Canine atopic dermatitis: ways of managing on a shoestring budget

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ABSTRACT

Canine atopic dermatitis (CAD) is a pruritic and inflammatory skin disease that can adversely affect the quality of life of pets and their owners. The pathogenesis is multifactorial, with allergic sensitisation, skin barrier defects and microbial and environmental factors being implicated. The available treatment options vary in their efficacy, adverse effects and expense, and the position of the UK economy has left many pet owners in financial hardship.

This article discusses an evidence-based, cost-effective approach to managing CAD for pets treated in charity veterinary practice. This includes ruling out flare factors that may exacerbate the patient's pruritus, such as ectoparasitic infestations, microbial infections and food allergy. This helps prevent the misdiagnosis of CAD and more efficient use of charitable resources when managing the disease.

A multimodal approach to cost-effective therapy is also discussed, using anti-inflammatory agents such as glucocorticoids, essential fatty acids (EFAs), antihistamines and shampoo therapy alone, or in combination. Glucocorticoids provide rapid relief from pruritus, with the drawback of several adverse effects. EFAs and antihistamines are less efficacious when used alone, but have relatively few adverse effects. Practitioners are reminded of the importance of client communication at each stage of the diagnostic and treatment process, as this will help contribute to the cost-effective management of the disease.

Canine atopic dermatitis (CAD; atopy) has been reported to be the second most common cause of pruritus in dogs after flea allergy dermatitis.

It has been defined as a genetically predisposed inflammatory and pruritic allergic skin disease with characteristic clinical features most commonly associated with IgE antibodies to environmental allergens (Halliwell, 2006). It is a disease that may be seasonal and will be associated with a range of clinical signs of varying severity.

It can have a considerable negative impact on the quality of life of affected pets and their owners, with up to 41% of owners in one survey falling into financial difficulty while having their pet treated (Linek and Favrot, 2010). In recent times of austerity, it is expected this figure may be higher and, as the general cost of living increases, many owners on low income face challenges between treating their pet or reserving finances for family and leisure. Furthermore, veterinary charitable organisations are facing an increasing strain on their resources and this can have the potential to considerably limit the scope of service they offer to clients.
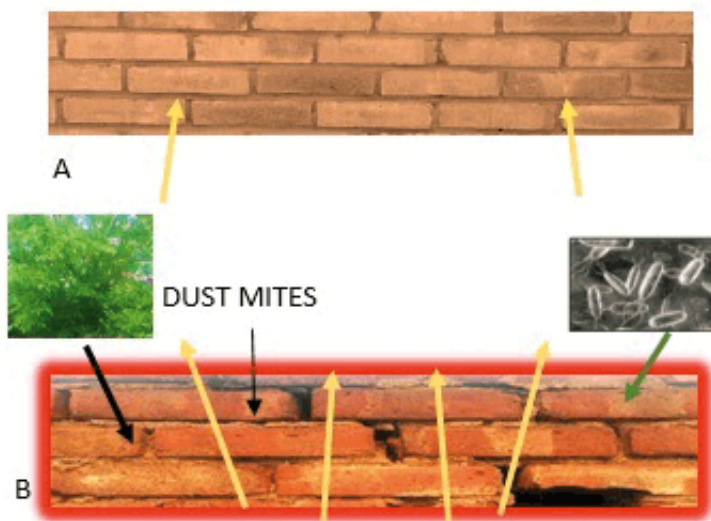

Figure 1. The canine stratum corneum in atopic dermatitis. **A**: an ideal skin barrier involves corneocytes (bricks) tightly adhered to each other by intercellular lipids (cement). **B**: in atopic dermatitis, abnormal lipid arrangement (the cement) causes a defective skin barrier, causing an increase in transepidermal water loss (yellow arrows). The abnormal skin barrier also facilitates the penetration of allergens (for example, the black arrows represent tree pollens and *Dermatophagoides* mites), which, in turn, further contribute to pruritus and erythema of the skin (red glow). Secondary microbial infections can follow (green arrow).

CAD can be a challenging condition to treat and this article discusses an evidence-based approach to improving the quality of life of dogs under charitable care.

The pathogenesis of CAD is considered multifactorial and, therefore, managing the disease in any patient is dependent on a multimodal approach. The pruritus and inflammation associated with a classical type one hypersensitivity reaction to environmental allergens can be augmented by skin barrier defects (**Figure 1**), whereby abnormalities in the stratum corneum lipids can lead to

increased transepidermal water loss with subsequent drying of the skin and pruritus. Microbial infections and environmental factors (Nuttall et al, 2013) are also contributory factors to the clinical disease.

A diagnosis of CAD can be supported by the application of Favrot's criteria, which are a set of clinical features that have been found to be associated with the disease. It must be noted, however, CAD is essentially a diagnosis of exclusion, whereby all other causes of pruritus (ectoparasite, microbial infection and cutaneous adverse food reaction; CAFR) that may produce similar clinical signs to CAD, have to be ruled out prior to making an initial diagnosis. Common sites of pruritus and clinical lesions include the concave aspects of the ear pinnae, face, axillae, ventral abdomen, interdigitae and perineum. For a further detailed discussion on the diagnosis of CAD, the reader is directed to standard dermatology texts, as well as an excellent publication by Hensel et al (2015).

Numerous options are available for managing CAD. Conventional measures include corticosteroids, essential fatty acids, antihistamines, allergen-specific immunotherapy, shampoo therapy and allergen avoidance. The use of immunosuppressive agents, such as ciclosporin A and oclacitinib, have increasingly been in vogue in recent years and, although reported to be highly efficacious, tighter budgetary constraints dictated by clients and charitable organisations keep the aforementioned therapies as the mainstay in their treatment protocols.

Corticosteroids

Corticosteroids are a well-recognised treatment option for CAD, particularly in the low-cost setting, with prednisolone and methylprednisolone being licensed for oral use in the UK. Prednisolone has been shown to have a rapid onset of action within four hours (Gadeyne et al, 2014) with a majority of owners estimated to report them, in the author's experience, as being very efficacious following the start of treatment. Studies have reported up to 70% (Ferrer et al, 1999) and 81% (Olivry et al, 2002) reduction in skin lesion and pruritus scores, respectively.

Guidelines for managing CAD suggest starting prednisolone/methylprednisolone at a dosage of 0.5mg/kg to 1mg/kg once daily, or divided into twice-daily dosing (Olivry et al, 2015). Once the pruritus and skin lesions are under control, this dosage can be tapered to once every 48 hours, with further 25% dose reductions if the patient remains in remission on the preceding alternate day dosage regime.

Corticosteroids are a very cost-effective tool in the management of CAD, with the owner/charitable organisation potentially saving £35 in the first month of treatment following diagnosis (cost saving based on comparing a four-week course of 1mg/kg [induction and tapered dosage] of prednisolone for a 10kg dog with a four-week course of ciclosporin A at 5mg/kg).

The adverse effects are attributed to their corticosteroid and mineralocorticoid effects. The most common clinical effects will include polyuria, polydipsia, polyphagia, weight gain, evidence of a

stress leukogram on haematological analysis (leukocytosis, lymphopenia, neutrophilia, monocytosis and eosinopenia) and an increase in serum hepatic enzyme levels (alkaline phosphatase and alanine aminotransferase). Dermatological effects may be manifested by the presence of a symmetrical non-pruritic alopecia, comedones, striae, seborrhoea sicca, recurrent pyoderma and calcinosis cutis.

During an initial course of treatment, clients commonly report a decrease in pruritus and erythema, and with the adverse effects having been communicated to the owner, steroids are usually well-tolerated on first use. However, given the recurrent nature of CAD in many patients, repeat courses of treatment with cumulative side effects form the focal point of frustration, especially in the charitable sector, as a limited budget may not permit the use of treatments that have less distressing side effects, such as ciclosporin A, oclacitinib and/or allergen-specific immunotherapy.

This is not an uncommon scenario whereby the owner will ask in frustration: "Is there anything else you can do for him/her? It is distressing to see him/her like this all the time."

Other management techniques that don't break the bank

The "summation of effect" should be considered when managing a case of CAD, based on the concept the atopic dog in your consult room may have additional diseases that will result in an exacerbation of its pre-existing pruritus. Not only does managing these causes – such as ectoparasites, infections and CAFR – help improve patient comfort, the stepwise approach also helps address the recurrent nature of pruritus in these patients, reduce premature prescription costs of antibacterial and/or immunosuppressive drugs and limit the number of recheck visits to the clinic – thus allowing charities to use their resources more efficiently.

Ectoparasites

Figure 2a. A dog affected by flea bite hypersensitivity with erythema and self-induced alopecia on the lumbar dorsum.

Figure 2b. A positive wet paper test showing digested blood diffusing out of flea faeces on contact with a wet surface.

In the author's experience, ectoparasites are frequent flare factors for pruritus in CAD patients in charity practice with fleas/flea bite hypersensitivity (FBH) being the most common (other ectoparasitoses that may be responsible for the flare in clinical signs – such as sarcoptic mange, cheyletiellosis, demodicosis, trombiculiasis and lice infestation – must also be ruled out in the diagnostic workup).

Historically, this was likely to have been attributed to clients not being able to fund the purchase of the more efficacious flea control products on a regular basis. In the modern-day competitive market, although previous POM-V drugs, such as fipronil and imidacloprid, are readily available (and cheaper), a lack of veterinary input at "general store" level often leads to inadequate flea control programmes being implemented in the home.

It is not uncommon for FBH patients to be presented at a chronic stage of the disease and the progression from a localised to a more generalised lesion distribution can potentially mislead the clinician into making a diagnosis of CAD. Briefly, the potential clues in the history that may allude to FBH include no flea control; the initial onset of pruritic behaviour mainly directed towards the caudal lumbar dorsum, medial thighs and ventral abdomen; and pruritus and papular skin lesions in other in-contact animals and humans.

Dermatological examination can often reveal the presence of live fleas, black flea dirt, an erythematous maculopapular eruption with variable scaling, crusting and self-induced alopecia (**Figure 2**).

Treating this flare factor should ideally be with a product that has a rapid "speed of kill" (which will reduce the exposure time to flea antigen), long duration of action and one that is easy to administer (improves compliance). Although a low budget can be a hurdle, spending time and educating owners can help them understand the importance of a rigorous flea treatment regime and, ultimately, will be cost-effective for them in the long term.

Spinosad and isoxazoline-based drugs, such as fluralaner and afoxolaner, are ideal as they are available in an easy-to-administer tablet form, have demonstrable 100% efficacy (Dryden et al, 2013 and 2015) and a long duration of action (Taenzler et al, 2014).

Microbial infections

Microbial infections are another flare factor that, if recognised and treated, may decrease the underlying primary pruritus to a level that is tolerable to both patient and owner and this may avoid the need for continuous steroid therapy or costly immunosuppressive therapy in some dogs. Achieving this in the low-cost setting can be economically challenging as funds for in-house cytology, culture and sensitivity, potentially protracted courses of systemic antibiotics and shampoo therapy may not be readily available.

The initial approach is to recognise and confirm the presence of a microbial infection. A full discussion of the clinical signs and diagnostic approach to bacterial pyoderma is beyond the scope of this article. The reader is directed to published guidelines on this aspect (Beco et al, 2013).

Surface, superficial and deep pyoderma associated with CAD may be manifested as acute pyotraumatic dermatitis (hot spot), folliculitis and interdigital furunculosis, respectively in the authors' experience.

Malassezia overgrowth is clinically similar to bacterial overgrowth syndrome (BOGS). The latter is a syndrome whereby there is pruritus in addition to erythema, greasiness, self-induced alopecia and malodour to the skin (**Figure 3**). The classical papular-pustular eruption and epidermal collarettes associated with superficial pyoderma are essentially absent in BOGS and the erythematous appearance of the skin can potentially be misinterpreted as allergic skin disease and get treated accordingly.

Diagnosis is by clinical signs and skin cytology. Tape strip examinations will reveal bacteria (usually groups of cocci, which are likely to be *Staphylococcus pseudintermedius*) with the minimal presence of inflammatory neutrophils. The number of bacteria suggested to be deemed pathogenic has varied between 5 (Colombo, 1997) to 15 cocci/high power field magnification (Viaud et al,

2012).

Figure 3a. A dog with bacterial overgrowth syndrome (BOGS). There is erythema, greasiness, malodour and self-induced alopecia localised to the ventrum, axillae, distal limbs and neck (not shown in this image).
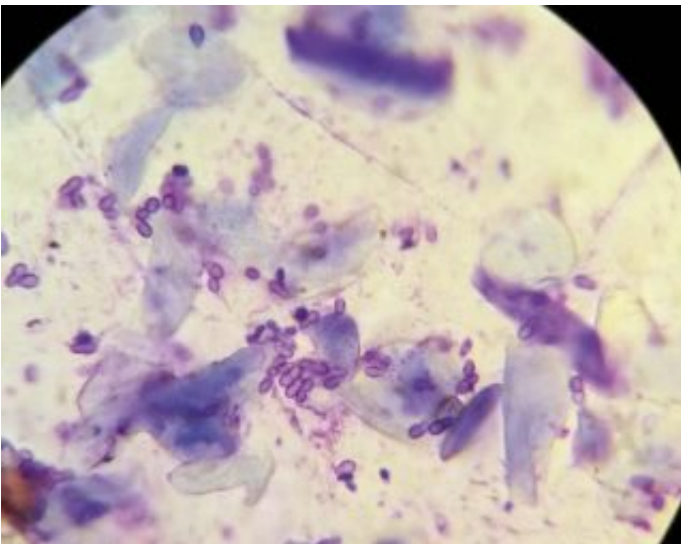

Figure 3b. Malassezia dermatitis may be clinically indistinguishable from BOGS, hence stressing the importance of cytology, which is a cheap and rapid diagnostic tool.

In the face of increasing multidrug resistance, the approach to treating microbial flares of pruritus should be via topical therapy. A 3% chlorhexidine shampoo has been shown to effectively reduce pruritus, bacterial counts and skin lesions three weeks after commencement of therapy for superficial pyoderma (Loeffler et al, 2011) with similar reduction in parameters also reported for treating *Malassezia* dermatitis (Maynard et al, 2011).

Shampoo therapy presents a considerable cost reduction for charitable clients, with a three-week course of 3% chlorhexidine shampoo therapy costing an average of £9 (for a 15kg to 20kg dog) in comparison to £22 with first-line systemic clavulanic-amoxicillin therapy. A further benefit of its use in CAD is any shampoo can potentially be antipruritic by washing off allergens, and by having a soothing and cooling effect.

As microbial infections resolve, clients can be recommended to taper the shampooing schedule from twice weekly to once weekly, then once every 10 to 14 days.

A common query by clients at this stage is whether money, as well as time and effort, should be spent on shampoo therapy once the infection has resolved. Reducing the risk of multidrug resistance, recurrent pyoderma and being economical for owners long term are common reasons mentioned to persuade owners to continue therapy. The drying effect of shampoos may be negated by the use of topical moisturising products.

Cutaneous adverse food reaction

CAFR is closely associated with CAD in that the clinical signs and lesion distribution can be identical. The former may have an earlier onset of disease than CAD and may also be associated with gastrointestinal signs. A dog suffering from CAD may have a CAFR as a concurrent source of pruritus all year round or as a flare factor. This is a very challenging area to investigate in charity practice as a homemade novel protein diet or commercial hydrolysed diet can incur significant costs to the client.

Furthermore, poor client compliance may preclude a strict diet trial from being adhered to for the suggested minimum period of eight weeks. The potential uptake of performing a diet trial in the low-cost setting is estimated to be approximately 5% of clients, in the author's experience.

If, after ruling out other flare factors, a patient continues to respond poorly to steroid therapy, CAFR is a possibility and clients should be advised accordingly. The reader is directed to the standard dermatology textbooks for further guidance on diet trials.

Multimodal therapy

To counter the frustration of recurrent pruritus and repeated steroid therapy, multimodal therapy is advised to aid in the management of the atopic patient. The following advice will discuss the cost-

effective options that may be feasible.

Essential fatty acid (EFA) therapy is based on the traditional concept that supplementation with a combination of omega-6/omega-3 fatty acids will help diminish pruritus and inflammation.

The most widely known mechanism of inflammation is via the arachidonic acid (AA) cascade (located in cell membranes) whereby the action of cyclooxygenase (COX) and lipoxygenase (LOX) enzymes will lead to proinflammatory prostaglandins (PG) and leukotriene (LT) formation. The sensitisation of peripheral nerves and an increase in vascular permeability are two of the many roles these mediators will play.

The omega-6 EFA, gamma-linolenic acid (GLA; found in borage and evening primrose oil) and omega-3, eicosapentaenoic acid (EPA; found in cold water marine fish oil) compete with AA for the COX and LOX enzymes, resulting in the formation of anti-inflammatory mediators, such as PGE and LTB5, respectively. These, as well as other mediators, help to modulate pruritus and inflammation.

From a practical point of view, it has been suggested 5ml of sunflower oil and 230g of dry food may help supplement a dog's diet with linoleic acid (Miller et al, 2013). This may help improve the skin and coat condition and also be very cost-effective for the client.

However, these benefits are offset by the conclusion from one set of guidelines that using EFA alone for treating CAD is unlikely to be of clinical benefit (Olivry et al, 2010).

Conversely, in a study by Saevik et al (2004), it was demonstrated the use of EFAs (in the form of borage and fish oil) in dogs with CAD halved their prednisolone dosage after two months' concurrent usage.

EFA therapy is unlikely to benefit dogs in the acute situation and this reflects the poor efficacy that may be reported by clients when they are used in this manner. The author recommends the use of EFA therapy at the commencement of long-term steroid therapy, and it would be reasonable to assess its efficacy after at least two months. Various EFAs are on the veterinary market and some products contain GLA of borage oil origin.

Antihistamines

Antihistamines are a favoured treatment among charitable clientele due to their low cost, wide availability from pharmacies and relative low incidence of side effects. The principal mechanism of their action is to block histamine-mediated pain and pruritus by occupying H1 receptors at the level of the skin and CNS. In one review, the authors concluded antihistamine therapy provided a small and limited benefit in the management of CAD with hydroxyzine – the metabolite of which is cetirizine – concluded to be the most beneficial (Olivry, 2015).

Despite limited evidence of their efficacy, some clients report a decrease in the severity of pruritus and skin lesions when using this drug class and, given the relatively good safety profile, they can be tailored into a low-cost treatment regime. They are best used before a pruritic event (to pre-occupy H1 receptors) and, in the case of seasonal atopy, clients may wish to commence therapy in advance of the pollen season.

The average monthly cost of treating a 20kg Staffordshire bull terrier with cetirizine at a dosage of 1mg/kg would be approximately £1.72.

Topical therapy

Shampoo therapy has traditionally been incorporated into CAD treatment plans and fulfils various roles, such as being antipruritic (by physically removing allergens from the skin surface; providing a cooling and soothing effect), antimicrobial and anti-seborrhoeic. Modern shampoos have ingredients incorporated in them that will aim to restore the skin barrier (for example, phytosphingosine, a ceramide precursor), thus reducing transepidermal water loss and drying of the skin (**Figure 1**).

Where possible, shampoo therapy should be an integral part of a patient's treatment regime. In the charitable setting, a single multipurpose shampoo would be cost-effective and may improve client compliance by reducing the need to remember regular tableting.

In reality, more than one treatment option is needed to help control clinical signs and shampoo therapy can be combined with any of the treatment options discussed. Should owners find themselves in extreme financial difficulty, reassure them washing with water alone may have some antipruritic effect in 50% of dogs, as was found in one study (Löflath et al, 2007). They should be advised, however, the effects of shampoo therapy will be short-lived and not curative.

Conclusion

CAD is a pruritic and inflammatory skin disease that can cause significant distress to both pets and their owners. In times of economic hardship, clients face a huge challenge between choosing to treat their pet or reserve finances for their own subsistence. With consideration to ruling out flare factors and adopting a multimodal approach to therapy, it is not impossible to considerably improve the quality of life for an atopic pet.

Regardless of treatment budget, it is paramount to communicate to the client this is, in a broad sense, an incurable disease and controlling the clinical signs may involve more than one therapeutic modality, and may potentially incur significant costs in the short to long-term future.

- All cost savings quoted in the article are based on average prices across several online pharmacies.

- **Some drugs mentioned are used under the cascade.**

Acknowledgement

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