Polish Journal of Veterinary Sciences Vol. 14, No. 2 (2011), 273-277

DOI 10.2478/v10181-011-0041-8

Original article

The study of canine atopic dermatitis involving the isolation of dogs

M. Fujimura

Fujimura Animal Allergy Hospital, 5-10-26 Aomatanihigashi,
Minou-city, Osaka, 562-0022, Japan

Abstract

Twenty-seven pruritic dogs were used in this study. When a hypoallergenic diet was fed to these 27 dogs for six weeks, none of the dogs showed improvement of the pruritus. These dogs had a history and clinical signs of atopic dermatitis (AD) as defined by Prelaud's diagnostic criteria. Subsequently, the 27 dogs were isolated for observation for two weeks in the hospital. In the isolation room in the veterinary clinic, cages and tableware were all stainless steel, and carpet was not used. A hypoallergenic diet was continuously fed to the 27 dogs for two weeks, during which time they were kept in the isolation room. PVAS (Pruritus Visual Analog Scale) was performed prior to starting the isolation, at the start of the study and 2 weeks after starting the isolation. In 17dogs (63%) the pruritus improved in the isolation room. A statistically significant reduction ($p<0.01$) of PLS (Pruritus liners score) was recorded 2 weeks after isolation. It was hypothesized that the 17 dogs whose pruritus improved in the isolation room had AD caused by an environmental antigen that was not present in the isolation room. Pruritus of the remaining 10 dogs (37%) did not improve. For 6/10 dogs, the intradermal allergy testing was positive for an environmental antigen. For4/10 dogs, the intradermal allergy testing was negative for all environmental antigens. Dogs for which sensitivity to an environmental antigen was not identified were thought to have atopic-like dermatitis.

Key words: atopic dermatitis, atopic-like dermatitis, isolation, dog

Introduction

The clinical signs of atopic dermatitis (AD) in humans and in dogs are variable, and there is no single physical or historical feature (DeBoer et al. 2001). In a recent study, the concept of human AD is explained as "extrinsic" and "intrinsic". Basically, "intrinsic" AD has the same symptoms as "extrinsic" AD, but no evidence of IgE involvement can be found in "intrinsic" AD (Brenninkmeijer et al. 2008). The ACVD (American College of Veterinary Dermatology) Task Force decided not to use the words "extrinsic" and "intrinsic" for dogs. Instead, the Task Force recommended "atopic dermatitis" for cases with positive allergy tests and IgE involvement, and for the cases with no positive allergy tests or IgE involvement, "atopic-like dermatitis" is the preferred term (Halliwell 2006).The aim of this study was to determine how many dogs improve when environmental antigens are intercepted by isolating of a general clinic to 27 dogs diagnosed with AD. Moreover, we determined whether atopic-like dermatitis existed in the isolated dogs that did not improve.

Correspondence to: M. Fujimura, e-mail: hope3413@gmail.com, tel.: +81 72 749 5225

Materials and Methods

Animals

The study was conducted over a period of six years (2001 to 2006) in Fujimura animal hospital (Osaka, Japan). Twenty-seven dogs presented with clinical signs of pruritus. These dogs had a history and clinical signs consistent with atopic dermatitis as defined by prēlaud's diagnostic criteria (DeBoer et al. 2001). The breed, sex, age, and age of disease onset of the 27 dogs are given in Table 1. Secondary infections such as bacterial pyoderma and dermatophytosis were treated. Diagnostic treatment using ivermectin or milbemycin oxime ruled out Scabiei.

Table 1. Characteristics and results of intradermal allergy testing of twenty-seven atopic dermatitis dogs.

No	Age years'	Age of onset "years"	Breed	Sex	Improvement in isolation	Intradermal	Allergy testing
						mumber of positivities	main antigen
1	3	1	Maltese	F	o	8	HDM
2	4	3	Shiba Inu	Fs	o	11	HDM – Grass
3	2	1	French Bulldog	Fs	o	4	HDM – Cotton
4	2	0.9	Shiba Inu	F	o	2	Grass
5	4	3	Cavalier King Charles spaniel	M	o	3	HDM
6	3	1	Wel ish Corgi	Mc	o	3	HDM
7	2	1	Toy Poodle	Fs	o	3	HDM – Cotton
8	3	2	Shiba Inu	Fs	o	8	HDM – Grass
9	3	3	Shiba Inu	M	o	6	HDM – Grass
10	3	2	Maltese	Mc	o	2	HDM
11	4	3	West Highland White Terrier	Mc	o	7	HDM – Grass
12	10	UN	Beagle	Mc	o	4	Grass
13	3	1	Labrador Retreiver	Mc	o	3	HDM
14	4	2	Maltese	Fs	o	1	HDM
15	1	0.7	Boston Terrler	F	o	2	HDM
16	2	1	West Highland White Terrier	M	o	2	HDM
17	4	2	West Highland White Terrier	M	o	2	HDM
18	5	1	Bernese Mountain Dog	M	o	4	HDM
19	5	1	Shiba Inu	Fs	×	2	HDM
20	8	3	Shiba Inu	Fs	×	3	HDM
21	6	2	Shiba Inu	F	×	4	HDM – Grass
22	7	5	Shiba Inu	M	×	7	HDM – Grass
23	5	0.5	Bull Terrier	M	×	2	HDM
24	5	1	West Highland White Terrier	F	×	0	–
25	9	UN	Shih Tzu	M	×	0	–
26	6	5	Miniature Dachshund	Fs	×	0	–
27	2	1	Chihuahua	Fs	×	0	–

o – Improvement; M – male; HDM – HOUSE DUST MITE
× – No improvement; F – Female; Mc – Castrated; UN – unknown; Fs – Spayed female; – – antigen negative

Intradermal allergy testing

Intradermal allergy testing was completed using 24 selected allergens. These were subdivided into six environmental antigen groups (mite mix; *Dermatophagoides farina, Dermatophagoides pteronyssinus,* dust, epithelia, trees, weeds, grasses, moulds) and flea antigen. The majority of commercial allergen preparations were purchased from Greer Laboratories (Lenoir, USA). The remainder (Japanese cedar) were obtained from Trii Medicine (Tokyo, Japan). The mixed house dust mite extract was used at a concentration of 1:50,000 wt/vol and 1:5,000 wt/vol. House dust extract was used at a concentration of 20 PNU/ml and other antigen were of a concentration of 10,000 PNU/ml. All extracts were prepared and diluted in sterile diluents. Dogs were premedicated with atropine (0.04 mg atropine sulphate per kg bodyweight subcutaneously) and sedated with xylazine (0.15 mg per kg bodyweight intravenously).

Dogs were placed in lateral recumbancy and the skin of the lateral thorax clipped.

Allergens were injected intradermally(0.05 ml of each extract was used).

Isolation and Concurrent Treatments

All owners agreed to hospitalization for two weeks. The 27 dogs were isolated in three kinds of cages: 0.70 m in length, 0.57 m in width, and 0.57 m in height for small dogs; 0.70 m in length, 0.57 m in width, and 0.78 m in height for medium dogs; 0.70 m in length, 1.20 m in width, and 0.78 m in height for large dogs. The floor, walls, and ceiling in the isolation cages were made of all stainless steel. The water and food containers were also made of stainless steel. Toweled pet sheets, newspaper, etc. were not used in the hospitalization isolation cages. The ventilation fan was not operated, and the isolation room was windowless. House dust mites were removed once each day, and the room was cleaned using a steam cleaner. Mite allergen in cages was detected once a week with monoclonal and polyclonal antibodies kit (DaniScan; Asahi, Tokyo). Neither steroid nor antihistamine therapy was used during hospitalization or during isolation. Each commercial hypoallergenic diet (Z/D ULTRA: a hydrolysate of chicken protein and corn starch or KO:Oat Floor and Kangaroo or FP:Catfish and Potato) was fed to these 27 dogs for six weeks before hospitalizetion and for two weeks in the isolation room.

PVAS: Pruritus Visual Analog Scale

A pruritus liners score (Olivry et al. 2002) was recorded on ascale of 0 to 5 as follows: 0 absence of pruritus. 1 almost no pruritus, 2 mild pruritus, 3 moderate pruritus, 4 severe pruritus, 5 extremely severe pruritus. Dogs were scored at three times in the study: prior to starting the isolation, at the start of the study and 2 weeks after starting the isolation.

Statistical Analysis

Statistical analysis was performed by use of a Wilcoxon signed-rank test and statistical significance was defined as $p<0.01$.

Results

The pruritus of 17/27 (63%) dogs kept in the isolation room improved (Table 1). Pruritus improved within three days for 10/17 dogs, and pruritus decreased even in slow cases (7/17) in one week. The pruritus of 10/27 (37%) dogs did not improve (Fig. 1). A significant decrease in PLS (Pruritus liners score) was found between prior to starting and 2 weeks after starting the isolation for the improvement group (Fig. 2; $P<0.01$, $N = 17$). No significant difference was found to in comparison to the no improvement group (Fig. 2; $P = 0.01$, $N = 10$). The intradermal allergy testing for environmental antigens was negative for four dogs within the group (ten dogs) that did not improve in the isolation room, while the test was positive for the other six dogs. These four dogs were not included in improve group in the isolation room.

Discussion

The clinical criteria for diagnosing AD are not perfect (DeBoer et al. 2001, Griffin et al. 2001). In addition, the Task Force defined two new terms: AD and canine atopic-like dermatitis (Halliwell 2006). AD is defined as an allergic skin disease of the prurigo related to the IgE antibody, meaning that a predisposition for AD is heritable.

Veterinarians generally think that IgE antibodies are activated in response to an environmental allergen (Halliwell 2006). Therefore, the improvement of AD by isolation in this study was assumed to dogs with AD consistent with the Task Force definition. The pruritus associated with AD disappeared in this group of dogs (17/27; 63%) because the environmental antigen had been removed. All dogs in this group had a positive skin test.

Interestingly, the percentage of dogs that showed improvement in isolation (63%) was very similar to the success rate of immunotherapy for AD that has been reported previously (Nesbitt 1978, Willemse et al. 1984, Nuttall et al. 1998, Saevik et al. 2002, Zur et al. 2002).

Ten of the twenty-seven dogs did not improve, and pruritus continued. This group in which pruritus did not improve was divided into two groups. In one group (6/10), the sensitivity to environmental antigens, as measured by a skin test was positive, just as it was for the group whose AD improved. The cause of pruritus in these groups kept in isolation is unknown. However, AD might be related to a concomitant food allergy (FA), which may explain why some dogs did not improve. The pathogenesis of FA is still unclear, and it has been hypothesized to be the result of type I, III, or IV immune reactions in humans (Goldstein et al. 1970, Chua et al. 1987, Halpern et al. 1987). The true cause of FA in dogs has not been completely elucidated (White 1986, Hillier et al. 2001). In a recent study, dog FAs were considered to be non-immediate (type IV), where as an earlier study demonstrated that dog FAs were immediate (Ishida et al. 2004). Therefore, a lymphocyte stimulation test in this group will be included in future studies for these reasons. For another group (4/10 dogs), the environmental antigen skin test was negative for all antigens. It is thought that these dogs have canine atopic-like dermatitis as defined by the Task Force (Halliwell 2006). Canine atopic-like dermatitis was defined as an inflammatory and pruritic skin disease with clinical features identical to those seen in

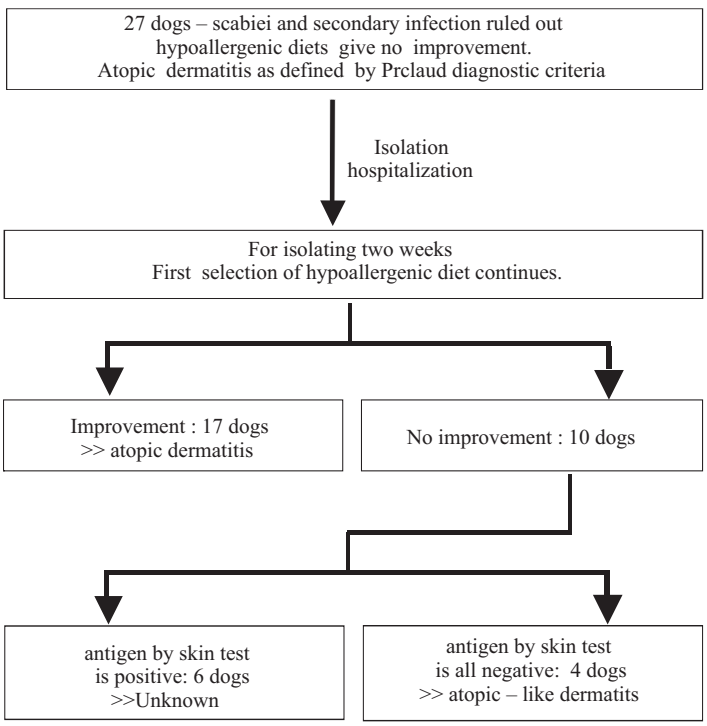

Fig. 1. Twenty-seven atopic dermatitis dogs in isolation.

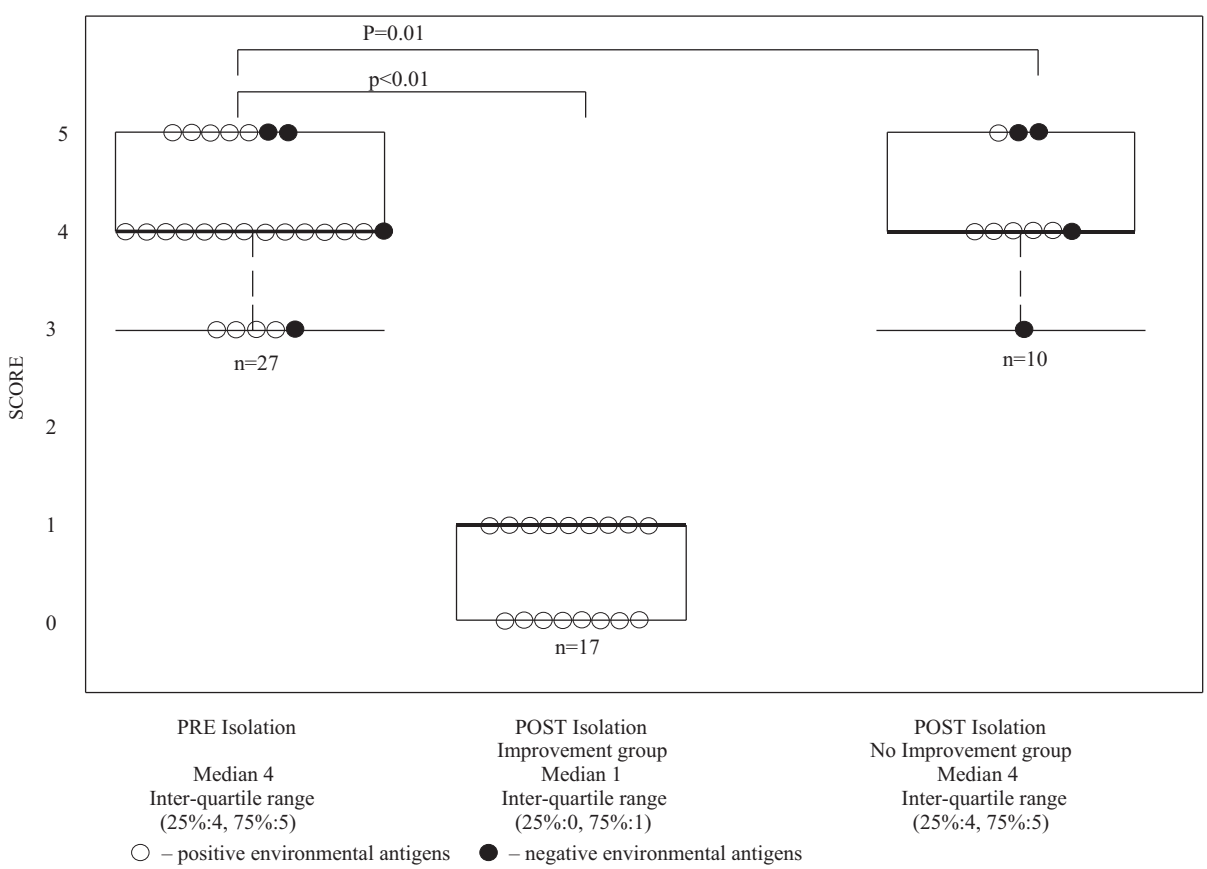

Fig. 2. PLS: Pruritus liners score

canine atopic dermatitis but in which an IgE response to environmental or other allergens cannot be documented (Halliwell 2006). In this study, the ratio of these groups was14% (4/27).

Canine atopic-like dermatitis is similar to "intrinsic" human AD, both of which have no evidence of IgE involvement (Brenninkmeijer et al. 2008). We expect to complete further studies on canine atopic-like dermatitis.

Acknowledgments

The author thanks Dr. Douglas J. DeBoer for suggestions on revising the manuscript.

References

Brenninkmeijer EE, Spuls PI, Legierse CM, Lindeboom R, Smitt JH, Bos JD (**2008**) Clinical differences atopic and atopiform dermatitis. J Am Acad Dermatol 58: 407-414.

Chua YY, Bremner K, Lakdawalla N, Llobet JL, Kokubu HL, Orange RP, Collins-Williams C (**1987**) In vivo and in vitro correlates of food allergy. J Allergy Clin Immunol 58: 299-307.

DeBoer DJ, Hillier A (**2001**) The ACVD task force on canine atopic dermatitis (XV): fundamental concept in clinical diagnosis. Vet Immunol Immunopathol 81: 271--276.

Goldstein GB, Heiner DC (**1970**) Clinical and immunological perspectives in food sensitivity. A review. J Allergy 46: 270-291.

Griffin CE, DeBoer DJ (**2001**) The ACVD task force on canine atopic dermatitis (XIV): clinical manifestations of canine atopic dermatitis. Vet Immunol Immunopathol 81: 255-269.

Halliwell R (**2006**) Revised nomenclature for veterinary allergy. Vet Immunol Immunopathol 114: 207-208.

Halpern GM, Scott JR (**1987**) Non-IgE antibody mediated mechanisms in food Allergy. Ann Allergy 58: 14-27.

Hillier A, Griffin CE (2001) The ACVD task force on canine atopic dermatitis (X): is there a relationship between canine atopic dermatitis and cutaneous adverse food reactions?. Vet Immunol and Immunopathol 81: 227-231.

Ishida R, Masuda K, Kurata K, Ohno K,Tsujimoto H (**2004**) Lymphocyte blastogenic responses to inciting food allergens in dogs with food hypersensitivity. J Vet Intern Med 18: 25-30.

Nesbitt GH (**1978**) Canine allergic inhalant dermatitis: a review of 230 cases. J AmVet Med Assoc 172: 55-60.

Nuttall TJ, Thoday KL, van den Broek AH, Jackson HA, Sture GH, Halliwell RE (**1998**) Retrospective survey of allergen immunotherapy in canine atopy. Vet Rec 143: 139-142.

Olivry T, Rivierre C, Jackson HA, Murphy KM, Davidson G, Sousa CA (**2002**) Cyclosporine decreases skin lesions and pruritus in dogs with atopic dermatitis: a blinded randomized prednisolone-controlled trial. Vet Dermatol 13: 77-87.

Saevik BK, Thoresen SI, Kristensen F (**2002**) A retrospective study of hyposensitization in canine atopy based on a polyclonal ELISA test. Vet Res Commun 26: 613-624.

White SD (**1986**) Food hypersensitivity in 30 dogs. J Am Vet Med Assoc 188: 695-698.

Willemse A, Van den Brom WE, Rijnberk A (**1984**) Effect of hyposensitization on atopic dermatitis in dogs. J Am Vet Med Assoc 184: 1277-1280.

Zur G, White SD, Ihrke PJ, Kass PH, Toebe N (**2002**) Canine atopic dermatitis: a retrospective study of 169 cases examined at the University of California, Davis, 1992-1998, Part II, Response to hyposensitization. Vet Dermatol 13: 103-11.