THE PRURITIC DOG

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INTRODUCTION

Pruritus can be defined as the sensation that elicits the desire to scratch and/or chew and is assumed to occur in dogs that show erythema, excoriations, alopecia, lichenification, or hyperpigmentation. Pruritus is the most common clinical manifestation of disease that causes owners to bring their pets in for treatment; however, the diagnosis of pruritic dermatitis is not always easily, partly because of the variation in clinical presentation and partly because of the range of different aetiologies. The diagnostic approach must thus be through and methodical and should include certain fundamental diagnostic steps.

PATHOPHYSIOLOGY

The skin functions as an "external nervous system," providing continuous sensory input to the central nervous system (CNS) through a network of free nerve endings responsible for transmitting the sensations of touch, temperature, pain, and pruritus. In humans, "sensory spots" or "itch points" coincide with areas of increased density of free nerve endings, which may be true in the dog as well. Pruritus commonly stimulates self-trauma; however, the mechanisms by which self-trauma relieves itching are unclear. Possible mechanisms would be that self-trauma may disturb the amplified, reverberating spinal pathways that perpetuate the sensation of itch and that self-trauma could substitute pain for pruritus.

Endogenous mediators that can trigger pruritus include histamine, peptides, proteases, prostaglandins, leukotrienes, monohydroxy fatty acids, and opioid peptides. Proteolytic enzymes and leukotrienes are thought to be more important mediators of pruritus than histamine. Bacterial and fungal endopeptidases also can initiate pruritus. The CNS can also amplify or reduce the sensation of pruritus: stress or anxiety may amplify pruritus by releasing opioid peptides; boredom or other cutaneous sensations such as pain, heat, cold, or touch can alter the perception of pruritus; and factors such as increased skin temperature, diminished skin hydration, and low humidity can heighten the sensitivity of the skin to pruritic stimuli. The pruritic threshold is often reduced at night in both humans and animals when other sensory inputs are diminished.

The concepts of *threshold phenomenon* and *summation of effect* are paramount in understanding and managing pruritus. The *threshold phenomenon* is where a certain pruritic load may be tolerated without initiating clinical signs, but a small increase in that load can provoke clinical signs. *Summation of effect* occurs when additive pruritic stimuli from coexistent skin diseases raise an animal above threshold. As an example, pruritus from mild flea allergy is additive to pruritus from other skin diseases during flea season, thus exacerbating and perpetuating itch-scratch cycles.

DIAGNOSIS OF PRURITUS

Signalment, history, clinical examination, diagnostic testing, and, occasionally, response to therapy are the cornerstones of diagnosis. Because many pruritic skin diseases are visually similar, clinical history coupled with signalment may offer more direct clues to diagnosis than the actual clinical examination.

Signalment

Age

Age provides critical information for prioritizing differential diagnoses. Scabies and demodicosis are pruritic skin diseases seen more commonly in young dogs whereas atopy, food allergy, and pyoderma occur more commonly in adult animals.

Breed
Certain skin diseases are breed specific: golden retrievers, Dalmatians, retrievers, beagles, and many small terrier breeds are at increased risk for the development of atopy; the West Highland white terrier for secondary *Malassezia* dermatitis; and the Chinese Shar Pei seems predisposed to atopy, food allergy, pyoderma, and demodicosis.

Sex
Although sex predilections are not common in pruritic skin diseases, pruritus may be seen with Sertoli cell tumours, male-feminizing syndromes, and female hyper-oestrogenism.

Historical findings
General history
A general history pertaining to diet, environment, use of home skin care, recent exposures, and the presence or absence of pruritus in other animals or people in the environment should be obtained, which may aid in prioritizing differential diagnoses.

Diet
Food allergy or intolerance can cause pruritus; however, adverse reactions to food frequently coexist with other allergic skin diseases such as atopy and flea allergy dermatitis. Lipid-deficient diets may exacerbate seborrhoea.

Environment and exposure
Flea allergy, scabies, and cheyletiellosis are all seen more frequently in dogs that are permitted to roam free. Acquisition of a new pet, sheltering a stray animal, recent kennelling, and grooming can all increase the likelihood of contagious disease.

Other household pets
Pruritus or lack of pruritus in other animals may offer clues to contagion. However, even though dogs and cats share the cat flea as a common ectoparasite, flea allergy is much more common in dogs. A seemingly unaffected cat is often the source of fleas in indoor dogs with flea allergy dermatitis. Although uncommon, asymptomatic carriers of canine scabies do exist because clinical disease requires hypersensitivity.

Human contacts
A pruritic papular rash in an owner with a pruritic pet may suggest zoonotic infestation with scabies or cheyletiellosis. Annular, erythematous lesions may suggest dermatophytosis.

Specific history
Specific history relates to the current pruritic skin disease. The initial site of skin lesion development, onset and progression, intensity of pruritus, seasonality or other pattern, and response or lack of response to previous therapy may aid in establishing a diagnosis.

Site, onset, and progression
Knowledge of the initial sites of skin lesions may be useful, especially if the disease has generalized before veterinary care is sought. Scabies often begins on the margins of the pinnae before generalizing. Rapid-onset pruritus should increase suspicion for ectoparasitic diseases and, less commonly, adverse drug reactions. Pruritus of insidious onset is more suggestive of slowly progressive, chronic skin diseases such as atopy, food allergy, pyoderma, seborrhoea, and *Malassezia* dermatitis.

Intensity
Most animals do not exhibit pruritus in the examination room with scabies and flea allergy dermatitis being notable exceptions. Frequency and intensity of pruritus may be inferred from asking the owner how many times the animal will scratch (or chew or lick) if it is ignored while the owner observes the animal at home.

Seasonality
Atopy and flea allergy dermatitis are often seasonal; *Malassezia* dermatitis may occur more frequently during months of higher humidity; cyclical pruritus without seasonality can sometimes signify contact

dermatitis associated with change of environment; and psychogenic pruritus may begin as a predictable, attention-getting device. Pruritus seen with food allergy should be continuous unless the diet is changed.

Response to previous therapy
Response or lack of response to previous medications, particularly corticosteroids, antibiotics, or parasiticides, may offer additional clues. Although allergic diseases all respond to corticosteroids to some degree, food allergy may be less responsive to corticosteroids than atopy or flea allergy dermatitis. Prior diminished pruritus in response to antibiotics in dogs is often overlooked and indicates the likelihood of pyoderma. Pruritus as the result of pyoderma may also diminish in response to corticosteroids.

Clinical examination
A complete clinical examination is extremely important when evaluating any animal with skin disease as skin disease may be an indicator of systemic disorders. Examination of the skin, muco-cutaneous junctions, oral cavity, ears, genitals, and lymph nodes should be emphasized. Objective signs of pruritus include excoriations and broken or barbered hairs with a dry lusterless hair coat.

Pruritus may occur with or without primary skin lesions. If present, primary skin lesions such as papules or pustules may be helpful in establishing a diagnosis. Coexistent alopecia may offer additional clues. Unfortunately, self-trauma often leads to the obliteration of initial, more diagnostic primary skin lesions substituting excoriations, lichenification, and alopecia. The concept of "a rash that itches" indicates primary skin lesions that are itchy, and "an itch that rashes" indicates that pruritic patients without primary lesions traumatize themselves. Ectoparasitic skin diseases, pyoderma, and cornification abnormalities are among the more common pruritic skin diseases where primary skin lesions are identified. Conversely, primary lesions are much less common in atopy and food allergy. The distribution of lesions, presence or absence of bilateral symmetry and major foci of pruritus can be valuable aids to diagnosis. Primary or secondary lesions, if present in a particular site, may be highly suggestive of specific diseases.

Diagnostic plan
Diagnostic plans should be formulated based on prioritization of differential diagnoses using signalment, history, and physical findings with specific diagnostic procedures selected based on the most likely differential diagnoses. Skin scrapings, fungal culture, exfoliate cytology, trial therapy for ectoparasites, and skin biopsy, are the most cost-effective diagnostic procedures for the pruritic animal.

Skin scrapings
Multiple skin scrapings should be performed on all pruritic dogs. Affected areas should be gently clipped, and then either a scalpel blade or glass slide dipped in mineral oil, should be scraped perpendicular to the skin surface in the direction of hair growth. The acquired debris is then placed on a slide, a cover slip applied, and the specimen examined microscopically using low light. Demodex mites usually are readily demonstrable (except in chronic pododemodicosis and in the Chinese Shar Pei). Scabies mites are documented in less than half of affected dogs, underscoring the need for trial therapy in suspected cases. Dry scrapings may be stained as smears to look for *Malassezia*.

Exfoliate cytology
Affected skin, intact pustules, or exudates should be smeared, stained with a rapid stain such as Diff Quik®, and examined microscopically for the presence of bacteria, *Malassezia*, and inflammatory cells. Clear tape preparations may demonstrate *Cheyletiella* and stained tape preparations may demonstrate bacteria or *Malassezia*.

Faecal examination
Faecal examination may document endoparasite infestations and may reveal the presence of mites.

Skin biopsy
Skin biopsy is especially valuable if primary skin lesions are free of self-traumatic excoriations. If only self-traumatic lesions are present, definitive diagnosis is less likely, but results may aid in prioritizing or ruling out various differential diagnoses.

Response to trial therapy
Trial therapy with parasiticidal agents is used routinely in suspected cases of scabies or flea allergy dermatitis. Despite the availability of effective modern flea control products, flea allergy dermatitis still remains the most common cause of pruritus. As lesions seen with superficial pyoderma may be pleomorphic, trial use of antibiotics may be indicated in an undiagnosed pruritic crusted papular dermatoses. Although response to corticosteroids is suggestive of underlying allergic disease, superficial pyoderma may partially respond to corticosteroid therapy.

Elimination diets
Animals suspected of having food allergy or food intolerance as a cause of pruritus should be fed an exclusive hypo-allergenic diet for 8-12 weeks.

In vitro testing
In vitro testing (allergen-specific IgE serology [ELISA] or radioallergosorbent test [RAST]) for atopy offers convenience and accessibility. Reproducibility of test results has increased dramatically over the past decade. However, problems still remain with antigen selection, grouped testing, and standardization of results.

Environmental restriction
If allergic contact dermatitis is suspected, an animal may be housed in a markedly different environment (water-rinsed kennel) for 10 days.

MANAGEMENT OF PRURITIS

The general considerations in managing the pruritic dog are avoidance of allergens (whenever possible), using topical and/or systemic medications, considering desensitisation therapy, and treating concurrent problems.

Environmental control is usually aimed at limiting exposure to offending allergens. For pollen allergies, keeping the dog indoors most of the day is partially effective. Bathing in cool water will soothe the skin directly but will also wash away pollens or moulds that might still be present on the skin or hair coat. Topical anti-pruritic agents include colloidal oatmeal, hydrocortisone, antihistamines (diphenhydramine), and anaesthetics (pramoxine).

Corticosteroids are often effective at managing the pruritus but must be used cautiously for long-term therapy. A starting dose of prednisolone is usually 0.5 to 1 mg/kg/day for 3-5 days. Long-term therapy should only be used on an alternate-day basis.

Cyclosporine at 5 mg/kg/day is often sufficient in controlling clinical signs without many of the long-term side effects of corticosteroids. Treatment is given daily for 6 weeks; after that, it is often possible to treat on an alternate-day basis, and sometimes even twice per week.

Antihistamines are reported to be satisfactory anti-pruritics in approximately 25-35% of atopic dogs with clemastine, hydroxyzine, chlorpheniramine, and diphenhydramine giving the highest percentage of control.

Combinations of omega-3 and omega-6 fatty acids (eicosapentaenoic acid and gamma linolenic acid), have been shown to have beneficial effect in approximately 20% of atopic dogs. Treatment should be based on the eicosapentaenoic acid (EPA) content of these products and dosed at 5-40 mg/kg of EPA daily. It takes approximately 12 weeks of supplementation to change leukotriene levels in the skin and blood.

Desensitisation therapy is a biologic method used to treat atopic dogs. Although the exact mechanism by which immunotherapy works is unknown, it is likely that it “down regulates” the allergic response, and may raise the pruritic threshold. Desensitisation therapy should be considered when the allergic signs last for 4 months or more each year; when there are side effects from medical treatment, regardless of the duration of allergic signs; and if the pruritus is not adequately controlled with medical treatment. Success rate of desensitisation therapy is approximately 60-75% and requires, on average, 4-12 months for the beneficial effects to be seen.

MANAGEMENT OF PYODERMA

In general pyoderma requires systemic treatment with antibiotics, although topical anti-microbial shampoos may also be effective. In all cases precipitating underlying disease should be sought and recurring previously well-managed atopic dogs may require antibiotic therapy to eliminate secondary bacterial aetiologies and not an increase in steroid dosage (bacterial “summation of effect”).

General principles
The antibiotic should have known spectrum of activity against *Staphylococcus intermedius* and be beta-lactamase resistant. Only about 40% of dermal blood flow reaches the dermal/epidermal junction and therefore in vitro sensitivity testing may be poorly correlated with clinical response. For cultures proper sampling technique is essential with superficial skin swabs generally unacceptable. A tissue biopsy is usually required.

Proper therapeutic dosing for the full duration of treatment is essential and the minimum treatment time for a superficial pyoderma is 14-21 days and for a deep pyoderma 8-12 weeks.

Antibiotic selection

- Erythromycin (10 - 15 mg/kg tid).
- Lincomycin (22 mg/kg bid, at least 2 hrs away from food).
- Clindamycin (10 mg/kg bid).
- Potentiated sulphonamides (20 –30 mg/kg bid).
- Cloxacillin (20 mg/kg bid).
- Amoxicillin with clavulanic acid (20 mg/kg bid).
- Cephalosporins (20 mg/kg bid-tid).

Poor choices include penicillin, ampicillin, amoxicillin, streptomycin, tetracycline, and non potentiated sulphas.