Demodex canis is the dog follicular mite, while Demodex injai is found within sebaceous glands and ducts. D. cornei lives in the stratum corneum.

Neonates are thought to acquire mites from their dam during nursing. Direct transmission, other than from dam to the pup rarely occurs.

Dogs may have either localized or generalized disease. There is no universally accepted definition of localized vs generalized disease but recently it has been suggested that with localized disease there are no more than four lesions with a maximum diameter of 2.5 cm. Demodicosis is also categorized based on age of onset- those less than 12 months of age (18 months in large or giant breeds) are considered juvenile onset while older dogs are considered adult onset. The prognosis is excellent for the localized form either in puppies or adult dogs while the generalized form carries a more guarded prognosis.

Demodex causes disease when there is an overgrowth of the commensal mites either associated with a genetic defect (juvenile onset) or immune suppression (adult onset). In the adult dog, hyperadrenocorticism (iatrogenic or spontaneous), hypothyroidism, leishmaniasis, or chemotherapy are the most identifiable causes of adult onset generalized demodicosis.

The lesions include non pruritic alopecia, scaling, follicular casts, follicular papules/pustules (if a secondary bacterial infection is present), comedones, crusts, erythema, hyperpigmentation, and lichenification. Pruritus is variable but is mild except in cases with a secondary bacterial folliculitis.

Lesions frequently involve the face and/or forelegs and may progress to affect other body sites. Since the lining of the external ear canal is epidermis, demodicosis may cause a bilateral ceruminous otitis externa. As the disease progresses, dogs may develop a deep bacterial folliculitis and furunculosis and draining tracts. In those cases peripheral lymphadenopathy, lethargy, and fever are commonly present. In some patients their presentation is exclusively pododemodicosis. In these cases a deep bacterial folliculitis and furunculosis is frequently present and the feet are swollen and painful leading to lameness.

In contrast to D.canis and cornea, D. injai tends to be associated with a greasy hair coat on the dorsum of the trunk. Many times alopecia is not present and only a low number of mites may be found on skin scrapings. It has been reported that terriers, especially wire haired fox terrier and West Highland white terrier, are at risk of developing this form of demodicosis.

Since demodicosis is a folliculocentric disease it will look identical to follicular lesions caused by a bacterial pyoderma and dermatophytosis. Superficial (for D.cornea) and deep skin scrapings (for the other species of demodex) are the most reliable method to diagnose demodicosis.

To perform a deep skin scraping it is best to squeeze the skin prior to and during the scraping to push the mites out of the hair follicles. Scrape the skin in the direction of the hair growth until capillary bleeding occurs. When lesions are present on the face or paws the animal should either be sedated before scraping or a hair pluck/trichogram may be performed in an awake animal. Hair plucks are performed with mosquito hemostat forceps that grasp and pull out hairs. It is best to collect hairs from the leading edge of the lesion. To increase your yield, squeeze the skin as you are plucking the hairs and be sure to collect a large number of hairs (50–100). Take the collected hairs and lay them on a slide containing a drop of mineral oil and add a cover slip. Sample multiple sites in each patient. Trichograms, or in cases of pustular demodicosis examination of the exudate, will detect Demodex mites in about 85% and 100% of dogs respectively with demodicosis. If the trichogram is negative but other sites are positive, sedation and skin scrapings of the feet should be performed since the mites may be present even if the feet appear alesional. It has been the author’s experience that pododemodicosis, if present, is usually the hardest component of generalized demodicosis to resolve and so should be used as one of the monitoring sites.

Recently it has been reported that applying tape to a skin lesion and then squeezing the skin is as an effective way to identify demodex mites in dogs. A study was performed to confirm this observation. Specifically the study was to evaluate and compare the sensitivities of acetate tape impression deep skin scraping for the diagnosis of canine demodicosis. They concluded that squeezing the skin followed by acetate tape prep was found to be as sensitive as deep skin scraping for the diagnosis of canine demodicosis. Unfortunately the author has not had the same experience.

Be sure to collect samples from multiple sites and note the site that the sample is collected from since localized disease is treated differently than generalized disease. When examining the slides you need to evaluate for the approximate number of each stage that is present (eggs, larva, nymph and adults). Also note how many of the mites alive vs are dead. These results will be important to compare to future skin scrapings as you are monitoring the dog’s response to therapy. With effective treatment a decreasing number of immature mites and the disappearance of eggs should occur. The number of live mites should also decrease. In all cases of demodicosis be sure to perform an examination of an otic swab. Otodemodicosis is identified by collecting roll swabs from each ear canal is epidermis, demodicosis may cause a bilateral ceruminous otitis externa.
using a cotton swab that has been dipped in mineral oil. The sample collected is placed onto a glass slide that also has a drop of mineral oil on its surface. A cover slip is applied and then the sample is examined.

If samples are collected as described it would be extremely uncommon to miss the presence of demodex mites. Occasionally this may occur, even with properly performed skin scrapings and hair plucks, if the dog has scarring due to chronic disease or because of the thickness of their dermis (therefore the deeper depth of their hair follicle making expulsion of the mite more difficult) (i.e. Shar-Pei).

**Error! Bookmark not defined.** If demodicosis is strongly suspected, but no mites are found on skin scrapings and hair plucks, skin biopsy is recommended to rule in or rule out their presence.

How to treat a dog with demodicosis depends on whether it is localized or generalized. In cases of localized demodicosis, less is best. In many cases, especially juvenile onset, the disease will spontaneously resolve within a couple months. Miticidal therapy is not required unless the disease becomes generalized. Since the progression of localized disease to more generalized form is not influenced by whether the localized form is treated or not, treatment of localized disease is not necessary. However, in the author’s practice “benign” topical treatment is prescribed. This is done so that if the disease does progress, the owner feels that something had been done to try to prevent for occurring. Topical therapy with benzoyl peroxide shampoo and/or gel can theoretically be helpful due to its antibacterial properties and follicular flushing activity. Due to its suppressive effect on the immune system you should avoid using any steroid containing product (topically or systemically) in patients with demodicosis (localized or generalized). Ensuring a proper diet and intestinal deworming program should also be part of the treatment of dogs with demodicosis. To evaluate the effectiveness of treatment, a follow up examination, including repeating skin scrapings, should be performed in 30 days.

Treating a dog with generalized demodicosis requires much more aggressive therapy than localized. Multimodal therapy, a common approach that is used to treat other diseases (e.g. arthritis, atopic dermatitis or congestive heart failure) will be necessary when treating generalized demodicosis. Acaricidal therapy and treating secondary bacterial infections if present is required for both adult and juvenile onset disease. In adult onset cases attempts should be made to identify and treat the underlying systemic disease.

Dogs with juvenile onset generalized demodicosis, in addition to the above mentioned treatment should be neutered. This is important not only to prevent the propagation of this genetic defect but also estrus may trigger recurrence of clinical disease.

As mentioned previously, in cases of adult onset generalized demodicosis attempts should be made to identify and treat the underlying disease. Evidence shows that successful treatment of an underlying cause increases the likelihood that adult onset demodicosis can be cured. In the author’s practice, diagnostics performed in cases of adult onset generalized demodicosis include a CBC, serum chemistry profile and a urinalysis. Depending on the age of onset, abdominal ultrasound and thoracic radiographs may be included in the minimum data base. Because of the influence that bacterial pyoderma or generalized demodicosis has on evaluating thyroid or adrenal gland disease, evaluation of these organs is delayed until any secondary bacterial infection has been resolved and the demodicosis has improved or is in remission.

Specific treatment of generalized demodicosis is outlined in table 1. This table is the result of the most recent consensus guidelines written by an international group of dermatologists. The author has indicated in bold the approach used in his practice.

Since dogs may look normal clinically but still have active disease (as determined by the presence of mites on skin scrapings) treatment must be continued beyond clinical resolution. Parasitic cure is defined as multiple negative skin scrapings, including lack of dead or fragmented mites, on 3 consecutive monthly visits. Skin scrapings should be used to determine the therapeutic end-point. This end point is reached when the dog looks normal clinically and skin scrapings have been performed monthly on the 4-6 most severely affected areas and have been negative for 3 consecutive visits. If during a visit the skin scraping is positive, it is important to compare the number of live and dead mites and the number of each stage of the mite life cycle to the previous visit. An indication of effective treatment is that during therapy the number of live mites found on skin scrapings and a urinalysis. Depending on the age of onset, abdominal ultrasound and thoracic radiographs may be included in the minimum data base. Because of the influence that bacterial pyoderma or generalized demodicosis has on evaluating thyroid or adrenal gland disease, evaluation of these organs is delayed until any secondary bacterial infection has been resolved and the demodicosis has improved or is in remission.

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### Table 1- Summarized treatment of canine demodicosis *

**Treatment of a dog with severe generalized disease**

1. Perform cytology and if there is evidence of a deep bacterial skin infection or the dog has been treated previously with antibiotics a bacterial culture and sensitivity. With inflammatory cells and bacteria present, appropriate oral antibiotic therapy is required.
2. Use topical therapy with chlorhexidene or benzoyl peroxide shampoo weekly to possibly twice weekly. (Unless amitraz is being applied)
3. There are several treatment options for the treatment of canine demodicosis. The best option will depend on the legalities pertaining to the use of veterinary pharmaceutical products in the country of residence, the finances of the owner and the clinical situation. However, independent of the treatment specifics the dog should be neutered because dogs in need of mite treatment should not be allowed to breed, and the disease may relapse in cycling bitches.
   a. Amitraz weekly or every 2 weeks in a concentration of (0.025–0.06%) can be used. Dogs with a medium to long hair coat need to be clipped, and skin should stay dry between rinses to avoid washing off the drug.
Rinsing should be performed in well-ventilated areas. The author only uses this therapy if the dog has failed to respond to ivermectin or is a herding breed. Please note that amitraz is EPA registered and doesn’t EVER allow any off label use (label states 1 bottle/2 gallons every 14 days)

b. Milbemycin oxime may be administered orally at a dose of 1–2 mg/kg/day. Moxidectin orally (see below) in the milbemycin family, is much less expensive than milbemycin, and is used if the dog fails to respond to ivermectin (again a non herding breed)

c. Moxidectin as a spot-on in combination with imidacloprid may be used weekly. This spot-on formulation has a markedly higher success rate in dogs with milder disease.

d. Ivermectin at an oral dose of 0.3–0.6 mg/kg (0.4 mg/kg) or moxidectin at 0.2–0.5 mg/kg p.o. daily are further options. With both drugs, a gradual increase from an initial dose of 0.05 mg/kg to the final dose (of 0.4 mg/kg) within a few days is recommended to identify dogs that cannot tolerate those drugs. Monitoring for neurological adverse effects should occur throughout the course of therapy. Ivermectin is the treatment of choice in the author’s practice.

e. Doramectin weekly at 0.6 mg/kg p.o. or SQ is a possible treatment. A gradual increase from an initial dose of 0.1 mg/kg to the final dose seems prudent to identify dogs that cannot tolerate the drug and will show neurological adverse effects.

So to summarize- this report states that “There is good evidence for the efficacy of weekly amitraz rinses and daily oral macrocyclic lactones such as milbemycin oxime, ivermectin and moxidectin for the treatment of canine demodicosis.”

Other recommendations are

Dogs should be evaluated monthly, and treatment should be continued until 3 consecutive visits with multiple negative skin scrapings have been achieved.

Treat secondary bacterial infections

Factors predisposing to demodicosis, such as malnutrition, endoparasites, endocrine disease, neoplasia and chemotherapy, should be identified and corrected to maximize response to therapy.


Diagnosis and management of malassezia dermatitis

Overview

Malassezia is a genus of lipophilic yeast found as a commensal of the skin and mucosal surfaces that may cause skin disease in a variety of mammalian species. In normal dogs these organisms are present in very small numbers on the skin (fold areas-lip, vulvar, axillae, interdigital), oral and anal mucosal surfaces, in the ear canals and in the anal saes. In contrast to Candida, MD is not associated w/recent antibiotic administration, in fact, there appears to be a symbiotic relationship between the surface staphylococcal organisms and the yeast. It is theorized that the organisms produce growth factors and micro-environmental changes (eg inflammation) that are beneficial to each so it is not uncommon to see concurrent infections w/Malassezia and staphylococcus. Why do animals develop Malassezia dermatitis (MD)? There have been numerous studies comparing the strain of Malassezia organisms found on skin of affected dogs vs. the skin of unaffected dogs. To date there has not been an identifiable difference in virulence and/or adhesion in Malassezia organisms found on skin of affected dogs vs. the skin of unaffected dogs. Since the organism virulence doesn’t explain MD, the explanation seems to be the host response to Malassezia organism. Both type I and type IV hypersensitivity reactions to Malassezia have been identified in dogs w/MD. Disorders that affect the barrier function of the skin (eg pruritic skin disease) or the cutaneous lipid content (eg hypothyroidism) are risk factors for developing MD

Signalment

There is no age or sex predilection

History

MD is always secondary to another skin disease. A clue that MD may be present is that the clinical features and/or the previously effective therapy of the underlying disease become ineffective. For example, pruritus that was seasonal becomes nonseasonal; the distribution of the pruritus changes, responsiveness to previously effective antibiotic and/or glucocorticoid therapy is decreased. Any allergic animal whose pruritus (intensity or distribution) or the therapeutic responsiveness of the pruritus changes suddenly should be evaluated for MD, pyoderma and ectoparasites.

Clinical findings

On physical examination lichenification, erythema, greasy exudate, dry scale, papules, plaques, alopecia or hyperpigmentation may be present. A moist dermatitis with a musty odor is not an uncommon clinical finding. Pruritus may vary from mild to intense and erythema may be present with minimal pruritus especially interdigitally.

The lesions may be focal or generalized and the distribution of the lesions overlaps with other pruritic diseases. Affected areas include interdigitally, intertriginous areas, face, nail folds, perioral (lateral muzzle), pinna and flexor surface of the elbow
Diagnosis

MD may cause a folliculitis that is clinically identical to staph pyoderma. Therefore if there are follicular papules, epidermal collarettes or lichenification you can’t assume that there is a bacterial component to the skin disease without performing skin cytologies. Remember to include skin scrapings for ectoparasites as part of your minimum data base.

Identifying *Malassezia* organisms budding yeast from the affected area is necessary to establish a diagnosis of MD. Tape impression or direct impression smear are the most common method used for sampling affected areas.

The question is “how many is too many organisms?” A previous report found that normal dogs had 1 yeast per 2700 oil field. MD is confirmed when, on cytology, you find ANY field that **has more 1** organism OR if there is 1 organism every 1-3 fields (1000X).

The ACVD task force on atopic dermatitis discussed MD as a complication of atopic dermatitis. The task force states that “Surface cytology of the skin and ear is useful to determine whether or not *Malassezia* or *Staphylococci* are present at lesional sites. Making antimicrobial treatment decisions based solely on microbe numbers is incorrect and inappropriate.” The article goes on to discuss that the host response to these normal organisms determines the severity of clinical signs. Their recommendation was “the result of cytology might better be limited to the sole report of ‘presence’ or ‘absence’ of detectable bacteria or yeast”.

Treatment

In order to prevent recurrence of MD the underlying cause must be identified and treated. As previously mentioned any disease that disrupts the barrier function, the lipid content of the skin surface, the cutaneous microclimate or host defense mechanisms may predispose the animal to MD. These include hypersensitivities (atopy, cutaneous adverse food reactions), ectoparasites (demodex, sarcoptes, and fleas), endocrinopathies (hypothyroidism, hyperadrenocorticism), metabolic diseases (metabolic epidermal necrosis), neoplasia (cutaneous T-cell lymphoma) and excessive skin folds. Genetic factors, as seen in Bassett hounds, predispose a dog to maintaining higher number of *Malassezia* organisms on their skin, putting them at greater risk for developing MD.

 unless the MD is very focal, the author prefers both topical and systemic therapy. This combination will be the most successful treatment of MD. Eliminating MD as the cause of pruritus is important so that when the dog is rechecked any remaining pruritus is a result of the underlying hypersensitivity reaction, not the MD.

There are a variety of effective topical agents including selenium sulfide, miconazole, ketoconazole, clotrimazole and chlorhexidine. In the authors experience any shampoo that contains at least 3% chlorhexidine or contains 2% chlorhexidine combined w/an azole is effective. Shampooing should be followed by a leave on conditioner containing an antifungal ingredient such as 2% miconazole. Depending on the severity and extensiveness of the lesions the frequency of application varies from daily to 3x/week.

Ketoconazole (KCZ) 5-10 mg/kg sid was the systemic drug of choice. Since the drug is now unavailable fluconazole (5-10 mg/kg/day). Another choice, especially for hard to medicate dogs is itraconazole 5 mg/kg given 2 consecutive days/week. A less costly therapy is terbinafine (30-40 mg/kg sid w/food). Regardless of which treatment is chosen the treatment should be continued for 14 days beyond clinical resolution BASED ON YOUR examination (not a phone call) with a minimum treatment time of 21 days. Please note that griseofulvin is ineffective against *Malassezia*.

Be sure to evaluate the dog for concurrent superficial bacterial pyoderma since MD and pyoderma occur simultaneously in dogs. In cases of concurrent superficial bacterial pyoderma, antibiotic therapy should be used simultaneously.