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CASE REPORT

Companion or pet animals

Allergen-specific immunotherapy guided by skin prick testing in a Canadian shepherd dog with atopic dermatitis

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Abstract

This case report describes the successful use of allergen-specific immunotherapy guided by skin prick testing in a Canadian shepherd dog with atopic dermatitis. The dog presented severe pruritus, hypotrichosis, erythema and generalised scaling. Hyperpigmentation and lichenification were observed on the hindlimbs, thoracic and lateral abdominal regions. Sensitisation to *Cynodon dactylon* and *Lolium multiflorum* was identified, and allergen-specific immunotherapy was administered subcutaneously in three phases, with increasing allergen concentrations. Symptomatic treatment with oclacitinib and prednisolone was gradually withdrawn after allergen-specific immunotherapy initiation. The therapy resulted in clinical improvement, with complete control of pruritus and skin lesions. Allergen-specific immunotherapy proved to be an effective and safe causal therapy for canine atopic dermatitis, reducing the need for symptomatic drugs and improving quality of life.

KEYWORDS

allergy, environmental allergens, house dust mites, prick test

BACKGROUND

Canine atopic dermatitis (cAD) is a chronic, pruritic, inflammatory skin disease associated with epidermal barrier dysfunction, secondary to bacterial and yeast infections, and hypersensitivity to environmental and food allergens.[1] Reported prevalence varies considerably among studies, influenced by geographic region, study design, population characteristics and diagnostic criteria.[2,3]

Disruption of the epidermal barrier facilitates allergen penetration and microbial colonisation, promoting immune-mediated inflammation and pruritus. The multifactorial and dynamic nature of cAD pathogenesis often complicates diagnosis and long-term management.[4]

Diagnosis is based on compatible clinical signs and history, together with the exclusion of other pruritic diseases. Skin or serum test results should not be used to confirm the diagnosis of cAD, but rather to identify clinically relevant allergens for allergen-specific immunotherapy (ASIT).[5] Current therapeutic approaches aim to reduce inflammation and pruritus, control secondary infections, manage ectoparasites, and restore the epidermal barrier. Among these, ASIT remains the only causal and disease-modifying therapy, offering good efficacy and a low rate of adverse effects.[6]

ASIT involves the gradual administration of allergenic extracts to induce immune tolerance through upregulation of regulatory T cells and suppression of allergen-specific inflammatory pathways.[6] Treatment protocols should be individualised in terms of allergen concentration and dosing frequency, with clinical evaluation recommended after at least 12 months of therapy. Adjunct antipruritic medication may be necessary during the initial treatment phase.[7] Regular follow-up with a dermatology specialist is essential, and concurrent use of systemic glucocorticoids or cyclosporine should be minimised to optimise the immunomodulatory effect.[8]

Therefore, this case report describes the clinical response to ASIT guided by skin prick testing in a Canadian shepherd dog diagnosed with atopic dermatitis.

CASE PRESENTATION

A 3.5-year-old, entire, male Canadian shepherd dog was presented in January 2024 in the interior region of São Paulo State, Brazil. The main complaint reported by the owner was intense pruritus with a 6-month history of progression. At the initial assessment, the dog presented generalised hypotrichosis, erythema and desquamation. Pruritus was severe and self-induced, and its intensity was rated by the owner as 10/10 on the Pruritus Visual Analogue Scale. Although pruritus was generalised at presentation, the history of a 6-month progression suggests a chronic and previously uncontrolled

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FIGURE 1 First assessment. The patient exhibited generalised hypotrichosis, erythema and desquamation. Hyperpigmentation and lichenification were observed on the hindlimbs, thoracic and lateral abdominal regions.

disease course, which may have contributed to a more diffuse distribution of lesions over time, despite involvement of sites commonly associated with atopic dermatitis. Bilateral blepharitis, otitis externa and paw licking were observed, consistent with predilection sites described in cAD, along with chronic lesions such as hyperpigmentation and lichenification on the hindlimbs, thoracic and lateral abdominal regions (Figure 1). Also, the dog lived in a rural area with constant exposure to grasses and spent most of the day outdoors. The owner reported regular use of ectoparasitic preventives, specifically sarolaner (Simparic, Zoetis), and no evidence of flea or tick infestation was observed on clinical examination.

Dermatological screening tests included skin cytology, examination of skin scrapings and ear canal samples for parasites, and fungal culture. Cytology revealed *Malassezia* sp. overgrowth in both ear canals and a moderate presence of *Malassezia* sp. and coccoid bacteria on the skin. Parasitological and fungal cultures were negative.

Based on the clinical findings and history, symptomatic therapy for atopic dermatitis was initiated. The therapeutic regimen included oclacitinib (0.4 mg/kg, Apoquel) on alternate days, alternating with prednisolone (0.5 mg/kg, anti-inflammatory dose), followed by one drug-free day.[9] This treatment approach deviates from the labelled dosing recommendations for oclacitinib and was adopted due to financial constraints associated with the dog's bodyweight (40 kg). Prednisolone was included to achieve rapid control of severe pruritus and inflammation. This protocol reflects an individualised, constraint-driven decision and should not be interpreted as a standard or recommended regimen. Adjunct therapy consisted of bathing with chlorhexidine and miconazole shampoo (Cloresten) every 3 days and a hydrolysed soy-based diet. In addition, continuous ectoparasite control was maintained throughout the treatment period with sarolaner.

After 3 months, the owner reported approximately 70% improvement in clinical signs. A soy-based elimination diet was administered for 6 weeks with medical therapy, followed by 2 weeks with diet alone. Pruritus exacerbated after treatment withdrawal despite continued dietary adherence, indicating no clinical response and supporting atopic dermatitis without a food component.

LEARNING POINTS/TAKE-HOME MESSAGES

- The skin prick test effectively identified *Cynodon dactylon* and *Lolium multiflorum* as clinically relevant allergens.
- Allergen-specific immunotherapy led to complete control of pruritus and skin lesions, allowing withdrawal of glucocorticoids and oclacitinib.
- Early discontinuation of corticosteroids and regular clinical monitoring were key factors for therapeutic success.
- Findings reinforce the potential of prick testing and allergen-specific immunotherapy as safe, causal and long-term treatments for canine atopic dermatitis.

Given the dog's young age and persistence of residual lesions, allergen identification through a skin prick test was proposed to guide ASIT. Prednisolone was discontinued, and oclacitinib was administered daily for 30 days, being withdrawn 3 days before testing.

The skin prick test was performed using the UniTest PC device.[10] Saline and histamine (10 mg/mL) served as negative and positive controls, respectively. Environmental allergen extracts included house dust mites (*Dermatophagoides farinae*, *Dermatophagoides pteronyssinus*, *Blomia tropicalis*), storage mites (*Tyrophagus putrescentiae*, *Acarus siro*), and grass pollens (*Cynodon dactylon*, *Paspalum notatum*, *Lolium perenne*, *Lolium multiflorum*), each standardised at 10 HEP (10,000 BU/mL). All extracts and controls were produced by Salute Vita Laboratory and contained 50% glycerol and 0.45% phenol.

The UniTest PC device was applied perpendicularly, allowing capillary flow for 5 seconds, then pressed to produce a superficial epidermal abrasion. Reactions were read at 15 minutes, and erythematous wheals were demarcated and measured with a calliper. The reaction was considered positive if the mean diameter was 3 mm greater than the negative control.[11,12]

OUTCOME AND FOLLOW-UP

The test revealed positive reactions to *C. dactylon* and *L. multiflorum*. Based on these results, an individualised ASIT formulation was prepared by the same laboratory. The immunotherapy protocol was administered subcutaneously in three phases (Table 1). The patient was monitored according to the immunotherapy protocol, with evaluations performed at scheduled administrations.

After four allergen extract applications (during the induction phase), prednisolone was discontinued. After 32 weeks of immunotherapy, oclacitinib was also withdrawn. Weekly baths with chlorhexidine and miconazole shampoo and the hydrolysed soy diet were maintained.

Photographic documentation demonstrated significant clinical improvement after the introduction of ASIT, with notable reduction of pruritus and skin lesions (Figure 2). Nowadays, the patient is stable with a good quality of life.

TABLE 1 Allergen-specific immunotherapy protocol administered to a Canadian shepherd dog diagnosed with atopic dermatitis.

Phase	Dilution/Concentration	Injection schedule	Week	Injection volume (mL)	Frequency
Phase 1 Induction	1:100 dilution	Weekly injections with progressive dose increase	1	0.1	Weekly
			2	0.2	
			3	0.4	
			4	0.6	
			5	0.8	
			6	0.8	
Phase 2 Intermediate	1:10 dilution	Initially weekly, then biweekly, and finally monthly	7	0.1	Weekly
			8	0.2	
			9	0.4	
			10	0.6	
			11	0.8	
			13	0.8	Biweekly
			15	0.8	Biweekly
			19	0.8	Monthly up to Week 28
Phase 3 Maintenance	1:4 concentration	Initially weekly, then biweekly, and subsequently monthly	29	0.1	Weekly
			30	0.2	
			31	0.4	
			32	0.6	
			33	0.8	
			35	0.8	Biweekly
			37	0.8	Biweekly
			41	0.8	Monthly thereafter



FIGURE 2 Patient with significant clinical improvement after the introduction of allergen-specific immunotherapy, with notable reduction of skin lesions.

DISCUSSION

The skin prick test remains the most practical and standardised method for assessing allergen sensitisation.[13] Its use in veterinary medicine is expanding, offering a minimally invasive and reliable diagnostic alternative for identifying clinically relevant allergens in cAD. Supporting this, Sechi[14] demonstrated that the prick test detected mite sensitisation more frequently than the intradermal test, highlighting its higher sensitivity for certain environmental allergens. In this report, the prick test proved instrumental in guiding successful immunotherapy.

Sensitisation to environmental allergens, particularly pollens, has been extensively documented in cAD. According to Mueller et al.,[15] plant-derived allergens, such as grass, tree and weed pollens, sensitise approximately 10%–25% of affected dogs, with regional variations influenced by climatic and environmental factors. The patient described herein lived in a rural area with constant exposure to grasses, which plausibly explains sensitisation to *C. dactylon* and *L. multiflorum*. Consistent with this, Cerdeiro et al.[16] reported that 25.6% of dogs tested positive for *L. multiflorum*, *C. dactylon* and *P. notatum* in prick testing, reinforcing the relevance of grass pollens as key allergen sources in dogs. Similarly, Roussel et al.[17] found that 21.5% of dogs were positive for at least one grass pollen, with sensitisation persisting year-round, supporting the perennial nature of the disease as observed in the present case.

Dogs may develop immune responses to pollens from Pooideae grasses such as *L. multiflorum* and *L. perenne*, which are major contributors to pollinosis in Brazil.[18] *L. multiflorum* is typically a seasonal allergen, whereas *C. dactylon* and *P. notatum* are highly adaptive and invasive species prevalent in tropical climates,[19] with *C. dactylon* potentially acting as a near year-round allergen due to its continuous growth in warmer regions, supporting their clinical relevance in allergic sensitisation.

House dust mites, particularly *D. farinae*, *B. tropicalis* and *D. pteronyssinus*, are the most prevalent allergens implicated in cAD.[20,21] Storage mites such as *T. putrescentiae* and *A. siro* also play a significant role and may coexist in the environment and commercial dog food. Warmth and humidity

favour their proliferation and potential food contamination.[15] Co-sensitisation between house dust and storage mites is common and may result from concurrent exposure or cross-reactivity.[20]

In a large cohort study by Fennis et al.,[8] 664 dogs undergoing ASIT for at least 9 months were evaluated. An excellent or good clinical response was achieved in 59.9% of the cases, confirming ASIT as an effective long-term treatment for cAD. At the immunological level, Majewska et al.[22] demonstrated that ASIT induces beneficial modulation of lymphocyte subpopulations and cytokine profiles, including reduced IL-13 and TNF-α levels and expansion of regulatory T cells. These mechanisms likely underlie the sustained clinical improvement observed in the present case.

According to DeBoer,[23] ASIT should be valued within the broader therapeutic landscape of cAD, as it uniquely addresses the immunopathogenesis of the disease rather than merely suppressing symptoms. While conventional therapies provide temporary relief and often require lifelong administration, ASIT offers the potential for long-term immunological tolerance and, in some cases, disease remission. Thus, it remains a cornerstone in the multimodal management of cAD.

ASIT effectively controlled pruritus and cutaneous lesions, significantly improving the patient's quality of life. Consistent veterinary monitoring is crucial for therapeutic success. This case highlights the value of individualised immunotherapy protocols and supports the use of allergy testing, particularly the skin prick test, as a complementary tool in managing cAD. ASIT remains the only treatment capable of inducing long-term allergen tolerance and reducing the need for chronic symptomatic therapy.

AUTHOR CONTRIBUTIONS

Aléxia Vitória Rocha Dutra conceived and designed the project, acquired, analysed and interpreted the data, and wrote the paper. **Júlia Meira** and **Victória Gardinal Mazziero** drafted and revised the manuscript critically, conceived and designed the project, and wrote the paper. All authors have read and approved the final version of the manuscript.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

ETHICS STATEMENT

The authors confirm that the journal's ethical policies, as noted on the journal's author guidelines page, have been adhered to. No ethical approval was required as this is a case report.

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