulatory cells (Brandt and others 2009). This may be useful in preventing cutaneous infection and in the development of tolerance. Ciclosporin does not preclude intradermal allergen testing or in vitro allergen testing and so can be used in the face of severe disease before testing, while withdrawing from the effects of steroids if these have been administered previously (Goldman and others 2010).

In dogs, ciclosporin capsules are given at 5 mg/kg daily for around one month and then the dose reduced to every other day and then after a further one to two months to twice weekly in many dogs. Dogs that respond quickly in the first month are more likely to achieve the twice-weekly dose level. Although the licence recommendation is to give ciclosporin at least two hours before or after feeding, the clinical efficacy does not seem to be adversely affected by food (Thelen and others 2006). Ciclosporin has a relatively slow onset of action and pruritus may need to be controlled in the interim to ensure that the welfare of the dog is maintained using a short course of steroids as described by Dip and others (2013). The clinical signs of CAD are generally controlled by 21 days. Adverse effects of ciclosporin include: intermittent vomiting, diarrhoea, hirsutism, viral papillomata

and hyperplasia of the gums, but since its launch 10 years ago, ciclosporin has been shown to be a reliable and effective treatment for atopic dermatitis in dogs. Data from a large number of trials suggest that satisfactory control is seen in 36 to 100 per cent of cases with better than a 50 per cent reduction in clinical signs in 66 to 79 per cent of cases with adverse events reported in 14 to 81 per cent of subjects (Olivry and others 2002, 2010, Steffan and others 2003, Radowicz and Power 2005). Ciclosporin is often the treatment of choice in moderately to severely affected dogs with generalised disease.

Oclacitinib

Oclacitinib (Apoquel; Zoetis) has recently been approved in the USA and Europe to treat pruritus associated with allergic dermatitis (including atopic dermatitis). This new antipruritic drug acts by inhibiting Janus kinase 1, which alters the production of a variety of cytokines including IL-31. This interleukin has been implicated in the triggering of pruritus in people and in mice. In dogs, IL-31 has been used experimentally to induce pruritus and serum IL-31 concentration is increased in dogs with CAD in about 60 per cent of cases, whereas levels of this cytokine were below detection levels in normal animals (Gonzales and others 2013).

A recent study examined the efficacy and safety of oclacitinib in dogs with CAD, including atopic dermatitis, food allergy dermatitis, flea allergy dermatitis, contact dermatitis and scabies. Where indicated, treatment for fleas and scabies was also used. In this clinical study of 436 client-owned animals, the patient received either oclacitinib or a placebo for a minimum of seven days and the effects of the treatment were judged by owner and veterinary investigator. Treatment was considered successful in 67 per cent of cases treated with oclacitinib and 29 per cent of the placebo group (Cosgrove and others 2013). In the UK we currently have no experience of oclacitinib, but available information suggests that it acts quickly and effectively in a proportion of dogs. Additional data regarding its use in chronic disease and where there are complications of yeast or bacterial infection will be forthcoming and with this information we will have an additional agent in our arsenal of treatments for CAD.

Antihistamines

A variety of antihistamines are used in dogs. These are unlicensed and appear to have poor efficacy in the majority of cases.

Fig 5: Severe *Malassezia* infection in a West Highland white terrier, showing severe alopecia with lichenification over the neck and upper legs, with severe erythema, hyperpigmentation and many small crusts. There is considerable greasiness. This case was presented on oral prednisolone with a three-year history consistent with atopic dermatitis. The pyoderma and severe *Malassezia* dermatitis in this case required effective topical treatment to reduce *Malassezia* numbers and anti-staphylococcal antimicrobials before further anti-inflammatory treatment was given. Further steroid medication at this point may perpetuate the infection. The signs were brought under control using daily baths for 14 days before ciclosporin was introduced



Although not helpful for pruritus, chlorphenamine, hydroxyzine and cypripheptadine can be useful in cases that have urticaria.

Allergen-specific immunotherapy

Allergen-specific immunotherapy (ASIT) is the only therapy that acts specifically at the level of antigen-specific responses to reduce clinical signs. Despite its use over many decades, there are very few blinded and/or placebo-controlled studies to support the use of this therapy and in the UK there are currently no licensed products available, although marketing authorisation was granted for one product last year (Veterinary Allergenic Extracts for the Treatment of Atopic Dermatitis in Dogs, Vm 37026/4000, September 17, 2012).

Allergen injection immunotherapy is defined as 'the practice of administering gradually increasing quantities of an allergen extract to an allergic subject to ameliorate the symptoms associated with subsequent exposure to the causative allergen' (WHO Definition – Bousquet and others 1998) and this summarises the process well. Once a definitive diagnosis of CAD has been made, the allergens for inclusion in ASIT can be chosen by intradermal allergen testing or by in vitro allergen testing. The mechanism of action remains uncertain, but a diminution of the allergic response is accompanied by a reduction in allergen-specific IgE and an increase in IL-10 and regulatory T cells (T regs), suggesting that the response is due to the induction of allergen-specific tolerance (Keppel and others 2008). Interestingly, skin infection may reduce this T reg/IL-10 response indicating that good control of bacterial and yeast infections may be essential during ASIT. Of course, allergen

selection is key, and one well-designed study examining the efficacy of ASIT using *Dermatophagoides farinae* (house dust mite) proteins alone showed no better response than placebo (Willemse and others 2009).

ASIT is usually given by injection and depending on the manufacturer and whether an adjuvant is included, there is a wide variation in the interval between doses and the rate of increase of allergen administered. Regardless of the protocol used to increase the allergen dose, ASIT is slow to act, taking up to nine months to have an effect. The response is best judged at 12 months to avoid seasonal variation in disease confusing the interpretation of the response. In general, clients should be told to expect that 50 to 60 per cent of cases will be improved with only 20 per cent being solely reliant on ASIT. Efficacy has been measured in many different ways with similar results being seen with immunotherapy based on intradermal allergen and in vitro allergen testing (eg, Park and others 2000).

Few adverse side effects are noted with ASIT, but there is a potential for perturbation of the existing hypersensitivity resulting in oedema, swelling, pruritus and potentially anaphylaxis. In some dogs, needle shyness has been reported. This can be helped by allowing the syringe and its contents to come to room temperature before administration.

Recently, orally administered (sublingual) immunotherapy (SLIT) has been advocated and made available through some importers. There is only limited evidence for the efficacy of this technique and further placebo-controlled studies are needed to support its use.

Making treatment choices

In deciding what to dispense at the time of consultation, clinicians should use their

knowledge of the dog's dermatological history. In cases with a record of recurrent pyoderma, the emphasis should be on treating this first and subsequently avoiding treatments that make the patient more susceptible to infection. In such cases, steroids may not be as useful as baths that improve barrier function, ciclosporin and topical antiseptics. On the other hand, in cases where there is an acute increase in pruritus, but where infection is not a problem, steroids may be a good choice. Discovering a flare factor in the development of clinical signs in a case can be the key to preventing disease. Fleas, differing environments and stressful events can be triggers in worsening the signs of CAD. In many cases, combining therapy will allow you to minimise adverse effects while bringing the dog under their pruritic threshold (Figs 3, 4, 5).

It is useful when deciding upon treatment combinations to be guided by some principles. The first rule is that flea control should always be maintained in atopic dogs and that other animals in the house must also be treated. Secondly, bacterial and yeast infection should be identified promptly and addressed aggressively, but if these infections return, the underlying treatment must be reconsidered. Finally, CAD is not a disease for a one-off diagnosis and treatment plan, but needs to be regularly reassessed, the treatment modified and checked and the cooperation of the client maintained through good communication and regular contact.

In deciding upon treatments for CAD, consider each case as an individual, remain open to the possibility of a wrong or new diagnosis and repeat skin scrapings, parasite-specific serology and perform a thorough repeat clinical examination whenever things go awry.

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Competing interests

The author is a previous employee of Novartis Animal Health, he has been a consultant and/or CPD provider for Novartis Animal Health, Schering Plough, Fort Dodge, Merial, CVS and Vetruus.

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