Canine demodiosis

Canine demodex mange can be caused by three different species of mites: Demodex canis, D. injay and, D. cornei. For many years, the belief has been that these mites live in the skin of dogs as part of the normal fauna. However, this theory had not been proven until recently when DNA of D. canis mites was found in the skin of every healthy dog tested using a PCR test. Why do dogs develop demodiosis? The reason is not completely understood but, there is enough evidence to support the presence of an immune dysfunction, at least in dogs with demodex mange caused by D. canis. This type of demodex mange is divided into juvenile-onset and adult-onset demodicosis. The generalized, juvenile form typically occurs in dogs 1 year old or younger and, the theory holds that these dogs inherit a dysfunction of T-cells which specifically recognize mite antigens and maintain them in small numbers. The young age of onset and breed predilection for this form of disease support a genetic predisposition. The localized, juvenile form is likely the result of an acquired, local alteration of the skin immune surveillance; it spontaneously resolves in most cases. The adult-onset form typically occurs in dogs older than 4 years of age and is associated with an underlying disease such as, Cushing’s, hypothyroidism, and neoplasia or, treatment with immunosuppressive drugs.

The pathomechanism of demodicosis caused by D. injay or D. cornei is not known at this time. Some dermatologists believe that D. cornei and D. canis are the same mite and, their different sizes and shapes reflect different life-cycle stages. Phylogenetic studies have shown their close relationship. However, D. cornei mites are not found consistently in demodicosis caused by D. canis. In addition, D. cornei is believed to dwell on the skin surface (similar to D. gatoi of cats and D. criceti of hamsters) and D. canis lives in the hair follicle canal. Interestingly, the author examined an old dog that developed pruritus after being in contact with another dog; D. cornei mites were found on multiple skin scrapings and the dog responded to lime sulfur dips. This suggests different mites causing different clinical signs.

Clinical signs

Demodicosis caused by D. canis

The only consistent clinical sign is alopecia that typically develops in the periorcular region, muzzle, chin, and feet. In severe cases, lesions can also be present on the neck, trunk and legs. In mild cases, erythema, scaling, and comedones (“black heads”) can be associated with alopecic areas. In moderate to severe cases, hyperpigmentation, seborrhea oleosa, prominent comedones, and furunculosis (rupture of hair follicles) may occur in areas of alopecia or in areas that have not yet developed frank hair loss. Clinical signs of furunculosis include the presence of draining tracts, serous-sanguineous exudate that form crusts when dried, and erosions or ulcerations. Lymphadenopathy can be present in severe, generalized cases. Dogs with D. canis demodicosis are not typically pruritic but, pruritus can develop in the presence of secondary bacterial infection. The classification of localized vs generalized disease is arbitrary and, most dermatologists consider the demodicosis “generalized” if the dog has more than 10 lesions affecting different body regions, or has pododemicosis. Knowing if a juvenile dog has the generalized disease is relevant because this form is considered inherited and the dog should not be bred. In addition, the generalized disease is more difficult to treat and, generally, does not spontaneously resolve.

The most common clinical presentation of demodicosis caused by D. injay is quite unique and characterized by the presence of hypotrichosis and excessive oil secretion that covers the dog’s hair coat. It affects primarily the face and dorsal trunk. Fewer cases may present with alopecia, erythema, hyperpigmentation, and comedones, similarly to dogs with demodicosis caused by D. canis. Skin biopsies of greasy areas have demonstrated large sebaceous glands and mites inhabiting both, the sebaceous glands and hair follicles. A large number of affected dogs are pruritic but, this could be related to the fact that many of these dogs have an underlying allergic disorder. The disease typically affects adult animals and has been diagnosed more frequently in terrier dog breeds but, it can affect any breed.

Textbooks mention that the clinical signs caused by D. cornei are the same as the signs caused by D. canis because these mites are morphological variations of the same mite. However, more studies and clinical experience will be required before it can be concluded that they are the same demodex mite species.

How to diagnose canine demodicosis

The gold standard diagnostic test for demodicosis is skin scraping. In cases of demodicosis caused by D. canis, the sensitivity of this test is very high if skin scrapings are performed properly. However, D. injay demodicosis is typically associated with much fewer mites that may be primarily located in the sebaceous glands; thus, it is not uncommon to have multiple negative scrapings even if they are properly performed. To increase the chances of finding D. canis and D. injay mites the following should be done: (i) pinch the skin to extrude mites from the follicle canals and sebaceous glands, (ii) perform multiple skin scrapings, (iii) scrape until capillary bleeding is obtained because these mites live deep in the hair follicle canal and sebaceous glands; (iv) avoid areas with hemorrhagic exudate as excessive blood may obscure the visualization of mites under the microscope. You can use a scalpel blade or spatula to perform skin
scrapings and, make sure to moisten the instrument (or the skin surface area chosen for scraping) with mineral oil to facilitate the collection of hairs and scales. The samples should be transferred to a glass slide with a few drops of mineral oil and covered with a coverslip. Lower the microscope condenser to increase contrast and allow better visualization of the mites (remember, they are transparent). Examine the whole slide systematically using the 10x or 40x objective. Demodex injay can be twice as long as D. canis mites and, D. cornei is typically one-third or half the size of D. canis. Trichoscopy (i.e. the examination of plucked hairs under the microscope) can also be used to diagnose demodicosis; however, its sensitivity is much lower than skin scrapings and, a negative result should not rule out demodicosis. The author likes to perform trichoscopy to evaluate areas that are difficult to scrape such as, the face and feet, because dogs tolerate plucking hairs much better than skin scrapings. Very rarely a skin biopsy will be required for the diagnosis of demodicosis (e.g. Shar-pei dogs or fibrotic skin).

**How to manage canine demodicosis**

The following parasiticidals are currently available in the US to treat canine demodicosis: (i) amitraz, (ii) ivermectin, (iii) 2.5 % moxidectin + 10% imidacloprid (Advantage Multi), (iv) moxidectin, and (v) doramectin.

**Amitraz (Mitaban) dips**

Off-label treatment protocols (every-other-day to weekly dips; various textbooks detail these protocols) are typically used because of better and faster responses with these protocols compared to the protocol recommended by the manufacturer. The amitraz solution has to be prepared immediately before use and, any leftover should not be reused. Dogs with medium or long hair coats should be shaved and the solution allowed to air dry. This is a labor intensive treatment option. Side effects may include: sedation, pruritus, hypothermia, ataxia, disorientation, and hyperglycemia.

**Ivermectin**

The dose range shown to be efficacious is 0.3 to 0.6 mg/kg/day, given orally (the author typically uses 0.6 mg/kg/day). However, do not use ivermectin in breeds that may carry a mutation in the ABCB1 gene that encodes for the transmembrane transporter, P-glycoprotein. These breeds (or their crosses) include: collie, Shetland sheepdog, Australian shepherd, white Swiss shepherd, longhaired whippet, silken windhound, McNab, and, border collie. Breeds heterozygous for the mutation include: Old English sheepdog, Waller, and English shepherd. Ideally, you should not use ivermectin in these breeds either. Side effects associated with ivermectin and that may develop in any breed include: midryasis (typically the first sign of toxicity), ataxia, tremors, seizures, coma, and, death. Currently, there is a genetic test available at the Washington State University Veterinary Clinical Pharmacology Laboratory. This test should be performed in these dog breeds or their crosses before starting treatment.

**2.5% moxidectin + 10% imidacloprid (Advantage Multi)**

It can be an alternative for breeds susceptible to ivermectin but, unfortunately, it does not work as well. The recommended treatment protocol is weekly to bi-weekly applications at the dose used for heartworm prevention or flea control.

**Moxidectin**

The recommended dose ranges from 0.2 to 0.5 mg/kg/day, administered orally. It is suggested to increase the dose daily by increments of 0.05 mg/kg until the recommended dose is achieved, to identify the dogs that do not tolerate this drug. Side effects include: midryasis (typically the first sign of toxicity), ataxia, tremors, seizures, coma, and, death. Vomiting may also occur.

**Doramectin**

It is a long-acting avermectin with twice the half-life of ivermectin. The recommended dose is 0.6 mg/kg administered subcutaneously, every 7 days. Do not use in the breeds susceptible to ivermectin. The side effects are the same as the ones described for ivermectin but, non-sensitive breeds that do not tolerate ivermectin tend to tolerate doramectin.

Independent of the parasiticidal chosen to treat demodicosis, continue treatment for 30 days past two consecutive negative skin scrapings performed, approximately, 4 weeks apart! At this point, the animal is considered in remission. Follow-up with skin scrapings for 1 year after remission, before considering the dog cured.

Other important points to remember when managing a dog with demodicosis: (i) identify and appropriately treat any secondary bacterial skin infection, (ii) advise owners to not breed dogs with juvenile-onset, generalized demodicosis, (iii) look for an underlying disease in dogs that develop adult-onset demodicosis, and, (iv) the localized form of D. canis demodicosis in juvenile dogs, typically spontaneously resolve and, may not need to be treated.

**Feline demodicosis**

Feline demodicosis can be caused by *Demodex cati, D. gatoi* and a yet unnamed species.

**Demodicosis caused by D. cati**

This mite lives in the hair follicle canal. The disease typically develops in middle age to older cats and, is generally associated with an underlying disorder (e.g. diabetes mellitus, feline leukemia virus infection, feline immunodeficiency virus infection, squamous cell carcinoma, etc.). The localized form typically affects the face and neck, and sometimes the ear canals. It is characterized by alopecia, erythema, scaling, and crusting. It often spontaneously resolves if no underlying disease is present or, has been controlled. The generalized form has similar lesions but, in addition to the head and neck, it affects many other areas of the body. Co-infection with other species of mites has been reported. Pruritus may or may not be present; if present, look for co-infection with D. gatoi.
Demodicosis caused by D. gatoi
This form of demodicosis is more common than the one caused by D. cati but, it seems to be more prevalent in some regions of the US than others. The mite dwells on the skin surface. The demodicosis can develop at any age and, cats are typically healthy otherwise. This demodex mange is pruritic and contagious; however, some cats can be asymptomatic carriers. The typical clinical sign is self-inflicted, symmetrical, non-inflammatory alopecia, localized to areas where the cat can reach. The skin in affected areas often looks normal but, can be erythematous, scaly, or hyperpigmented.

Demodicosis caused by the unnamed mite
Very little is known about the disease caused by this mite. However, published reports suggest that it occurs primarily in middle age to older cats that recently recovered from or had an underlying disease at the time of diagnosis (e.g. upper respiratory disease). Alopecia to hypotrichosis has been reported but, in one report the main sign was large amounts of hair in the cages of affected cats (cats were housed in shelters). Pruritus is variable.

How to diagnose feline demodicosis
Demodicosis caused by D. cati is easily diagnosed by performing proper, deep skin scrapings (use the same technique described above for dogs). Trichoscopy can also be used but, typically has a lower sensitivity compared to skin scrapings. This mite is microscopically similar to D. canis but all immature stages are narrower than D. canis and the eggs more oval rather than spindle shaped.

Mites are difficult to find in demodicosis caused by the surface dweller D. gatoi, because affected cats easily ingest these mites when overgrooming in response to pruritus associated with this mange. To increase the chances of finding mites, perform broad superficial scrapings of affected and non-affected areas where the cat cannot reach (e.g. dorsal neck). Perform fecal flotation if mites cannot be found in multiple scrapings. Mites are much more easily found in asymptomatic cohabiting cats because they are in larger numbers. D. gatoi are shorter and blunter than D. cati mites. Response to a treatment trial can also be used as a diagnostic method. If the clinical signs are caused by the mites, significant improvement should be noticed after the 4th parasiticidal application (see treatment below), in most cases.

Very little is known, at this time, about the best way to diagnose the unnamed mite species. In one report, mites were frequently found via trichoscopy (proximal portion of the hair shaft in most cases), indicating that they live in the hair follicle canal. In another report, mites were easily found in multiple skin scrapings. The recommendation at this time is to perform multiple, deep skin scrapings in addition to trichoscopy, when suspecting of this demodicosis.

How to manage feline demodicosis
Follicular demodicosis caused by D. cati
An important aspect of the treatment regimen is to identify and treat any underlying disease. Weekly 2% lime sulfur or amitraz (125 or 250 ppm) dips has been used successfully to treat D. cati demodicosis. In addition, ivermectin given orally at the dose of 0.3 mg/kg per day or, doramectin given subcutaneously at the dose of 0.6 mg/kg per week, has also shown good results. It is important to continue treatment for 30 days past two consecutive negative skin scrapings performed approximately 4 weeks apart.

Surface demodicosis caused by D. gatoi
It may appear that the surface demodicosis caused by D. gatoi is easier to treat because it is typically not associated with an underlying disease. However, this is not always the case. The various treatment modalities reported in textbooks, scientific papers or, anecdotally, have shown variable results. The following are the commonly used treatment protocols: (i) 2% lime sulfur dips given weekly for 6 to 8 weeks; (ii) 2.5% moxidectin + 10% imidacloprid (Advantage-Multi) applied weekly for 8 to 12 weeks; (iii) amitraz dips at 125 to 250 ppm given weekly for 6 to 8 weeks (use carefully due to potential side effects); and, (iv) ivermectin at 0.3 to 0.6 mg/kg given orally and, daily to every-other-day, for 8 to 10 weeks. Because this is a contagious disease it is important to treat all cohabiting cats.

Demodicosis caused by the unnamed species
Very little is reported on the treatment of this, apparently rare, feline demodicosis. Ivermectin given orally at 0.3 mg/kg per day in association to weekly lime sulfur dips resulted in good response. One cat was treated with 0.6 mg/kg of ivermectin daily, given subcutaneously, and no mites were found on multiple skin scrapings after 4 weeks. Look for associated underlying disorders.