CHAPTER 5 Parasitic Skin Disorders

Canine Localized Demodicosis

Features
Skin lesions occur when there is a localized overpopulation of *Demodex canis*, a normal commensal inhabitant of canine skin. Demodectic overgrowth is often associated with a predisposing factor such as endoparasitism, poor nutrition, immunosuppressive drug therapy, or transient stress (e.g., estrus, pregnancy, surgery, boarding). Canine localized demodicosis is common in dogs, with highest incidence reported in puppies 3 to 6 months old.

Canine localized demodicosis may appear as one to five patchy areas of alopecia with variable erythema, hyperpigmentation, and scaling localized to one region of the body. Lesions are most common on the face, but they can be anywhere on the body. Lesions are not usually pruritic unless they are secondarily infected.

Top Differentials
Differentials include superficial pyoderma, dermatophytosis, and trauma.

Diagnosis
1. Microscopy (deep skin scrapes): many demodectic adults, nymphs, larvae, or ova
2. Dermatohistopathology: intrafollicular demodectic mites with varying degrees of perifolliculitis, folliculitis, or furunculosis

Treatment and Prognosis
1. Any predisposing factors and secondary pyoderma should be identified and treated.
2. Lesions should be treated topically with 2.5% to 3% benzoyl peroxide shampoo, lotion, cream, or gel every 24 hours.
3. Miticidal treatment may not be necessary because many cases resolve spontaneously.
4. Rotenone-containing products or benzyl benzoate lotion may be miticidal when applied to lesions every 24 hours.
5. Alternatively, 0.03% to 0.05% amitraz solution applied to lesions every 24 hours is often effective.
6. Topical therapy is continued until follow-up skin scrapings are negative and lesions have resolved.

7. The prognosis is good. Most cases resolve within 4 to 8 weeks, but a few may progress to generalized demodicosis. Systemic therapy or total body dips should not be used in intact animals as this may mask the development of generalized demodicosis, which is thought to be an inherited disease. *D. canis* is not considered contagious to other dogs (except for newborn puppies), to cats, or to humans.

FIGURE 5-3 Canine Localized Demodicosis. Multiple alopecic papular lesions on the face of an adult Shetland Sheep dog. (Courtesy D. Angarano.)

FIGURE 5-4 Canine Localized Demodicosis. Focal area of alopecia and hyperpigmentation typical of a folliculitis.
Canine Localized Demodicosis

Numerous comedones on the abdomen of a dog with hyperadrenocorticism. Comedones are often caused by demodicosis or Cushing’s.

Microscopic image of *demodex Demodex* mites as seen with a 10x objective.

This circular area of alopecia with central hair regrowth typical of folliculitis is often misdiagnosed as dermatophytosis.

Focal area of papular dermatitis caused by demodex.

A focal area of alopecia on the muzzle of a young dog. (Courtesy D. Angarano.)

Papular dermatitis with hyperpigmentation typical of Demodicosis.
Canine Generalized Demodicosis

Features
Canine generalized demodicosis may appear as a generalized skin disease that may have genetic tendencies and can be caused by three different species of demodectic mites: *D. canis*, *D. injai*, and an unnamed short-bodied *Demodex* mite. *D. canis*, a normal resident of the canine pilosebaceous unit (hair follicle, sebaceous duct, and sebaceous gland), is primarily transmitted from the mother to neonates during the first 2 to 3 days of nursing, but adult-to-adult transmission may rarely occur. *D. injai*, a recently described, large, long-bodied *Demodex* mite, is also found in the pilosebaceous unit, but its mode of transmission is unknown. Mode of transmission is also unknown for the short-bodied unnamed *Demodex* mite, which, unlike the other two species, lives in the stratum corneum. Depending on the dog's age at onset, generalized demodicosis is classified as juvenile-onset or adult-onset. Both forms are common in dogs. Juvenile-onset generalized demodicosis may be caused by *D. canis* and the short-bodied unnamed *Demodex* mite. It occurs in young dogs, usually between 3 and 18 months of age, with highest incidence in medium-sized and large purebred dogs. Adult-onset generalized demodicosis can be caused by all three mite species and occurs in dogs older than 18 months of age, with highest incidence in middle-aged to older dogs that are immunocompromised because of an underlying condition such as endogenous or iatrogenic hyperadrenocorticism, hypothyroidism, immunosuppressive drug therapy, diabetes mellitus, or neoplasia. To date, only adult-onset disease has been reported with *D. injai*, with highest incidence noted in terrier breeds and their crosses, especially West Highland White terriers.

Clinical signs of infestation with either *D. canis* or the unnamed *Demodex* mite are variable. Generalized demodicosis is defined as five or more focal lesions, or two or more body regions affected. Usually, patchy, regional, multifocal, or diffuse alopecia is observed with variable erythema, silvery grayish scaling, papules or pruritus. Affected skin may become lichenified, hyperpigmented, pustular, eroded, crusted, or ulcerated from secondary superficial or deep pyoderma. Lesions can be anywhere on the body, including the feet. Pododermatitis is characterized by any combination of interdigital pruritus, pain, erythema, alopecia, hyperpigmentation, lichenification, scaling, swelling, crusts, pustules, bullae, and draining tracts. Peripheral lymphadenomegaly is common. Systemic signs (e.g., fever, depression, anorexia) may be seen if secondary bacterial sepsis develops.

*D. injai* infestations are typically characterized by greasy seborrhea (seborrhea oleosa), especially over the dorsum of the trunk. Other skin lesions may include alopecia, erythema, hyperpigmentation, and comedones.

Top Differentials
Differentials include pyoderma (superficial or deep), dermatophytosis, hypersensitivity (flea bite, food, atopy), and autoimmune skin disorders.

Diagnosis
1. Microscopy (deep skin scrapes): many demodectic adults, nymphs, larvae, and ova are typically found with *D. canis* and the short-bodied, unnamed demodectic mite, although *D. canis* may be difficult to find in fibrotic lesions and in feet. With *D. injai*, mites may be low in number (e.g., 1-2 mites per high power field)
2. Dermatohistopathology: minimal to mild suppurative perivascular dermatitis with mites in stratum corneum, or intrafollicular demodectic mites with varying degrees of perifolliculitis, folliculitis, or furunculosis

Treatment and Prognosis
1. If adult-onset, any underlying conditions should be identified and corrected. Spontaneous resolution of generalized demodicosis after treatment of underlying hypothyroidism without miticidal treatment has been reported in one dog with *D. injai* infestation.
2. Intact dogs, especially females, should be neutered because estrus or pregnancy may trigger relapse.
3. Any secondary pyoderma should be treated with appropriate long-term (minimum 3-4 weeks) systemic antibiotics that are continued at least 1 week beyond clinical resolution of the pyoderma.
4. Traditional miticidal treatment entails the following:
   - Total body hair coat clip if dog is medium- to long-haired
   - Weekly bath with 2.5% to 3% benzoyl peroxide shampoo, followed by a total body application of 0.03% to 0.05% amitraz solution. The cure rate ranges from 50% to 86%.
   - For demodectic pododermatitis, in addition to weekly amitraz dips, foot soaks in 0.125% amitraz solution should be performed every 1 to 3 days.
6. Alternatively, treatment with ivermectin 0.6 mg/kg PO every 24 hours is often effective against generalized demodicosis. Initially, ivermectin 0.1 mg/kg PO is administered on day 1, then 0.2 mg/kg PO is administered on day 2, with oral daily increments of 0.1 mg/kg until 0.6 mg/kg/day is being administered, assuming that no signs of toxicity develop. If the dog cannot tolerate a 0.6-mg/kg/day dosage, treatment with 0.4 mg/kg/day can be attempted. The cure rate for 0.6 mg/kg/day ivermectin is 85% to 90%.

7. Another effective alternative therapy is milbemycin oxime, 0.5 to 2 mg/kg PO every 24 hours. The cure rate is 85% to 90%, with the prognosis for cure being better for juvenile-onset cases than for adult-onset cases.

8. Doramectin is also reported to be effective against canine demodicosis at a dose of 0.6 mg/kg SC once weekly. The cure rate is approximately 85%. Adverse effects are uncommon but include, as for ivermectin, dilated pupils, lethargy, blindness, and coma.

9. For dogs ≤20 kg, the use of 9% amitraz collars may be effective. The dog’s neck is shaved to ensure that the collar is in close contact with the skin, and the collar is replaced every 2 weeks for the duration of treatment. In small dogs, use of 9% amitraz collars alone may be as effective as ivermectin (0.6 mg/kg/day PO). Also, the combined use of oral ivermectin and 9% amitraz collars may be more effective than either oral ivermectin or 9% amitraz collars alone.

10. Moxidectin 1% injectable for cattle has been reported effective when administered at a dosage of 0.4 mg/kg PO every 24 to 72 hours. However, adverse effects are common, especially when the drug is administered SC.

11. Regardless of the miticidal treatment chosen, therapy is administered over the long term (weeks to months). Treatments should be continued for at least 1 month beyond the time when follow-up skin scrapings become negative for mites.

12. The prognosis is good to fair. Relapses may occur, requiring periodic or lifelong treatment in some dogs. The use of glucocorticosteroids in any dog that has been diagnosed with demodicosis should be avoided. Because of its hereditary predisposition, neither female nor male dogs with juvenile-onset generalized demodicosis should be bred. _D canis_ is not considered contagious to cats or to humans. It is transmitted from bitch to newborn puppies during the first 2 to 3 days of nursing, and possibly between adult dogs that are close cohabitants. The mode of transmission for _D inai_ and the unnamed short-bodied _Demodex_ mite is unknown.
Feline Demodicosis

Features
Feline demodicosis is a skin disease that can be caused by two different species of demodectic mites—D cati, a normal commensal of cat skin, and D gatoi, a short-bodied Demodex mite whose normal habitat is unknown. Skin disease may be localized or generalized. D gatoi is a relatively recently recognized infection that is contagious and usually causes pruritic skin disease. D cati infections are often associated with an underlying immunosuppressive or metabolic disease such as feline immunodeficiency virus (FIV), feline leukemia virus (FeLV), toxoplasmosis, systemic lupus erythematosus, neoplasia, or diabetes mellitus. Localized or generalized demodicosis caused by D cati infection is rare in cats. D gatoi infections are emerging as a common cause of pruritic skin disease in cats, especially in the southern United States.

Localized disease is characterized by a variably pruritic ceruminous otitis externa or by focal patchy alopecia and erythema that may be scaly or crusty. Localized skin lesions are most common around the eyes, on the head, or on the neck. Generalized disease is characterized by variably pruritic, multifocal, patchy, regional, or symmetrical alopecia, with or without erythema, scaling, crusts, macules, and hyperpigmentation. Lesions usually involve the head, neck, limbs, flanks, or ventrum. Ceruminous otitis externa and secondary pyoderma may be present.

Top Differentials
Differentials include dermatophytosis, other ectoparasites (Cheyletiella, Notoedres, ear mites), hypersensitivity (flea bite, food, atopy), psychogenic alopecia, and other causes of otitis externa.

Diagnosis
1. Microscopy (deep and superficial skin scrapings, ear swabs): demonstration of demodectic adults, nymphs, larvae, or ova. D gatoi may be difficult to find
2. D gatoi: history, clinical signs, and response to weekly lime sulfur dips
3. Dermatohistopathology: minimal to mild suppurative perivascular dermatitis with mites in stratum corneum, or intrafollicular mites with varying degrees of perifolliculitis and folliculitis

Treatment and Prognosis
1. Any predisposing factors should be identified and corrected.
2. D gatoi may be difficult to find on microscopy but respond well to lime sulfur dips.
   a. 2%-4% lime sulfur dips applied q 3-7 days for 4-8 weeks. Clinical improvement is often observed within 3-4 weeks, but therapy should be continued for a total of 6-8 weeks to resolve the infection
   b. Anecdotal reports suggest that ivermectin and milbemycin may be variably effective, but studies are lacking
   a. For localized lesions, topical therapies that may be effective when applied q 24 hours include the following:
      Rotenone
      0.025%-0.03% amitraz solution
   b. For generalized lesions, treatments that may be effective include the following:
      2% lime sulfur solution applied to the entire body q 7 days
      Doramectin 0.6 mg/kg SC once weekly
      0.015%-0.025% amitraz solution applied to entire body q 1-2 weeks. Note: Do not use amitraz on diabetic cats
   c. For both localized and generalized disease, treatments should be continued until lesions have resolved and follow-up skin scrapings are negative for mites (approximately 3-4 weeks)
4. The prognosis for localized demodicosis is good. The prognosis for generalized demodicosis is good to guarded, depending on the underlying cause. D cati is not considered contagious to other cats (except for newborn kittens), to dogs, or to humans. The mode of transmission for D gatoi is unknown, but reports of unrelated household cats being simultaneously affected suggest that it may be contagious between adult cats.