Case report

Cutaneous syndrome possibly caused by heartworm infestation in a dog

A SEAVERS
Oak Flats Veterinary Clinic, Oak Flats, New South Wales 2529

A 9-year-old German Shepherd bitch was presented with a recent onset of seborrhoea oleosa, hyperpigmentation, erythema, pruritus and alopecia along the neck, thorax, ventrum and the dorsal area of the carpus. The skin changes were believed to be caused by Dirofilaria immitis infection. A combination of topical and parenteral anti-heartworm therapy led to the resolution of the lesions.

Key words: Dog, skin, Dirofilaria immitis.

Halliwell1 noted the presence of a highly pruritic condition resembling Sarcoptes scabiei in heartworm endemic areas as a potential indicator of Dirofilaria immitis infection. According to Arwel1,2 dogs with severe heartworm disease may have obvious skin changes such as a dry, dull or scurfy coat. Muller et al1 described numerous rare skin disorders associated with D immitis and suggested the pathogenesis to be a hypersensitivity to the microfilariae. The cutaneous lesions included a pruritic, papulocrustaceous dermatitis resembling canine scabies or an erythematous, alopecic dermatitis of the chest and limbs, and/or seborhoeic skin disease. Mozos et al4 documented five cases in which D immitis was implicated, with generalised erythema, depilation and papules, mostly of a ventral distribution with some facial involvement and moderate to severe pruritus.

The dog had been presented annually for vaccination from 4 months of age and no skin disease had been noted previously. The animal had also been dewormed regularly with praziquantel and febantel.

Microscopic examination of multiple superficial and deep skin scrapings did not reveal evidence of ectoparasites. Financial constraints prevented any further investigation at this stage. In view of the poor retrieval rate of Sarcoptes scabiei on scrapings, the age of the animal and the possible exposure to the local indigenous wildlife population, which itself is frequently infested with Sarcoptes spp, a provisional diagnosis of Sarcoptes infestation was made. Generalised Otodectes was considered unlikely given the negative skin scrapings and the absence of mites or exudate on otoscopic examination.

A regimen of reducing environmental contamination was instigated along with two washes with a shampoo containing selenium sulphide, chlorhexidine and miconazole (Sebolyse®, Dermcare-Vet, Queensland) 3 days apart, followed 28 h later with a 6-week course of weekly phosmet dips (Vet-Kem Insecticidal Rinse, Zocon-Pitman Moore Australia). At the end of the 6 week course, the owner was instructed to apply the shampoo once to twice weekly to help normalise the microenvironment of the skin. Glucocorticoids were not prescribed.

The dog was rechecked after completion of the 8 week course and was much improved with regard to the erythema and hyperpigmentation. The seborrhoea oleosa appeared to be controlled by the twice weekly shampooing. The owner was keen to continue with the shampoo as needed, along with monthly phosmet dips as an added preventive measure against re-exposure or reinfection with what was thought to be a sarcoptic mange mite.

The dog was presented 4 months later with worsening seborrhoea, hyperpigmentation and lichenification. The owner had increased the frequency of the treatment with insecticide when he became concerned about reappearance of erythematous seborrhoea and hypotrichosis. Clinically, the chest and abdomen were hyperpigmented, there was seborrhoea oleosa and hair could be easily epilated. Pruritus had increased. The dog had recently been in oestrus. Microscopic examination of multiple skin scrapings again failed to reveal mites.

In view of the possible link between generalised skin disease and heartworm disease, the dog was given a 50 μg/kg dose of ivermectin (Ivomec, MSD-Apvet, New South Wales). The owner was warned of the risks1 and that no recommendation or claim is made by the manufacturer with respect to ivermectin as a microfilaricide. The owner was also advised to continue twice weekly shampooing.

Two weeks later, there was an appreciable regrowth of hair and seborrhoea oleosa had lessened. Given the rapid
response to the low ivermectin dose, the index of suspicion rose that *D. immitis* was the primary pathogen. A *D. immitis* antigen blood test was performed and a Vet-Red test (Canine Antigen Test kit, Agen Biomedical Ltd, Brisbane) recorded a strong positive result. Microfilariae were not demonstrated on a Difil test (Difil Heartworm Test Kit, Apex Lab, New South Wales) before adulticide treatment. Operator error may have been a factor. However, in about 20% of dirofilariaisis cases, microfilariae cannot be demonstrated due to an immune-mediated reaction against microfilarial antigen. In addition, papulonodular dermatitis has been recorded in a dog with occult dirofilariaisis. A biochemical profile was within normal values. Melarsomine dihydrochloride (Immiticide®, A Webster, New South Wales) 2.5 mg/kg was administered intramuscularly on alternate sides 24 h apart and 0.22 mg/kg prednisolone given per os twice daily.

At follow-up 6 weeks later, hypotrichosis and hyperpigmentation were effectively resolved. Ivermectin was again given as a microfilaricide and the administration of prednisolone gradually ended. At follow-up 5 weeks later, the shampoo and an essential fatty acid and Zn supplement were dispensed to treat some mild residual seborrhoea and erythema.

The dog was finally seen 5 months later for routine vaccination. She had been off all medication except a once monthly ivermectin dose (272 µg) as a heartworm preventative. There was total resolution of the previous dermatological abnormalities and the dog appeared clinically normal.

The dog was euthanased 2 months later for a pyometra which the owner declined to have treated. The skin and coat at the time appeared normal.

**Discussion**

The historical and clinical features of this case are suggestive of a *D. immitis* infestation manifesting as a cutaneous syndrome. There had been no change in diet, housing or behavioural patterns. While no lethargy, exercise intolerance or cardiopulmonary signs were ever reported by the owner, the possibility that heartworm infestation played a role in the pathogenesis of this dog’s condition cannot be discounted.

There is a possibility the dog was atopic and that prednisolone achieved prolonged remission (6 months) after 2 months of treatment. However, the mature age of onset of signs in an animal which had spent its entire life in the area reduces the likelihood of late onset allergic skin disease. Over 75% of cases of canine atopy exhibit clinical signs by age 3 years and a first incidence after 6 years of age is extremely rare (E Rosser unpublished). The animal’s condition was not monitored for a full year after completion of treatment, but it went through a subsequent winter without recurrence of dermatoses. Winter is in itself an unusual season for presentation, as 80% of atopics exhibit their first signs within a spring to autumn range. The dog was kennelled outside and whilst the house dust mite may survive in a kennel environment (REW Halliwell unpublished), one would not have expected a response to the treatment given. Treatment with prednisolone was initiated 2 weeks after the first ivermectin dose, when clinical improvement had already been observed. There was never any cheilitis, rhinitis or conjunctivitis nor xerosis of the skin.

Contact allergens such as carpets, powders, rubber, disinfectants and wandering jew (*Tradescantia albiflora*) were discounted due to the generalised distribution of the lesions and the failure of resolution with specific topical treatment and appropriate avoidance therapy. Vesicles that could be suggestive of irritant contact dermatitis were not observed.

There had been no change to the original diet throughout the 17 months so improvement could not be ascribed to a dietary change. A percentage of food allergic cases may respond well to glucocorticoid therapy but if this were the case, one would have expected a relapse after cessation of prednisolone.

Whilst hormonal hypersensitivity was always a consideration in an intact bitch, she experienced two further oestrous cycles without deterioration of the skin.

Dosages of ivermectin as low as 50 µg/kg can have some effect against *Sarcoptes* spp but there can be a lag period of 3 weeks before it achieves its full effect. A follow-up dose 2 to 3 weeks later is also routinely advised. However, *S cati* infestation often exhibits a more intense pruritus than described herein. The usual predilection sites of the ears, elbows and axillae were relatively normal. Lymphadenopathy and weight loss sometimes associated with *Sarcoptes* infestation were not found, and no animals or humans in contact with the dog demonstrated skin disease.

A recent report suggests *D. immitis* is a common and important parasite of dogs in certain areas, including Sydney. The likelihood of acquiring infection with heartworm increases with the period of exposure to the intermediate host. The dog lived adjacent to a large lake which harbours a heavy mosquito burden, yet she had never been on heartworm prevention. The dog had been healthy prior to the onset of dermatoses at a mature age.

The initial response to the combination of topical therapy may have been an illustration of the ‘pruritic threshold’ theory, whereby in the early stage of the disease with low microfilarial numbers, any therapy that returned the microenvironment of the skin to normal would give an initial corresponding external clinical improvement. The shampoo used here reduces populations of *Malassezia pachydermatis* and potentially pathogenic bacteria such as coagulase-positive staphylococci on the skin. The shampoo improved the condition initially but, once the dermatoses became severe, it was no longer effective. However, once the *D. immitis* was eliminated, the shampoo again began to exert an effect. Thus, the initial response to therapy may have been one of controlling the secondary and not the primary pathogen.

Alternatively, the initial dermatitis seen in July may have been due to the migrating larvae rather than the microfilariae. A lag period may have then ensued giving a clinical cure that relapsed upon production of the microfilariae some months later. The time phase from initial presentation to relapse was 7 months which fits the prepatent period of *D. immitis* of 6 to 7 months. It is conceivable that the shampoo and the insecticide may have
had some efficacy against the cutaneous stages but the topical therapy may have been entirely coincidental.

Marked suppression of microfilariae following a single 250 μg/kg dose of ivermectin occurs within 18 h, whilst 90% of animals treated with 50 μg/kg of ivermectin are negative for microfilaria within 3 weeks of a single dose. The animal in this case responded rapidly in the first 10 to 14 days and did not receive any further ivermectin treatment until 8 weeks later.

It is important to be aware of the possibility of skin disease secondary to *D immitis*. The diagnosis in this case is somewhat retrospective. However, for general practitioners in endemic areas the link between the pruritic dog and an endemic parasite other than a mite as a possible cause should not be overlooked. Because the mechanism of the cutaneous syndrome is that of a hypersensitivity reaction, the true incidence may always remain sporadic even in heartworm endemic areas.

I suggest a possible basic protocol to elucidate more information:

1) Take multiple superficial and deep skin scrapings in liquid paraffin and acetate. Microfilariae have been demonstrated on microscopic examination of skin scrapings and pus from a pustular follicular eruption on the limbs of a dog with microfilariasis.

2) If the scrapings do not reveal microfilariae or mites, test blood for the presence of microfilariae and antigen *D immitis*. In view of the poor retrieval rate of *Sarcoptes* on scrapings and the subsequent empirical use of ivermectin, testing blood for heartworm should be a routine part of a work-up before beginning treatment for mite infestations in cases not covered by effective heartworm prevention. Though rare, reactions, sometimes fatal, may occur after ivermectin treatment of microfilaraemic dogs.

3) Regardless of whether step 2 is negative or positive, offer the client the option of a biopsy to be taken. At the very least a full blood count should be performed as haemograms will often reveal an eosinophilia. The biopsy may reveal the presence of missed *Sarcoptes* mites or other conditions all of which contribute to the more accurate diagnosis and treatment of the itchy dog. A positive biopsy characteristically demonstrates a ppy granulomatous superficial and deep angiocentric dermatitis coupled with the presence of both intravascular and intragranulomatous microfilariae. An early biopsy may reveal the significance, if any, of the larve in this condition.

4) There are many and varied treatments for heartworm infestation but given that the current opinion is that the cutaneous lesions are a type II hypersensitivity to the microfilariae, attention must be given to eliminating both the production and presence of the microfilariae. In cases where adult positive antigen and microfilarial blood tests were validated on biopsy and subsequently treated with an intra-venous sodium thiacetarsamide and subcutaneous ivermectin protocol, total resolution of pruritus and dermatitis was recorded. Melarsomine dihydrochloride would be an alternative adulticide.

5) Topical adjutant therapy such as the use of the shampoo and an essential fatty acid and Zn supplement may be an option to help speed the normalisation of the microenvironment of the skin.

In conclusion, the most relevant fact in this case is a long exposure of the unproctected animal to the intermediate host of *D immitis* in what is a locally known area of heartworm infection. The mature age of onset, the findings in routine biochemical tests and negative skin scrapings, the geographical location and the rapid improvement with a low-dose of ivermectin in a heartworm-positive animal suggests that this was a case of a cutaneous manifestation of *D immitis* infestation.

Acknowledgments
I thank Dr Ralph Mueller for his advice and Dr Mark Weingarth for assistance with case management and computer typesetting.

References

(Accepted for publication 13 October 1997)