**#1 Canine Atopy** (environmental, pollen allergies)

**Features**

Canine atopy is a hypersensitivity reaction to inhaled (possibly a historic theory) or cutaneously absorbed environmental antigens (allergens) in genetically predisposed individuals. It is common in dogs, with age of onset ranging from 6 months to 6 years. However, in most atopic dogs, symptoms first appear at between 1 and 3 years of age.

Symptoms begin as skin erythema and pruritus (licking, chewing, scratching, rubbing), which may be seasonal or nonseasonal, depending on the offending allergen. The distribution of the pruritus usually involves the **feet**, flanks, groin, axillae, face, and ears. Self-trauma often results in secondary skin lesions, including salivary staining, alopecia, excoriations, scales, crusts, hyperpigmentation, and lichenification. Secondary pyoderma, *Malassezia* dermatitis, and otitis externa are common. Chronic acral lick dermatitis, recurrent pyotraumatic dermatitis, conjunctivitis, hyperhidrosis (sweating), and, rarely, allergic bronchitis or rhinitis may be seen.

**Top Differentials**

Differentials include food allergy, scabies, *Malassezia* dermatitis, bacterial pyoderma, as well as other hypersensitivities (flea bite, contact), parasites (cheyletiellosis, pediculosis), and folliculitis (dermatophyte, *Demodex*).

**Diagnosis**

**1.** Seasonal foot-licking is the most unique and typical symptom of atopy. If year-round allergens (house dust mites) are causing the allergy, the foot-licking may be nonseasonal.

**2.** Allergy testing (intradermal, serologic): allergy testing can be highly variable according to the method used. Positive reactions to grass, weed, tree, mold, insect, dander, or indoor environmental allergens are seen. False-negative and false-positive reactions may occur.

**3.** Dermatohistopathology (nondiagnostic): superficial perivascular dermatitis that may be spongiotic or hyperplastic. Inflammatory cells are predominantly lymphocytes and histocytes. Eosinophils are uncommon. Neutrophils or plasma cells suggest secondary infection.

**Treatment and Prognosis**

1. **Infection Prevention:**
   1. Any secondary pyoderma, otitis externa, and *Malassezia* dermatitis should be treated with appropriate therapies. Controlling and preventing secondary infection is an essential component of managing atopic dogs. Bathing every 3 – 7 days and treating the ears after every bath helps wash off pollens and disinfect the skin and ear canals, preventing the secondary infections from recurring.
2. **Symptomatic Therapy (itch control):**

**a** An integrated flea control program should be instituted to prevent flea bites from aggravating the pruritus.

**b** Topical therapy with antimicrobial shampoos and anti-itch conditioners, and sprays (i.e., those containing oatmeal, pramoxine, antihistamines, or glucocorticoids) applied every 2 to 7 days or as needed may help reduce clinical symptoms.

**C** Systemic antihistamine therapy reduces clinical symptoms in many cases (Table 7-1). Antihistamines can be used alone or in combination with glucocorticoids or essential fatty acids for a synergistic effect. One- to two-week long therapeutic trials with different antihistamines may be required to determine which is most effective.

**D** Oral essential fatty acid supplements (180mg EPA/10 lb) help control pruritus in 20% to 50% of cases, but 8 to 12 weeks of therapy may be needed before beneficial effects are seen. Also, a synergistic effect is often noted when essential fatty acid supplements are administered in combination with glucocorticoids or antihistamines.

**E** Dextromethorphan, an opioid antagonist, may also be a useful adjunct in managing the licking, chewing, and biting behaviors associated with allergic dermatitis in dogs. Dextromethorphan 2mg/kg PO should be administered every 12 hours. A beneficial effect should be seen within 2 weeks.

F Systemic glucocorticoid therapy is often effective (75%) in controlling pruritus but almost always result in adverse effects ranging to mild (PU/PD) to severe (immuno-dysfunction, Demodicosis, and calcinosis cutis). It is a therapeutic option if the allergy season is very short but may result in unacceptable adverse effects, especially if used over the long term.

1. Potent, long acting injectable steroids are contraindicated for the treatment of allergies due to their comparatively short anti-inflammatory benefits ( 3 weeks) relative to the prolonged metabolic and immuno-depressive effects (6-10 weeks).

2. Injectable short acting steroids (dexamehtasone Sodium Phosphate, 0.5 – 1 mg/kg or prednisilone acetate 0.1mg – 1 mg/kg) are effective at providing relief and may last 2 to 3 weeks if there are no concurrent secondary infection. This treatment option allows the clinician to more closely control and monitor the patients steroids use compared to oral treatments that are administered by the owner.

3. Temaril-P (trimeprizine and prednisilone combination) is a unique drug that provides significant antipruritic effects at a relatively lower dose of the prednisilone. One tablet should be administered per 10 to 20 kg every 24 to 48 hours. The dosage should be tapered to the lowest possible dose and frequency.

4. Prednisone 0.25 to 1mg/kg (or methylprednisolone 0.2-0.8mg/kg) PO should be administered every 24 to 48 hours for 3 to 7 days. The dosage should be tapered to the lowest possible dose and frequency.

5. All dogs treated with long-term steroids (more than 3 months) should be frequently monitored for liver disease and UTI.

**8. Allergy Treatment (immune-modulation)**

**a.** Exposure to offending allergens should be reduced, if possible, by their removal from the environment. High-efficiency particulate (HEPA) air and charcoal filters should be used to reduce pollens, molds, and dust in the home. For house dust mite–sensitive dogs, household treatments for carpets, mattresses, and upholstery with the acaricide benzyl benzoate once a month for approximately 3 months, then every 3 months thereafter, may effectively eliminate house dust mites from the environment. Old dog beds should be discarded as these accumulate house dust mite antigens. Dehumidifying the house to below 40% relative humidity decreases house dust mite, mold, and flea antigen loads. To achieve this, high-efficiency dehumidifiers that are capable of pulling several liters of water from the air per day are required.

**b.** Cyclosporine (Atopica) helps control pruritus in 75% of atopic dogs. A dose of 5mg/kg PO should be administered every 24 hours until beneficial effects are seen (approximately 4‑6 weeks). Then, dosage frequency should be tapered down to every 48 to 72 hours. For long-term control, approximately 25% of dogs require daily dosing, 50% can be controlled with every-other-day dosing, and approximately 25% can be controlled with twice-weekly dosing. Glucocorticoids can be used initially to speed response. As of this writing, there are no statistically significant increases in tumor risk or severe infection resulting from the immune effects of cyclosporine.

**c.** With immunotherapy (allergy vaccine), 60% to 75% of atopic dogs show good (some medical therapy still needed) to excellent (no other therapy needed) response. Clinical improvement is usually noted within 3 to 5 months of initiation of immunotherapy, but it can take up to 1 year in some dogs.

11. The prognosis is good, although lifelong therapy for control is needed in most dogs. Relapses (pruritic flare-ups with/without secondary infections) are common, so individualized treatment adjustments to meet patient needs may be required periodically. In dogs that become poorly controlled, one should rule out secondary infection (e.g., that caused by bacteria or *Malassezia*); sarcoptic mange; demodicosis; concurrent food, flea bite, and recently acquired hypersensitivity to additional environmental allergens. Because a strong genetic component is present, the breeding of any male or female dog with clinical signs of atopic dermatitis should be discouraged.

Box

Author’s Note

Our profession has excelled at reducing the use of steroids for arthritis; however, we have failed to make similar achievements for allergic disease including atopy. Since both disease have many similarities, including chronicity and multimodal therapeutic options, our goal should be to minimize the use of steroids for allergic diseases through the use of alternative, safer treatment options. To achieve best medicine, the frequency of steroid use should be similar for patients with arthritis and allergy.

The use of long-acting, injectable steroids should be stopped due to the profound impact on the metabolic and immune systems as well as the growing concern of legal liability for the practitioner.

Author’s Note

The only real, long-term options for treating the allergic immune response to environmental allergens are avoidance, allergy vaccine, or cyclosporine (Atopica). Based on typical cgeneral practice demographics, every full time small animal veterinarian should have approximately 20-30 patients who are no longer controlled with symptomatic therapy and need more aggressive treatment (allergy vaccine or cyclosporine).

#2 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 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. Pododemodicosis 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 a focal areas of greasy seborrhea (seborrhea oleosa), especially over the dorsum of the trunk. Other skin lesions may include alopecia, erythema, hyperpigmentation, and comedones. Small breeds and terriers seem to predisoosed to Demodex injai infections.

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 and difficult to find requiring skin biopsies.

Treatment and Prognosis

 1. If adult-onset, any underlying conditions should be identified and corrected. All steroid containing therapies should be discontinued as steroid administration is the mostcommon cause of adult onset demidicosis.

 2. Intact dogs, especially females, should be neutered. 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. Topical shampoo therapy using a 1-3% benzoyl peroxide shampoo every 3-7 days will help speed resolution and enhance the mitacidal treatments.

5. Effective Mitacidal therapies include the following:

  \*Ivermectin 0.2-0.6mg/kg PO every 24 hours is often effective against generalized demodicosis. Initially, ivermectin 0.1mg/kg PO is administered on day 1, then 0.2mg/kg PO is administered on day 2, with oral daily increments of 0.1mg/kg until 0.2-0.6mg/kg/day is being administered, assuming that no signs of toxicity develop. The cure rate for 0.4mg/kg/day ivermectin is 85% to 90%.

  \*Milbemycin oxime, 0.5 to 2mg/kg PO every 24 hours. The cure rate is 85% to 90%.

  \*Doramectin is also reported to be effective against canine demodicosis at a dose of 0.6mg/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.

  \*For dogs 20kg, the use of 9% amitraz collars may be effective. In small dogs, use of 9% amitraz collars alone may be as effective as ivermectin (0.6mg/kg/day PO).

\* Topical application of Promeris (topical metaflumizone and amitraz solution) (topical metaflumizone and amitraz solution) every two weeks has demonstrated good efficacy.

\*Moxidectin has demonstrated variable efficacy when applied every 2-4 weeks.

Historical Treatment Include:

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. 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 the first follow-up skin scrapings becomes negative for mites (total of two negative skin scrapings).

7. 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 injai* and the unnamed short-bodied *Demodex* mite is unknown.

Author’s Note:

Steroids are the most common cause of adult onset Demodicosis.

Products containing amitraz tend to be the most toxic usually due to the product vehicle.

Aggressive treatment should be tried for up to six months before giving up.

One of the most common causes of treatment failure is that the patient will look greatly improved before negative skin scrapes are achieved. Many owners will discontinue treatment prematurely resulting in relapse.

The average time to achieve clinical improvement is 4-6 weeks; the first negative skin scrape usually occurs around 6-8 weeks; and most patients need approximately 3 months of treatment to resolve the infection based on two negative skin scrapes at least 3 weeks apart.

**#3 Flea Allergy Dermatitis**

**Features**

Flea allergy dermatitis is a common skin disease in dogs and cats sensitized to flea salivia proteins through repeated and intermittent flea bites. Symptoms are usually seasonal (warm weather months and in the fall) in temperate zones and often nonseasonal in subtropical and tropical areas. Fall is often the most severe season relating to when the first cold snap occurs.

**Dogs**

The distribution typically involves the caudodorsal lumbosacral area, dorsal tail head, caudomedial thighs, abdomen, and flanks. Lesions include pruritic, papular, crusting eruptions with secondary erythema, seborrhea, alopecia, excoriations, pyoderma, hyperpigmentation, and lichenification.

**Cats**

Cats do not have a pattern unique to flea allergy dermatitis. Patients commonly present with pruritic miliary dermatitis with secondary excoriations, crusting, and alopecia of the neck, dorsal lumbosacral area, caudomedial thighs, and ventral abdomen. Other symptoms include symmetrical alopecia secondary to excessive grooming and eosinophilic granuloma complex lesions.

**Top Differentials**

Differentials include atopy, food hypersensitivity, other ectoparasites (scabies, cheyletiellosis, pediculosis, demodicosis), superficial pyoderma, dermatophytosis, demodicosis, and *Malassezia* dermatitis.

**Diagnosis**

**1.** Lumbar dermatitis in the dog is the most consistent and unique feature of flea allergy dermatitis. In cats, flea allergy should be highly suspected in any cat with skin disease.

**2.** Visualization of fleas or flea excreta on body: may be difficult on flea-allergic animals as flea-allergic animals are very effective at removing fleas through grooming

**3.** Allergy testing (intradermal, serologic): positive skin test reaction to flea antigen or positive serum immunoglobulin (Ig)E antiflea antibody titer is highly suggestive, but false-negative results are possible

**4.** Dermatohistopathology (nondiagnostic): varying degrees of superficial or deep perivascular to interstitial dermatitis, with eosinophils often predominating

**5.** Response to aggressive flea control (nitenpyram administered every other day for 1 month): symptoms resolve

**Treatment and Prognosis**

**1.** Integrated flea management program (Insect growth regulator combined with an adulticide combined with environmental treatments) is essential due to the progressive tolerance of the flea to available adulticides. With time, specific active ingredients typically loose efficacy due to the chronic exposure and genetic drift of the flea.

**2.** Topical or systemic insect growth regulators (lufenuron, piriproxyfen, methoprene) may be effective alone or used in combination with adulticidal therapy.

3. Affected and all in-contact dogs and cats should be treated with adulticidal flea sprays, spot-on solutions, orals, or dips every 7 to 30 days, as instructed on the label. Products that contain spinosid , imidocloprid, selamectin, or fipronil are especially effective when used topically every 2 to 4 weeks. In heavily flea-infested environments, fleas may continue to be found on animals for several months in spite of topical flea control. In these cases, affected animals should also be administered nitenpyram at a minimum dose of 1mg/kg PO every 24 to 48 hours for 2 to 4 weeks, or until fleas are no longer seen. The environment should be treated (see number 5 below).

**4.** Flea-allergic animals should be treated prophylactically with nitenpyram, minimum dose 1mg/kg PO, on any day that an encounter is planned with other potentially flea-infested animals (e.g., a visit to the groomer, veterinary hospital, park, another household with animals). No more than one treatment with nitenpyram should be administered per day.

**5.** In heavily flea-infested environments, areas where pets spend the most time should be treated. Indoor premises should be treated with an insecticide and an insect growth regulator (e.g., methoprene, piriproxyfen). The outdoor environment should be treated with insecticidal or biologic products designed for such use.

**6.** Flea control therapy should be continued from spring until first snowfall in temperate areas and year-round in warm climates. Year-round flea infestations can be perpetuated indoors and on wildlife despite extreme cold outdoors.

**7. Symptomatic Therapy (itch control):**

**a** Topical therapy with antimicrobial shampoos and anti-itch conditioners, and sprays (i.e., those containing oatmeal, pramoxine, antihistamines, or glucocorticoids) applied every 2 to 7 days or as needed may help reduce clinical symptoms.

**b** Systemic antihistamine therapy reduces clinical symptoms in many cases (Table 7-1).

c Systemic glucocorticoid therapy is often effective (75%) in controlling pruritus but almost always result in adverse effects ranging to mild (PU/PD) to severe (immuno-dysfunction, Demodicosis, and calcinosis cutis). It is a therapeutic option if the allergy season is very short but may result in unacceptable adverse effects, especially if used over the long term.

1. Potent, long acting injectable steroids are contraindicated for the treatment of allergies due to their comparatively short anti-inflammatory benefits ( 3 weeks) relative to the prolonged metabolic and immuno-depressive effects (6-10 weeks).

2. Injectable short acting steroids (dexamehtasone Sodium Phosphate, 0.5 – 1 mg/kg or prednisilone acetate 0.1mg – 1 mg/kg) are effective at providing relief and may last 2 to 3 weeks if there are no concurrent secondary infection. This treatment option allows the clinician to more closely control and monitor the patients steroids use compared to oral treatments that are administered by the owner.

3. Temaril-P (trimeprizine and prednisilone combination) is a unique drug that provides significant antipruritic effects at a relatively lower dose of the prednisilone. One tablet should be administered per 10 to 20 kg every 24 to 48 hours. The dosage should be tapered to the lowest possible dose and frequency.

4. Prednisone 0.25 to 1mg/kg (or methylprednisolone 0.2-0.8mg/kg) PO should be administered every 24 to 48 hours for 3 to 7 days. The dosage should be tapered to the lowest possible dose and frequency.

5. All dogs treated with long-term steroids (more than 3 months) should be frequently monitored for liver disease and UTI.

**8.** The prognosis is good if strict flea control is practiced. Fleas may infest other in-contact animals and humans. They may carry blood-borne diseases in a manner similar to ticks.

Author’s note

The use of long-acting, injectable steroids should be stopped due to the profound impact on the metabolic and immune systems as well as the growing concern of legal liability for the practitioner.

Any dog with lumbar dermatitis or any cat with skin disease should be highly suspected of having flea allergy dermatitis even if the patient has been treated with seemingly good flea control therapies.

A nitenpyram trial (every other day for 1 month) is the most efficient and cost effective way to convince the owner and yourself of the role of flea allergy in a pruritic patient.

**#4 Superficial Pyoderma**

**Features**

Superficial pyoderma is a superficial bacterial infection involving hair follicles and the adjacent epidermis. The infection is almost alwayss secondary to an underlying cause; allergies and endocrine disease are the most common causes (Box 3-3). Superficial pyoderma is one of the most common skin diseases in dogs but rare in cats.

Superficial pyoderma is characterized by focal, multifocal, or generalized areas of papules, pustules, crusts, and scales, epidermal collarettes, or circumscribed areas of erythema and alopecia that may have hyperpigmented centers. Short-coated dogs often present with a “moth-eaten” patchy alopecia, small tufts of hair that stand up, or reddish brown discoloration of white hairs. In long-coated dogs, symptoms can be insidious and may include a dull, lusterless hair coat, scales, and excessive shedding. In both short- and long-coated breeds, primary skin lesions are often obscured by remaining hairs but can be readily appreciated if an affected area is clipped. Pruritus is variable, ranging from none to intense levels. Bacterial infections secondary to endocrine disease may cause pruritus, thereby mimicking allergic skin disease.

*Staphylococcus pseudintermedius* (previously *Staphylococcus intermedius*)is the most common bacterium isolated from canine pyoderma and is usually limited to dogs. *Staphylococcus schleiferi* is a bacterial species in dogs and humans that is emerging as a common canine isolate in patients with chronic infections and previous antibiotic exposure. Both *Staphylococcus pseudintermedius* and *Staphylococcus schleiferi* may develop methacillin-resistence especially if subtherapeutic doses of antibiotics or flouroquinilone antibiotics have been previously used in the patient. Additionally, methicillin-resistant *Staphylococcus aureus* (human MRSA) is becoming more common among veterinary species. All three *Staphylococcus* may be zoonotic, moving from humans to canines or from canine to human; immunosuppressed individuals are at most risk.

Causes of Secondary Superficial and Deep Pyoderma

 Demodicosis, scabies, *Pelodera*

 Hypersensitivity (e.g., atopy, food, flea bite)

 Endocrinopathy (e.g., hypothyroidism, hyperadrenocorticism, sex hormone imbalance, alopecia X)

 Immunosuppressive therapy (e.g., glucocorticoids, progestational compounds, cytotoxic drugs)

 Autoimmune and immune-mediated disorders

 Trauma or bite wound

 Foreign body

 Poor nutrition

**Treatment and Prognosis**

**1.** The underlying cause must be identified and controlled.

**2.** Systemic antibiotics (minimum 3‑4 weeks) should be administered and continued 1 week beyond complete clinical and cytological resolution (see Box 3-1).

**3.** Concurrent bathing every 2 to 7 days with an antibacterial shampoo that contains chlorhexidine or benzoyl peroxide is helpful.

**4.** If lesions recur within 7 days of antibiotic discontinuation, the duration of therapy was inadequate and antibiotics should be reinstituted for a longer time period and better attempts to identify and control the underlying disease should occur.

**5.** If lesions do not completely resolve during antibiotic therapy or if there is no response to the antibiotics, antibiotic resistance should be assumes and a bacterial culture and sensitivity submitted.

7. If antibiotics resistance is suspected or confirmed, frequent bathing (up to daily) and the frequent application of topical chlorhexidine solutions combined with the simultaneous administration of two different class antibiotics at high doses seem to produce the best results. Monitoring the infection with cytology and cultures with antibiotic sensitivities is important to determine when the treatments can be stopped. Premature discontinuation of therapy, not completely controlling the primary disease, and the use of fluoroquinilone antibiotics will likely perpetuate the resistant infection.

**8.** The prognosis is good if the underlying cause can be identified and corrected or controlled.

Author's Note:

\*\* Superficial Pyoderma is one of the most common skin diseases in dogs and almost always has an underlying cause (allergies or endocrine disease).

\*\* Cefpodoxime, Ormetoprim/sulfadimethoxine (Primor), and Convenia provide the most consistent compliance which seem to help reduce the development of resistance when used at high doses.

\*\* MRSA, MRSS, MRSI, and MRSP are becoming an emerging problem in some regions of the US.

>>The most likely risk factors include previous exposure to fluoroquinilone antibiotics, sub-therapeutic antibiotic dosing, and concurrent steroid therapy.

>> Daily baths and topical treatments can be very beneficial in the resolution of the infection.

>> Maximize the dose of antibiotics and consider using two antibiotics simultaneously to protect additional resistance from developing.

>> Practice good hygiene (HAND WASHING) to prevent zoonosis.

\*\* Consider screening dogs who visit the elderly or sick to prevent zoonosis. Cultures from the nose, lips, ears, axilla, and perianal areas are best for screening patients for MRS.

**#5 Otitis**

**Features**

Otitis externa is an acute or chronic inflammatory disease of the external ear canal. Its causes are numerous and almost always have an underlying, primary disease (Table 15.1) that alters the normal structure and function of the canal resulting in a secondary infection (Table 15.2). Otitis externa is common in cats and dogs, with Cocker spaniels especially at risk for developing severe and chronic disease.

Otic pruritus or pain is a common symptom of otitis externa. Head rubbing, ear scratching, head shaking, aural hematomas, and a head tilt, with the affected ear tilted down, may be noted. An otic discharge that may be malodorous is often present. In acute cases, the inner ear pinna and ear canal are usually erythematous and swollen. The ear canal may also be eroded or ulcerated. Pinnal alopecia, excoriations, and crusts are common. In chronic cases, pinnal hyperkeratosis, hyperpigmentation, and lichenification, as well as ear canal stenosis from fibrosis or ossification, are common. Decreased hearing may be noted. Concurrent otitis media should be suspected if otitis externa has been present for 2 months or longer, even if the tympanic membrane appears to be intact and no clinical signs of otitis media (drooping or inability to move ear or lip, drooling, decreased or absent palpebral reflex, exposure keratitis) are evident. Rarely, symptoms of otitis interna (head tilt, nystagmus, ataxia) may be present. Oral examination may reveal pain (severe otitis media), inflammation, or masses (especially polyps in cats). Depending on the underlying cause, concurrent skin disease may be seen.

**Diagnosis**

**1.** Based on history and clinical findings

**2.** Otoscopic examination: assess degree of inflammation, ulceration, stenosis, and proliferative changes; amount and nature of debris and discharge; presence of foreign bodies, ectoparasites, and masses; and integrity of tympanic membrane

**3.** Mineral oil prep (ear swab): look for otodectic and demodectic mites and ova

**4.** Cytology (ear swab): look for bacteria, yeasts, fungal hyphae, cerumen, leukocytes, and neoplastic cells

**5.** Bacterial culture (external or middle ear exudate): indicated when bacteria are found on cytology in spite of antibiotic therapy, or when otitis media is suspected

**6.** Fungal culture: indicated when dermatophytic otitis is suspected, especially in long-haired cats that have ceruminous otitis

**7.** Radiography (bulla series), computed tomography (CT), magnetic resonance imaging (MRI): evidence of bullous involvement (sclerosis, opacification) is seen in approximately 75% of otitis media cases

**8.** Dermatohistopathology: may be indicated to identify primary cause (e.g., autoimmune disease, sebaceous adenitis, erythema multiforme), if neoplasia is suspected (ear canal mass), or if ear canal resection or ablation is performed because of end-stage otitis

**Treatment and Prognosis**

**1.** Primary causes of the otitis should be identified and corrected, if possible (Table 15-1).

**2. For swimmer’s ear**, maceration of ear canals can be prevented by prophylactic instillation of a drying agent after the dog gets wet (swimming, bathing), or two to three times per week in very humid climates. Effective products are ear products that contain astringents/alcohol

**3. For allergic otitis**, long-term management includes control of underlying allergies, resolution of any secondary bacterial and yeast otitis, and institution of ear cleaning and treatment every 3-7 days to prevent the recurrence. In animals whose underlying allergies cannot be identified or completely controlled, the judicious use of steroid-containing otic preparations as infrequently as needed may prevent otitis flare-ups

**4. For mild/acute otitis**, at home, the owner should perform ear cleaning every 2 to 7 days with a ceruminolytic agent (that does not need to be flushed out) to prevent earwax and debris from accumulating. Lifelong ear cleaning every 3 – 7 days may be necessary to prevent relapses of otitis. The use of cotton swabs (which may damage the epithelium) is not recommended.

**5.  For severe/chronic otitis**, in-hospital ear cleaning and flushing should be performed to remove accumulated exudate and debris from the vertical and horizontal ear canals (under sedation or anesthesia if necessary). The procedure should be repeated every 2 to 7 days until all debris has been removed.

Products that can be used for ear flushing include the following:

n Water or saline

n DSS diluted in warm water or saline

Non-ototoxic ear cleaning product

n Povidone-iodine 0.2%-1% solution (may be ototoxic)

n Chlorhexidine 0.05%-0.2% solution (may be ototoxic)

n Pretreatment (5 minutes before lavage) with a urea peroxide cleaning product is very effective at dissolving exudate but MUST be flushed out of the canal(may be ototoxic)

**6. Systemic glucocorticoids** should be administered if the ear is painful or the canal is stenotic from tissue swelling or proliferation. For dogs, prednisone 0.25 to 0.5mg/kg PO should be administered every 12 hours for 5 to 10 days. For cats, prednisolone 0.5-1.0mg/kg PO should be administered every 12 hours for 7 to 14 days.

**Individual Diseases**

**7. For ear mites**, affected and all in-contact dogs and cats should be treated. When otic treatments are used, additional treatment to eliminate ectopic mites should be concurrently administered. Effective therapies for ear mites include the following:

n Otic miticide as per label directions (ivermectin and milbemycin products are safe and highly effective)

n Selemectin 6-12mg/kg topically on skin twice 2-4 weeks apart (dogs)

n Tresaderm 0.125-0.25 mL AU q 12 hours for 2-3 weeks

n Ivermectin 0.3mg/kg PO q 7 days for 3-4 treatments, or 0.3mg/kg SC q 10-14 days for 2-3 treatments

n Fipronil 0.1-0.15 mL AU q 14 days for two to three treatments (based on anecdotal reports)

**8. For demodectic otitis**, Effective therapies for ear mites include the following:

n Otic miticide as per label directions (ivermectin and milbemycin products are safe and highly effective)

An alternative treatment is to use 1% injectable ivermectin solution 0.1 to 0.15 mL instilled AU every 24 hours, continuing at least 2 weeks past complete clinical resolution with no evidence of mites on follow-up ear smears.

**9. For yeast otitis**, antifungal-containing ear preparations should be repackaged into a bottle to provide more accurate dosing; dropper bottle, brown amber bottle, etc. Then, 0.2 to 0.4 mL (1/4-1/2 dropperful) should be instilled in the affected ear every 12 hours for at least 2 to 4 weeks. Treatment should be continued until follow-up ear smears are cytologically negative for microorganisms, the external canals are no longer edematous or inflamed, and the ear canal epithelium has normalized.

Effective products include the following:

n Clotrimazole

n Miconazole

n Thiabendazole

n Nystatin

**10.**  **For severe refractory yeast otitis externa or otitis media**, in addition to topical antifungal treatment, systemic antifungal therapy may be helpful if administered for at least 3 to 4 weeks, then continued 1 to 2 weeks beyond complete clinical cure. Effective therapies include the following:

n Ketoconazole 5mg/kg PO q 12 hours, or 10mg/kg PO q 24 hours with food

n Fluconazole 5mg/kg PO q 12 hours, or 10mg/kg PO q 24 hours with food

n Itraconazole 5-10mg/kg PO q 24 hours with food

n Pulse itraconazole 5-10mg/kg PO q 24 hours with food on 2 consecutive days each week

**11. For bacterial otitis**, antibiotic-containing ear preparations should be repackaged into to provide more accurate dosing; dropper bottle, brown amber bottle, etc. Then, 0.2 to 0.4 mL (1/4-1/2 dropperful) should be instilled in affected ears every 8 to 12 hours for at least 2 to 4 weeks. Treatment should be continued until follow-up ear smears are cytologically negative for microorganisms, the external canals are no longer edematous or inflamed, and the ear canal epithelium has normalized.

Effective products include the following:

n Gentamicin

n Neomycin

n Polymixin B and neomycin

n Polymixin E and neomycin

**12. For bacterial otitis media**, Systemic antibiotics may not achieve sufficient tissue concentrations to kill *Pseudomonas* and to prevent antibiotic resistance, the highest possible dose of antibiotic that is safe should be administered with concurrent high-concentration topical therapy of the same antibiotic. If topical has been used aggressively and has been unsuccessful, systemic antibiotics may be indicated, based on culture and sensitivity results, for a minimum of 4 weeks, and continued 2 weeks beyond complete clinical cure.

Antibiotics include the following:

n Ormetoprin-sulfadimethoxine 27.5mg/kg PO q 24 hours

n Trimethoprim-sulfa 22mg/kg PO q 12 hours

n Cephalexin, cephradine, or cefadroxil 22mg/kg PO q 8 hours

n Ciprofloxacin 5-15mg/kg PO q 12 hours

n Enrofloxacin 20mg/kg PO q 24 hours *(may increase the risk of resistant bacteria)*

n Orbifloxacin 7.5mg/kg PO q 24 hours *(may increase the risk of resistant bacteria)*

n Marbofloxacin 5.5mg/kg PO q 24 hours *(may increase the risk of resistant bacteria)*

**13. For *Pseudomonas* otitis**, aggressive treatment should be provided for at least 2-4 weeks, then continued 2 weeks beyond complete clinical cure. All underlying/primary diseases should be identified and addressed. Currently, the most effective treatments include tris–ethylenediaminetetra-acetic acid (EDTA) solutions with high concentrations of antibiotics instilled in high volumes (to ensure deep penetration and prevent dilution by exudate). Antibiotics should be selected according to culture and sensitivity results. Systemic antibiotics may not achieve sufficient tissue concentrations (mutation prevention concentration) to kill *Pseudomonas* and prevent antibiotic resistance. If systemic antibiotics are used, the highest possible dose that is safe should be administered, along with concurrent high-concentration topical therapy of the same antibiotic.

n The tris–ethylenediaminetetra-acetic acid (EDTA) solution should be combined with enrofloxacin to make a 10- to 20-mg/mL solution. The solution should be used q 12-24 hours to completely fill the ear canal. Even as the sole therapy, the surfactants in T8 Solution act to clean the ear while allowing the high concentration of enrofloxacin to penetrate into the deep canal. This treatment is 80% effective in chronic, recurrent otitis cases, even if the bacteria are reported to be resistant to enrofloxacin (because of the tris-EDTA and high concentration of antibiotic)

n Tris-EDTA solution (with/without enrofloxacin 10mg/mL, gentamicin 3mg/mL, or amikacin 9mg/mL) 0.4 mL instilled q 8-12 hours

n Combination 3 mL enrofloxacin (Baytril Injectable 22.7mg/mL) plus 4mg dexamethasone sodium phosphate plus 12 mL ear cleanser: 0.2-0.4 mL instilled q 12 hours

n Amikacin sulfate (Amiglyde V Injectable 50mg/mL), undiluted, 0.1-0.2 mL instilled q 12 hours

n Silver sulfadiazine (Silvadene) 0.1% solution (mix 1.5 mL [1/3 tsp]) of Silvadene Cream with 13.5 mL distilled water, or mix 0.1 g silver sulfadiazine powder with 100 mL distilled water), and instill 0.5 mL q 12 hours

n Ticarcillin equine intrauterine infusion (Ticillin), undiluted, 0.2-0.3 mL q 8 hours

n Ticarcillin powder for injection (vial should be reconstituted as directed, then frozen in TB syringes as 1-mL aliquots). New syringe should be thawed each day and kept refrigerated. A dose of 0.2 to 0.3 mL should be instilled into affected ears q 8 hours

**For End-Stage Ears**

**14. For chronic proliferative otitis**, aggressive medical therapy is needed. Weekly ear cleaning should be instituted. For bacterial/yeast otitis externa and media, long-term (minimum, 4 weeks) systemic and topical antibiotics or antifungal medications should be administered, then continued 2 weeks beyond complete clinical resolution of the infection. To reduce tissue proliferation, prednisone 0.5mg/kg PO should be administered every 12 hours for 2 weeks; then, 0.5mg/kg PO should be administered every 24 hours for 2 weeks, followed by 0.5mg/kg PO every 48 hours for 2 weeks. These ears rarely return to complete normalcy, so long-term maintenance therapy with steroid-containing otic preparations, as described for allergic otitis, is almost always necessary.

**15. For end-stage ears, indications for surgery** include the following:

n Traction-avulsion or surgical resection of inflammatory polyps/masses

n Lateral ear canal resection, which aids in ventilation and drainage and allows for easier application of medication but rarely results in cure because a large amount of diseased tissue is still present

n Vertical ear canal ablation, if proliferative changes are present in the vertical canal but the horizontal canal is not affected. Total ear canal ablation and lateral bulla osteotomy is usually indicated to alleviate chronic pain and discomfort when end-stage otitis externa and otitis media are no longer responsive to medical management

**16.** The prognosis is variable, depending on whether the underlying cause can be identified and corrected, and on the chronicity and severity of the otitis externa. Because Cocker spaniels are especially at risk for chronic and severe otitis externa, early and aggressive management of primary otitis externa and secondary inflammation is warranted in this breed.

**Author’s Note:**

The most important component for successful long-term treatment of otitis are

1. Identify and control the underlying, primary disease; infectious otitis is secondary and recurrence can only be prevented if the primary disease is treated.
2. Use sufficient volumes of otic medications to completely coat and penetrate the ear canal; most patients need a minimum of 0.25 ml to provide enough medication.
3. Don’t treat and stop; treat and stop but rather use frequent (every 3-7 days) cleaning or treatment to prevent otitis recurrence.

It is extremely important to base treatment decisions on cytology (initial and recheck) and clinical impression together.

The most consistently successful and least ototoxic treatment for Pseudomonas otitis is a high concentration enrofloxcin solution mixed into a trisEDTA (making a 10mg/ml final solution). If the infection is not improving, consider a deep otic lavage and evaluation of the otic bullae. Systemic antibiotics do NOT seem to improve treatment efficacy beyond the topical solution alone.