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Case report

Epidermal dysplasia and *Malassezia* infection in two West Highland White Terrier siblings: an inherited skin disorder or reaction to severe *Malassezia* infection?

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Abstract Two 9-month-old West Highland White Terrier siblings were referred to our clinic with pruritus, alopecia and lichenification. Cytological examination of Scotch™ tape strippings revealed Malassezia organisms and cocci. Skin biopsy specimens showed epidermal dysplasia. Treatment included bathing with a 2% miconazole/chlorhexidine-containing shampoo, orally administered ketoconazole (5 mg kg-1, every 12 h) and cloxacillin (25 mg kg-1 every 8 h). Six weeks later, the dermal infection had resolved and there was hair regrowth. However, the dogs were still moderately pruritic. Intradermal allergy testing was positive for house dust mites, storage mites and Malassezia. Immunotherapy was initiated, and treatment with ketoconazole and cloxacillin was stopped. Skin biopsies, which were performed in both dogs 4 months after the first presentation, revealed mild superficial perivascular dermatitis. The remaining mild facial pruritus was easily controlled with topical treatment. These two cases indicate that epidermal dysplasia might be an inflammatory or hypersensitivity reaction to the Malassezia infection or a result of excessive self-trauma, rather than a congenital keratinization disorder.

Keywords: dermal reaction to *Malassezia* infection, epidermal dysplasia, West Highland White Terrier.

INTRODUCTION

In 1989, Scott & Miller[1] described a severe chronic dermatosis with pruritus, seborrhoea and lichenification in eight West Highland White Terriers. It was presumed to be an inherited disorder of keratinization, with typical early onset in dogs between 2 and 6 months of age. It was named epidermal dysplasia with *Malassezia* infection, because of the very characteristic dysplastic changes seen in skin biopsy specimens and the associated *Malassezia* infection. This chronic skin condition is commonly referred to as 'Armadillo Westie Syndrome' or 'Westie seborrhoea', because of the numerous skin folds caused by lichenification.

Researchers have presumed that an inherited disorder of the epithelium predisposes the animal to *Malassezia* infection, and that the hypertrophic stratum corneum allows the invasion of yeast organisms.[1–4] Histological examination of skin biopsy specimens shows hyperplastic, superficial and perivascular dermatitis with marked abnormalities in keratinization.[1,4–6] Antifungal treatment to *Malassezia* infection provides some resolution of clinical signs, but relapses are frequent.

This study describes two young West Highland White Terrier siblings with clinical and histological skin lesions consistent with epidermal dysplasia and *Malassezia* infection. Whether these changes are an inherited keratinization disorder of the West Highland White Terrier or rather an inflammatory, or even hypersensitivity, reaction to severe *Malassezia* infection is further discussed.

CASE REPORT

Two 9-month-old West Highland White Terrier siblings (male and female) were referred to our veterinary teaching hospital with a 2-month-history of generalized pruritus, alopecia, lichenification and greasy, malodorous skin exudate. The referring veterinarian had suspected sarcoptic mange and treated both dogs with 300 μg kg^{-1} ivermectin, administered orally, once weekly for 6 weeks.

At admission, both dogs were almost bald and had severe generalized chronic dermatitis. The most

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Figure 1. Two 9-month-old West Highland White Terrier siblings at first presentation; both dogs have severe alopecia and severe generalized chronic dermatitis.

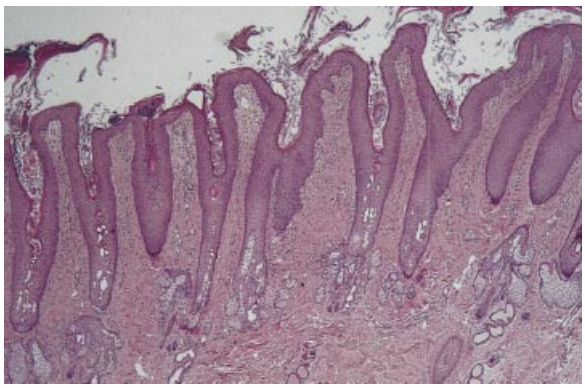

Figure 2. Skin biopsy from one of the dogs in Figure 1; epidermal dysplasia is evident. Haematoxylin and eosin stain, ×10 magnification.

prominent dermal changes were generalized erythema, greasy exudate, scaling and numerous folds, which were a result of extensive lichenification (Fig. 1). The head, legs and abdomen of both dogs were most severely affected and showed marked secondary hyperpigmentation. The female dog had a small number of papules and pustules, particularly on the ventral neck and chest. Both dogs had otitis externa, which was characterized by greasy ceruminous exudates and scaling. Microscopic examination of the exudates revealed numerous *Malassezia* organisms. In addition, the dogs had generalized peripheral lymphadenopathy and cytological examination of fine-needle aspirates revealed reactive lymphoid hyperplasia. Microscopic examination of skin scrapings for ectoparasites and dermatophytes was negative in both dogs. Fungal cultures on Dermatophyte Test Medium incubated at room temperature for 14 days did not change colour to red and were therefore interpreted as negative (BBL™ Becton Dickinson; agar slide with Trypticase® Soy Agar with chloramaphenicol, gentamycin and cycloheximide). Scotch™ tape strippings of the skin of both dogs yielded moderate numbers of *Malassezia* organisms, and cytological examination of the pustules revealed neutrophils with intracellular cocci. Serological examination for *Sarcoptes scabiei* was negative, and the results of a routine complete blood count and biochemical profile were normal in both dogs. Punch biopsies of the skin were performed on a hind limb, shoulder and lateral abdomen in both dogs. They revealed moderate parakeratosis and severe hyperplasia of the epidermis with diffuse spongiosis and lymphocytic exocytosis. There was irregular hyperplasia of hair follicle infundibula, and a superficial perivascular dermatitis, which consisted of infiltration of neutrophils, macrophages, plasma cells, lymphocytes and a few mast cells (Figs 2 and 3). No *Malassezia* organisms were observed in the biopsy samples. The histological findings were consistent with epidermal dysplasia of West Highland White Terriers.

Although, according to the literature, the prognosis of epidermal dysplasia is poor,[1,2,7] both dogs were treated with 5 mg kg^{-1} ketoconazole every 12 h, and

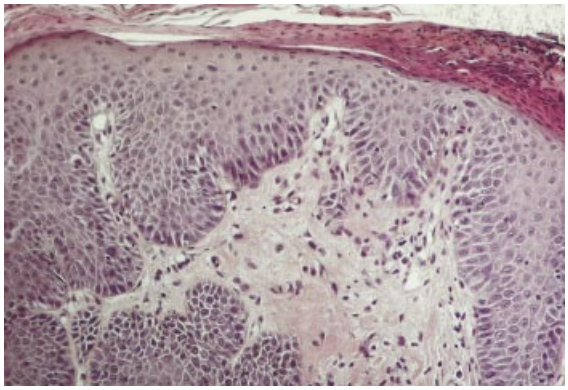

Figure 3. Higher magnification of skin biopsy in Figure 2 showing dysplastic epidermis and spongiosis. Haematoxylin and eosin stain, ×50 magnification.

2% miconazole/chlorhexidine shampoo every other day. The superficial pyoderma in the female dog was also treated with 25 mg kg^{-1} cloxacillin, every 8 h. In addition, both dogs were treated for fleas with fipronil spray and were fed a hypoallergenic diet based on capelin fish and tapioca (Waltham, Selected Protein®).

Three weeks after the first presentation, the skin condition of both dogs had improved markedly. There was regrowth of hair and moderate resolution of pruritus and scaling. The dogs were tested for atopy via analysis of serum samples for allergen-specific IgE using an enzyme-linked immunosorbant assay (ELISA), which measures monoclonal IgE antibodies (imovet CAC®). Both dogs were negative for indoor and outdoor allergens.

Six weeks after the first presentation, the skin of both dogs had improved further, such that intradermal allergy testing could be performed. Each dog was tested using 45 commercial aeroallergens from two different manufacturers (Trimedal AG and imovet bg), flea allergen, which is routinely used in our veterinary teaching hospital, and *Malassezia* antigen (500 μg mL^{-1} protein dissolved 1:1000 in NaCl, imovet bg). The aeroallergens consisted of dust and food storage mites, grass, tree and weed pollens indigenous to the geographical area and seven different moulds.

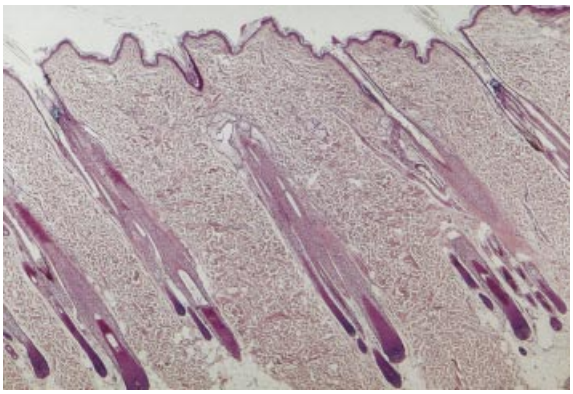

Figure 4. Skin biopsy collected from one dog 4 months after first presentation. The histological lesions comprising mild superficial perivascular dermatitis are consistent with a type-1 hypersensitivity reaction. Haematoxylin and eosin stain, × 10 magnification.

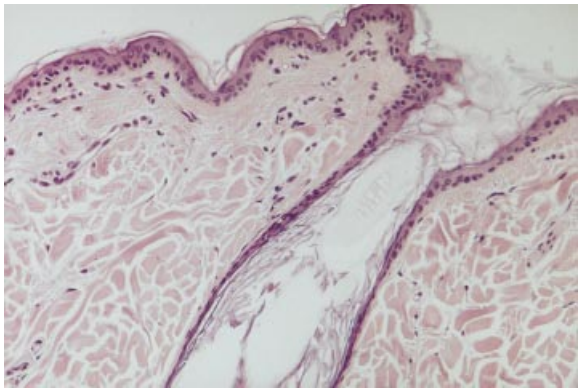

Figure 5. Higher magnification of the skin biopsy in Figure 4. The epidermis is now only mildly hyperplastic. Haematoxylin and eosin stain, × 50 magnification.

The results of allergen testing were the same for both manufacturers. Both dogs had positive reactions to house dust mites (*Dermatophagoides pteronyssinus*) and food storage mites (*Acarus siro*, *Tyrophagus putrescentiae*). The female dog also reacted positively to *Malassezia*. Based on these results, immunotherapy using aluminium-dissolved allergen extracts (imovet CAC®) was started. Both dogs received an allergen solution containing the mite extracts, and the female also received a separate allergen solution containing *Malassezia* extract. The manufacturer's hyposensitization scheme was used. Because cytological examination of Scotch™ tape stripping was negative for *Malassezia* and cocci, ketoconazole was reduced to 5 mg kg^{-1} every other day for another 2 weeks and treatment with cloxacillin was discontinued. The diet remained the same. After 10 weeks of treatment, shampooing was reduced to once weekly.

Four months after the first presentation, the skin of both dogs was almost normal. Skin biopsies at this time revealed mild epidermal hyperplasia and hyperkeratosis. In the superficial dermis there was mild and predominantly perivascular infiltration of lymphocytes and mast cells and mild activation of vascular endothelium. These lesions were consistent with a type-1 hypersensitivity reaction (Figs 4 and 5).

Six months after the first presentation, the skin of both dogs had improved further (Fig. 6). Pruritus resolved to only a slight residual face rubbing and paw licking. There was no change in the pruritus after several different food trials each over 8 weeks (two commercial hypoallergenic diets and a home-cooked diet based on horse meat and potatoes).

By 11 months after the first presentation, the skin condition in both dogs was controlled with monthly immunotherapy and shampooing with 2% miconazole/chlorhexidine every 2 weeks. To date, there has been no recurrence of *Malassezia* infection or other worsening of skin conditions.

The final diagnosis was atopic dermatitis with secondary *Malassezia* infection in both dogs and superficial pyoderma in the female dog.

Figure 6. The same dogs as in Figure 1 6 months after the initiation of treatment.

DISCUSSION

In this case report, two West Highland White Terrier siblings were presented with skin lesions typical for epidermal dysplasia. This skin condition was first described by Scott & Miller in 1989[1] and is characterized by severe chronic dermatitis with seborrhoea, lichenification and pruritus. A definitive diagnosis of epidermal dysplasia is based on the consistent finding of focal proliferation of epidermal and follicular basal cells with resultant loss of epidermal polarity (dysplasia) seen in skin biopsies and the cytologic finding of persistent *Malassezia* infection. It is presumed to be due to an inherited disorder of keratinization in affected West Highland White Terriers. The prognosis is guarded, because recurrence of *Malassezia* infection and deterioration of skin condition frequently occur.[1] In the case reported here, there were no clinical or histological signs of epidermal dysplasia after 4 months of therapy; thus, a congenital keratinization disorder was unlikely. Mason & Stewart[8] described *Malassezia* dermatitis in 21 dogs, in which skin biopsies showed histological changes characteristic of epidermal dysplasia as described by Scott & Miller.[1]

Malassezia spp. is a lipophilic budding yeast, which is potentially pathogenic. In healthy dogs, it is part

of the normal cutaneous microflora and commonly colonizes the external ear canal, anus, lips and interdigital skin.[9,10] *Malassezia* dermatitis can be primary,[8] but is more frequently secondary to an underlying problem such as ectoparasitism, allergic or seborrhoeic skin disease or endocrine disease.[8,11–14] Particular changes such as accumulation of moisture, disruption of the epidermal barrier and alterations in sebum quality allow the yeast to proliferate and become an opportunistic pathogen rather than a commensal organism.[11,14–16] Several breeds, including the West Highland White Terrier, Poodle, Basset Hound, Spaniel, Maltese Terrier, Dachshund, Shar Pei, Shih Tzu, Beagle, English Setter and German Shepherd Dog, appear to be predisposed to secondary *Malassezia* infection.[5,8,11,15,17]

Malassezia dermatitis is characterized by intense pruritus. Primary skin lesions are limited to erythema; however, secondary changes comprise alopecia, excoriations, seborrhoeic plaques, hyperpigmentation and lichenification. Histological findings in *Malassezia* dermatitis frequently include moderate to severe parakeratotic hyperkeratosis, irregular hyperplasia of the epidermis and follicular infundibula, diffuse spongiosis and lymphocytic exocytosis.[3,4,13,14,17] These lesions were seen in the skin biopsies in this report. *Malassezia* organisms may not be observed during histological examination of skin biopsies, because they are lost during processing, along with the superficial layers of the stratum corneum.[4,8,11,13,14,17] More reliable methods of detecting *Malassezia* organisms are Scotch™ tape stripping, direct impression smears or swabbing of the skin and culture.[10,14,16,18] In this report, *Malassezia* organisms were seen on Scotch™ tape strippings, but not in skin biopsies.

Based on the results of intradermal allergen testing and response to antifungal and hyposensitization therapy, we concluded that the primary skin disorder in the two West Highland White Terrier siblings was allergic dermatitis. In addition, the female dog had a wheal-and-flare reaction to intradermal *Malassezia* allergen. A type-1 hypersensitivity reaction to *Malassezia* has been described previously by Morris *et al.*[18] These authors reported that atopic dogs with no cytological evidence of *Malassezia* dermatitis had significantly smaller wheal-and-flare reactions to intradermal injection of a crude extract of *Malassezia pachydermatis* than atopic dogs with cytological evidence of *Malassezia* dermatitis. Based on histological and immunohistological studies, Maudlin *et al.*[17] suggested that *Malassezia* infection elicits delayed and immediate hypersensitivity reactions. There have been numerous reports of immediate and delayed hypersensitivity reactions to yeast antigen in human.[19–22] In this report, immunotherapy for *Malassezia* hypersensitivity appeared to be successful in the female dog, although it is not known whether the positive response was due to complete elimination of the *Malassezia* organisms with ketoconazole or due to immunotherapy for *Malassezia.* Furthermore, it is possible that successful immunotherapy against the other allergens was sufficient to control the clinical signs despite co-existent hypersensitivity to *Malassezia*. Further investigation is required to determine whether immunotherapy for *Malassezia* type-1 hypersensitivity is beneficial. Both dogs in this study had wheal-and-flare reactions to intradermal testing with allergens from two different manufacturers, but had negative results using the *in vitro* allergen test (imovet CAC®). Depending on the *in vitro* test used, the correlation between intradermal testing and different monoclonal *in vitro* allergen testing ranged from 52 to 98%.[23–25] The false-negative results might have been attributable to the relatively low sensitivity of the *in vitro* allergen test, which was 66% for all relevant allergens and 71% for mite allergen.[23] The success of immunotherapy in both dogs supports a true positive result of the intradermal testing.

The results of this report support the idea that epidermal dysplasia is not a primary congenital skin disorder, but a specific epidermal reaction to severe chronic dermatitis with secondary *Malassezia* infection.[4,5,8,26] The West Highland White Terrier and other breeds appear to have a predilection for *Malassezia* infection, secondary to a primary inflammatory skin disease.[14] Our results are in agreement with those of Lee Gross *et al.*,[4] who stated that severe chronic skin disease of any aetiology results in hyperplastic dermatosis. Severe pruritus leads to excessive skin damage, inducing epidermal hyperplasia as a mechanism of self-protection of the skin in order to improve the anchoring of the epidermis to the dermis by increasing the basal membrane zone surface. We conclude that epidermal dysplasia in the West Highland White Terrier is not a congenital defect of keratinization, but might be the consequence of a severe hypersensitivity reaction to *Malassezia* infection or secondary as a result of severe inflammation and excessive self-trauma.

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Résumé Deux chiens West Highland White Terrier, âgés de neuf mois, provenant d'une même portée, ont été référés à notre clinique pour prurit, alopécie et lichénification. L'examen cytologique de tests à la cellophane adhésive a montré la présence de *Malassezia* et de cocci. Les biopsies cutanées ont montré une dysplasie épidermique. Le traitement a consisté en des shampooings contenant de la chlorhexidine et 2% de miconazole, et en l'administration de kétoconazole (5 mg kg^{-1}, 2 fois par jour) et de cloxacilline (25 mg kg^{-1} trois fois par jour). Six semaines plus tard, l'infection cutanée était guérie, et le poil repoussait. Cependant, un prurit modéré était encore présent. Des tests intradermiques étaient positifs pour les acariens des poussières et de stockage et pour *Malassezia*. Une immunothérapie a été mise en place, le traitement au kétoconazole et à la cloxacilline étant stoppé. Des biopsies cutanées pratiquées chez les deux chiens 4 mois après la première visite ont montré une dermatite périvasculaire superficielle modérée. Il persistait un prurit facial modéré, qui a été contrôlé facilement avec un traitement topique. Ces deux cas indiquent que la dysplasie épidermique pourrait représenter une réaction inflammatoire ou d'hypersensibilité vis à vis de *Malassezia* ou être secondaire à des traumatismes répétés, plutôt que correspondre à un trouble congénital de la kératinisation. [Nett, C. S., Reichler, I., Grest, P., Hauser, B., Reusch, C. E. *Epidermal dysplasia and Malassezia infection in two West Highland White Terrier siblings: an inherited skin disorder or reaction to severe Malassezia infection?* (Dysplasie épidermique et dermatite à *Malassezia* chez deux West Highland White Terrier d'une même portée: désordre génétique ou infection grave à *Malassezia*?) *Veterinary Dermatology* **12**: 285–290.]

Resumen Se refirieron dos hermanos West Highland White Terrier de 9 meses a nuestra clínica con prurito, alopecia y liquenificación. El examen citológico de cinta adhesiva Scotch™ mostraron *Malassezia* y cocos. Las muestras cutáneas mostraron displasia epidérmica. El tratamiento incluyó baños con un champú con un 2% de miconazol/clorhexidina, ketoconazol oral (5 mg kg^{-1}, cada 12 h) y cloxacilina (25 mg kg^{-1} cada 8 h). Seis semanas más tarde, la infección dérmica había resuelto y se produjo crecimiento de pelo. Sin embargo, los perros mostraban

todavía un prurito moderado. Las pruebas intracutáneas de alergia fueron positivas al ácaro del polvo doméstico, al ácaro de almacén y a *Malassezia*. La inmunoterapia fue iniciada, y el tratamiento con ketoconazol y cloxacilina fue suspendida. Las muestras cutáneas, que se realizaron en ambos perros 4 meses después de la primera presentación, revelaron una dermatitis superficial perivascular leve. El resto de prurito facial leve fue controlado fácilmente con tratamiento tópico. Estos dos casos indican que la displasia epidérmica puede ser una reacción inflamatoria o de hipersensitibilidad a la infección por *Malassezia* o ser resultado de una auto-mutilación excesiva, más que un trastorno congénito de la queritinización. [Nett, C. S., Reichler, I., Grest, P., Hauser, B., Reusch, C. E. *Epidermal dysplasia and* Malassezia *infection in two West Highland White Terrier siblings: an inherited skin disorder or reaction to severe* Malassezia *infection?* (Displasia epidérmica e infección por *Malassezia* en dos hermanos West Highland White Terrier: un trastorno cutáneo hereditario o una reacción intensa a la infección por *Malassezia*?) *Veterinary Dermatology* **12**: 285–290.]

Zusammenfassung Zwei 9 Monate alte West Highland White Terrier Wurfgeschwister wurden mit Juckreiz, Alopezie und Lichenifikation in unsere Klinik überwiesen. Zytologische Untersuchung von Tesapräparaten ergab *Malassezia*-Organismen und Kokken. Hautbiopsieproben zeigten epidermale Dysplasie. Behandlung erfolgte mit 2% Mikonazol/Chlorhexidinshampoo und oralem Ketokonazol (5 mg kg^{-1}, alle 12 h) und Cloxacillin (25 mg kg^{-1} alle 8 h). Nach 6 Wochen war die Hautinfektion verschwunden und Haar wuchs nach. Allerdings zeigten die Hunde immer noch moderaten Juckreiz. Intradermaler Allergietest zeigte positive Reaktionen auf Hausstaubmilben, Futtermilben und *Malassezia*. Immuntherapie wurde begonnen und die Behandlung mit Ketokonazol und Cloxacillin beendet. Vier Monate nach der ersten Vorstellung durchgeführte Hautbiopsien ergaben leichte, oberflächliche, perivaskuläre Dermatitis. Der verbleibende Juckreiz im Gesicht wurde mit lokaler Therapie kontrolliert. Diese zwei Fälle deuten darauf hin, dass epidermale Dysplasie anstatt eines kongenitalen Keratinisierungsdefekts eine Entzündungs- oder Überempfindlichkeitsreaktion auf Malasseziainfektionen oder ein Ergebnis exzessiver Selbsttraumen sein könnte. [Nett, C. S., Reichler, I., Grest, P., Hauser, B., Reusch, C. E. *Epidermal dysplasia and Malassezia infection in two West Highland White Terrier siblings: an inherited skin disorder or reaction to severe Malassezia infection?* (Epidermale Dysplasie und *Malassezia* Infektion bei zwei West Highland White Terrier Wurfgeschwistern: eine ererbte Hautkrankheit oder eine Reaktion auf eine schwere Malassezia-Infektion?) *Veterinary Dermatology* **12**: 285–290.]