DERMATOLOGY CLINIC FOR ANIMALS

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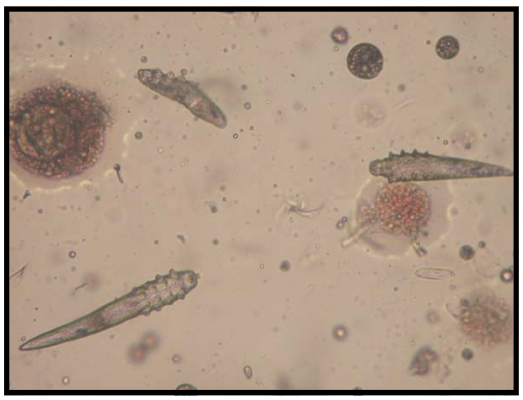
Dermatology Discourse

Canine Demodicosis

Signalment and History

"Buffy", a 10 year old spayed female Shih Tzu, presented for a 6 month history of mildly pruritic patchy alopecia and scaling of the face, legs and truncal regions which had failed to respond to several courses of antibiotics or steroid injections. She had also become lethargic the last few months.



D. canis mites and nymphs

Differential Diagnoses

The patchy alopecia and scaling were consistent with a folliculitis (bacterial infection, demodicosis, dermatophytosis), and/or malasezzia dermatitis possibly due to an underlying endocrine disease (hypothyroidism, iatrogenic or endogenous hyperadrenocorticism) or hypersensitivity (atopy or food allergy). Less common causes of alopecia and scaling included neoplasia (cutaneous T cell lymphoma), and sebaceous adenitis.

Laboratory Findings

Dermatitis on the neck of a dog with demodex.

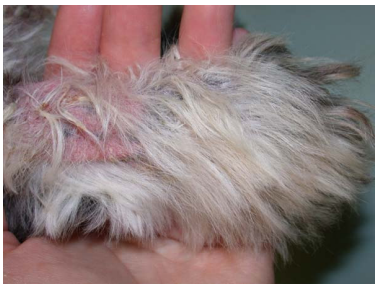
A dry skin scraping for cytology revealed occasional neutrophils with scattered intracellular and extracellular cocci bacteria. A dermatophyte culture was negative. Deep skin scrapings revealed demodex mites in all life stages (adults, larvae, nymphs and eggs). Full bloodwork was performed. The complete blood count revealed mild normocytic, normochromic anemia. The chemistry panel revealed mildly elevated ALP consistent with prior steroid therapy and a mild hyperglobulinemia. A thyroid profile revealed a low total T4, low free T4 by equilibrium dialysis and a high TSH level consistent with the diagnosis of hypothyroidism.

Treatment

Buffy was started on cephalexin (22 mg/kg PO BID for 3 weeks) to treat the bacterial folliculitis. Thyroid supplementation was initiated with Soloxine at (0.1 mg/10 lbs PO BID). The demodicosis was treated with 1% ivermectin at a gradually increasing dosage regimen beginning at 0.1 mg/kg PO once daily over 2 weeks reaching a final treatment dosage of 0.5 mg/kg PO once daily.

Follow Up

Buffy was rechecked at 4 week intervals and skin scrapings were repeated at each visit to ensure the demodectic mites were dead and decreasing in number. A total thyroid level 4-6 hours after the morning dosage was checked to ensure it was at therapeutic levels. After 4 months, complete hair regrowth had occurred at all sites and Buffy had become increasingly active. Her pruritus also resolved as infections were treated. Ivermectin treatment was discontinued once all clinical lesions had resolved

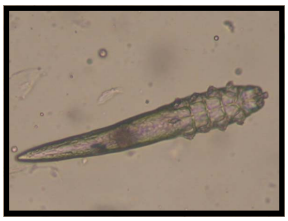


Pododemodicosis with alopecia and erythema.

and two consecutive skin scrapings, 4 weeks apart, were negative. Follow up skin scrapings 2 and 6 months later continued to be negative for mites.

Clinical signs

Demodicosis is an inflammatory parasitic skin disease. *Demodex canis* (a normal commensal of a dog's skin and ear canal, occupying the hair follicles, sebaceous ducts and sebaceous glands) is the most common causative agent in dogs. *D. injai*, a long-bodied mite which also found in hair follicles, and an unnamed short-bodied *Demodex* mite which lives in the stratum corneum are rarer causes of skin disease in dogs.



Demodex canis

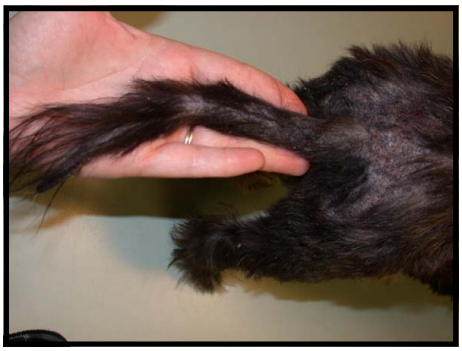
In dogs, demodex infections can present as localized (more common in puppies 3-6 months old) or generalized (juvenile-onset or adult-onset) disease. Juvenile-onset generalized demodicosis occurs in young dogs, between 3-18 months of age, most commonly in medium to large purebred dogs. Adult-onset generalized demodicosis occurs in dogs greater than 18 months of age, with the highest incidence in middle-aged to older dogs that are immunocompromised due to an underlying condition such as hypothyroidism, iatrogenic or acquired hyperadrenocorticism, diabetes mellitus, immunosuppressive drug therapy or neoplasia. Demodicosis lesions begin as patchy alopecia with variable erythema, scaling, and papules. Pruritus is variable. With chronicity, the affected skin may become hyperpigmented, lichenified, pustular, eroded and/or ulcerated from a secondary superficial or deep pyoderma. Lesions can occur anywhere on the body, including the feet, and severe pododermatitis is not uncommon. Peripheral lymphadenopathy can be observed. Differential diagnoses for canine demodicosis include superficial or deep pyoderma, dermatophytosis, hypersensitivity (flea allergy, food allergy, atopy), and pemphigus foliaceus.

Diagnosis of demodicosis

Multiple skin scrapings (superficial and deep) in mineral oil should be performed and analysed microscopically under 4-10X for the presence of demodectic adults, nymphs, larvae, and /or ova. It is important to scrape deeply (capillary oozing on the dulled scapel blade), or mites may be missed. Although *D. canis* is a commensal organism, it is very unusual to find demodex mites in scrapings from normal dogs, so finding one mite is suspicious for infection and indicates the need for more scrapings. In infections with *D. injai*, only 1-2 mites per field may be noted. Cutaneous biopsies may be needed to identify the organism in cases of canine pododemodicosis where tremendous granulomatous inflammation and fibrosis can occur.

Treatment of demodicosis

In terms of therapy, always identify and treat any underlying immunosuppressive diseases. All intact dogs with generalized demodicosis should be neutered, especially intact females, as pregnancy or estrus may stimulate a relapse. Predisposition for generalized demodicosis also seems to have a genetic basis in dogs, so affected animals should not be bred. Localized demodex lesions may resolve spontaneously. If necessary, localized lesions in dogs can be treated with Rotenone (Canex) or 0.025% amitraz solution daily. Traditional miticidal therapy for generalized demodicosis in dogs entails clipping medium- to long-coated breeds, bathing in 2.5-3% benzoyl peroxide shampoo, and then following up with a total body application of 0.025%-0.05% amitraz solution weekly (Cure rate 50-86%).



Generalized demodex and tail alopecia due to hypothyroidism.

Additional treatment options for generalized canine demodicosis include ivermectin (except in Collies, Shelties, herding breeds, and white German Shepherds, breeds which have an increased risk of having a genetic defect enabling penetration of the blood-brain barrier by ivermectin). 1% ivermectin is given orally beginning at a dose of 0.1 mg/kg/day and increased by 0.1 mg/kg every 3 days assuming toxicity does not occur. The therapeutic dosage range is from 0.3-0.6 mg/kg/day. A cure rate of 85-90% can be achieved at 0.6 mg/kg/day. Expect to treat cases for 3-6 months. Some sensitive animals will experience signs of toxicity including mydriasis, blindness, ataxia, dullness or seizures, and if toxic signs occur the drug must be immediately discontinued. Prior to instituting ivermectin therapy in a potentially sensitive dog, testing for the MDR-1 genetic defect is now available at Washington State University (www.vetmed.wsu.edu/depts-VCPL).

Pododemodicosis secondary to hypothyroidism.

An alternative treatment option, which is safe for the ivermectin-sensitive breeds but more expensive, is milbemycin oxime (Interceptor) 1-2 mg/kg orally once daily. All the above mentioned treatments should be continued until 2 separate skin scrapings, 30 days apart, are negative (no live or dead mites). Relapses may occur in up to 15% of cases and require pulse or daily long-term treatment for control. Lime sulfur dips, selamectin, and once weekly ivermectin injections are ineffective for treatment of generalized canine demodicosis. The prognosis for localized demodicosis is good, while for generalized disease the prognosis varies from good to guarded depending on the underlying cause.

Suggested readings:
Scott D, Miller M, Griffin C. Small Animal Dermatology, 6th edition. W.B. Saunders Company, Philadelphia, PA 2001. pp 457-476.
Hnilica K, Medleau L. Small Animal Dermatology: A Color Atlas and Therapeutic Guide. W.B. Saunders Company. Philadelphia, PA 2001. pp 65-70.