THE FOUR STEP APPROACH TO THE PRURITIC DOG

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Pruritus is the most common presenting dermatology complaint of pet owners. The itchy dog's symptoms may be manifested by licking, chewing, biting and/or rubbing the skin, resulting in visible alopecia, erythema, dermatitis and odor.

To establish a diagnosis when faced with the pruritic dog, note the signalment – the age, breed, and gender as some breeds are more likely to have certain diseases (ex. brachycephalic puppies-demodicosis, young Labrador and Golden retrievers-atopic dermatitis, German shepherds- food allergy, West Highland White terriers- Malassezia dermatitis, atopic dermatitis). Dogs under 6 months old are more likely to have parasitic or infectious causes of pruritus (sarcoptic mange, demodicosis, dermatophytosis) or food allergy, onset of pruritus between 6 mo and 3 years is typical of atopic dermatitis while older dogs may be more prone to endocrine disease or cutaneous neoplasia. A middle-aged to older dog with no prior history of skin disease who suddenly becomes intensely itchy should have sarcoptic mange ruled-out first.

Take the pet with skin disease to a designated dermatology examination room as soon as possible: avoid having them climb up on waiting room chairs or mingle with other pets as this can spread parasites or infection Obtain a thorough history from the owner. This can be made easier by making sure your appointment time is long enough: a minimum of 30 minutes is needed for any dermatology patient. Try to work with the primary caregiver of the pet. Have your front office request the owners bring all the previously used medications, shampoos, ear cleansers, ear medications and food and treats. Using a dermatology history form, ideally linked to your hospital website, to be completed in advance will greatly facilitate and standardize the history taking process.

Needed historical information for a dermatology patient:

1. Age the dog was obtained
2. Source of the dog- breeder, pet store, shelter/ rescue, stray
3. Age at onset of the pruritus
4. Owner's observations regarding the distribution of the pruritus
5. Past and current diet/treats including a list of ingredients
6. Where the dog spends time (indoors, outdoors, hiking in the woods, swimming, free-roaming)
7. Exposure to other animals (wildlife, dog park, training classes, grooming parlor)
8. Pruritus of other pets or people
9. Previous occurrences and any observed seasonality
10. Previous therapies and response to those therapies, esp. glucocorticoids
11. Routine medications, shampoos, and flea/ heartworm/internal parasite control

Evaluation of the dog's response to therapy such as antibiotics, antiparasitics, antihistamines, or corticosteroids may provide information useful in making a diagnosis. For example, a pruritic

dog that is highly responsive to very low doses of corticosteroids is likely to have atopic dermatitis, whereas the dog that fails to respond to anti-inflammatory doses of corticosteroids may have sarcoptic mange, flea allergy dermatitis, food allergy or Malassezia dermatitis.
It is helpful if it can be determined from the history whether the dog has "an itch first that rashes later" or "a rash first that itches later". The former may support the presence of an allergic dermatitis (chronic pruritus causing secondary excoriations and erythema), while the latter would be consistent with an inflammatory or infectious disease that has resulted in secondary pruritus (i.e. bacterial pyoderma due to hypothyroidism).

Perform a complete general physical examination with attention to lymph nodes, ears, and all mucous membranes. Next perform a thorough examination of all skin surfaces. Wear gloves when examining patients with skin disease and wash/ santitize your hands before and after each examination so to not spread (resistant) Staphylococcal infections from one pet to another or to hospital surfaces and equipment. Dermatology diagnostic equipment needed is dedicated clippers and otoscope, box of disposable gloves, sharpie marker, glass slides, cotton swabs, clear single-sided and double-sided tape, mineral oil, scalpel blades, culturettes, 25 gauge needles for obtaining cultures. Using a new sterilized “derm pack” for each dermatology patient, as is done in human hospitals- will reduce fomite spread of parasites and resistant infection. This pack would include clipper blade, otoscope cone, flea comb, scalpel blade, microspatula.

Start the dermatology exam by performing a pinnal-pedal reflex and doing a thorough flea combing looking for fleas, flea dirt or lice. If the patient is not too large or too fractious I prefer to examine them on a table with good lighting; it is amazing how much you can miss while examining a dog on the floor in a dark corner of the exam room. After examining the head, pinnae, ear canals, neck, legs, paws, dorsum, sides, tail and perineal areas, the patient should be placed in lateral recumbency by an assistant to examine the axilla, groin, ventrum, interdigital skin and nails/ nailbeds, and scrotal/ vaginal skin folds. Clip matted or thick hair away for you and the pet owner to fully appreciate the full extent of lesions. Pay extra attention to skin folds in brachycephalic breeds (face, lip, tail). Note any alopecia, indicate if it is partial or total, and record the locations. The presence of primary lesions such as erythema, papules, pustules, vesicles, bulla, or nodules should be noted. Next record any secondary lesions such as scales, crusts, excoriations, erosions, ulcers, purulent or greasy/ seborrheic discharge, hyperpigmentation and lichenification. Indicating lesions on a drawing of the dog’s body or using digital photographs at each visit is helpful to determine disease progression and response to treatment.

This complete written and photographic description of the dog’s general and cutaneous physical exam findings allows later comparisons of the dog’s skin condition for you, the dog’s owner (who may not see gradual improvement), other veterinarians in the practice, the dermatologist who may see the dog and for a dermatopathologist who may be reading the patient’s skin biopsy in the future.

Now move to the standard dermatologic diagnostic tests. Recommended diagnostics for pruritic dogs are: skin scraping, wood’s light, skin cytology, ear smear for mites and cytology (if any otic discharge), and bacterial culture if recurring problem.

Multiple superficial and deep skin scrapings or double-sided tape preparations are performed to identify the presence of external parasites such as *Sarcoptes* or *Cheyletiella* as well as *Demodex* follicular mites. Cytologic examination of the contents of a pustule, of purulent material beneath a crust, or of debris collected from the surface of the skin can identify the presence of *Malassezia* yeast, *Staphylococci*, or other bacteria. Remember for tape preparations that only the third purple Diff Quik stain is used.

A Wood's light examination will demonstrate positive fluorescence in about 50% of the *Microsporum canis* infections and will aid in the collection of hairs or scale for a fungal culture (all puppies, in adult dogs with negative cytology and scrapes and that have failed to respond to antibiotic therapy).

Samples for bacterial culture can be collected and submitted if indicated, such as failure to respond to previous antibiotic therapy, recent hospitalization or a history of multiple antibiotics used in the last 6 months.

A skin biopsy may be indicated if the skin lesions appear unusual or extremely severe, if the mucous membranes are affected, if the patient has failed to respond to rational therapy, if you suspect an autoimmune or neoplastic skin disease.

Four Step Diagnostic Approach for the Itchy Dog

STEP 1: Ectoparasitic Causes of Pruritus
Rule-out:

- **a. Fleas**
- **b. Scabies[1], Cheyletiella, Otodectes, lice**
- **c. Demodex**

Fleas

Ctenocephalides felis is the most prevalent species of flea found on dogs and cats. Flea bite hypersensitivity (flea allergy dermatitis; FAD) is the most common hypersensitivity disorder of dogs and cats. Allergenic components of flea saliva injected into the skin during blood sucking feeding behavior can elicit both an immediate and delayed type hypersensitivity response. Affected animals generally manifest their pruritus on the caudal half of the body including the tailhead, caudal thighs, and ventral abdomen. Lesions are usually secondary to pruritus and consist of partial alopecia, papules, erythema, lichenification, hyperpigmentation, and excoriations.

The diagnosis of flea allergy dermatitis is made using several pieces of information. The owner should be questioned about observation of fleas or flea feces ("flea dirt"), and whether year round flea control is applied to all pets. On physical examination, the presence fleas or flea feces on the dog or cat is diagnostic but the location of pruritus to dorsal lumbar, caudal thigh region is also diagnostic.. Many flea allergic animals have no evidence of fleas or flea dirt due to active grooming or pruritic behavior.

Recent studies by Dr. Michael Dryden at Kansas State University show that flea allergy dermatitis is related to the degree of hypersensitivity of the individual animal, number of fleas, and amount of antigen injected through blood sucking behavior.[9] Thus, reduced flea burden and decreasing flea feeding time rather than complete elimination leads to clinical improvement in FAD.[9] This is in contrast to the commonly believed "one flea bite is all it takes" dogma that is prevalent in the veterinary profession.

Fleas may bite humans in severe infestations and can carry zoonotic blood-borne diseases such as bartonellosis that can also be passed to people via contact with flea feces through breaks in the skin barrier on your hands. Fleas transmit tapeworm eggs to animals.

Sarcoptes scabiei

Sarcoptes scabiei mites are the cause of one of the most intensely pruritic skin diseases of dogs. The mites are also known to cause disease in foxes, humans and rarely in cats. Many cases have a history of exposure to wildlife or their burrows, boarding kennels, grooming facilities, stray dogs or shelters. The mites burrow through the superficial cornified layer of the epidermis where they lay eggs. The pruritus is most likely caused by an allergic (IgE-mediated) reaction to the intestinal proteins secreted in the mite fecal material. Puppies, very old dogs, dogs treated with immunosuppressive doses of corticosteroids, or dogs that have not yet developed an immunologic reaction to the mites may be asymptomatic carriers, but most animals are very pruritic. A history of more than one dog in the house being severely pruritic is suggestive of scabies. Mites are zoonotic and approximately 10 -50 % of people exposed to the affected dog will develop a pruritic papular eruption of the arms, thighs and trunk. The elderly, children and immunosuppressed people are more likely to be infected. Mites can survive for 2-6 days at room temperature in the environment.

Affected dogs exhibit generalized intense pruritus with the edges of the pinnae, elbows, hocks, and ventral abdomen having the most severe lesions. Alopecia, erythematous papules, thick, yellowish crusts, and excoriations are noted on physical examination. In many dogs a positive pinnal-pedal reflex can be elicited by rubbing the edge of the dog's pinnae with the thumb and forefinger (gloves, please) and noting an attempt to scratch the ear region with the ipsilateral hind leg- this test is 80-85% diagnostic for scabies. Chronic cases may have weight loss and lymphandenopathy. Some cases exhibit only severe pruritus without skin lesions; this is especially seen in small frequently groomed pampered dogs and is termed "scabies incognito".

Collection and identification of the mites can be difficult. The ear margins, elbows, or hocks are most likely to yield a positive scraping, but scrapings are positive only 25-30 % of the time. At least 4to 5 superficial scrapes, collecting as much crust as possible, should be performed. One mite, egg or fecal pellets is diagnostic. A presumptive diagnosis of scabies can be made by a positive response to treatment with a miticidal product. All in-contact dogs need to be treated. Infected people should be referred to their physician or dermatologist for treatment (the disease is self-limiting in healthy people).

***Cheyletiella* spp.**
Cheyletiella spp. mites affect cats, dogs, rabbits, and humans. The life cycle is completed on one host within 21 days. Larvae, nymphs, and adult male mite are thought to die soon after leaving the host but the female may live free of the host for up to 10 days. The mites are highly contagious. Eggs are shed into the environment with the pet's hair and scale and may be an important source of re-infestation.

Signs in infested animals range from none to an intensely pruritic dorsal dermatitis. A dry scale, dorsally oriented, and loss of undercoat secondary to the pruritus are the most common clinical signs.

Collection and identification of the mites can be difficult. The most reliable method involves using a flea comb but some veterinarians collect scales and debris with double-sided tape for microscopic examination. *Cheyletiella* mites and eggs can sometimes be identified in a fecal flotation. Like scabies the diagnosis made be made from response to miticidal treatment.

***Demodex* spp.**
The mite *Demodex canis* is an obligatory parasite of dog skin and is present in small numbers on all dogs residing in hair follicles, sweat glands, and sebaceous glands. The entire life cycle of the mite is spent on the dog. *D. canis* mites feed on skin cells, sebum, and epidermal debris. There are four stages in the life cycle: egg, larvae (6 legged), nymph, and adult (both 8 legged). Mites are transmitted by direct contact from the bitch to the puppies within the first 3 days of life. A "long-tailed" follicular mite (*Demodex injai*) has been recently identified that causes a pruritic greasy seborrhea in terrier breeds.

The disease demodicosis is caused when *D. canis* mites proliferate beyond the capacity of the host's immune system to control the mite population. There is some evidence that generalized demodicosis is a manifestation of a hereditary mite-specific T-cell defect that allows the mite population to increase, inducing further T-cell suppression. The clinical lesions include hair loss, erythema, and secondary deep pyoderma. Clinical disease is by convention classified as either localized (LD) or generalized (GD).

Veterinary dermatologists have defined localized demodicosis (LD) as a dog with fewer than 5 lesions in fewer than 2 body regions. No more than 1 paw should be involved. Pruritus is variable. Lesions usually are mild and consist of alopecia, erythema, and mild scaling or crusting, especially periocularly, around the commissures of the lips and on one paw. LD tends to be a benign disease that most often spontaneously resolves within 3 months.

Generalized demodicosis (GD) differs from localized disease in clinical appearance, possible underlying cause(s), and prognosis. Lesions may consist of one or more of the following: alopecia, erythema, scaling, crusting, comedones, hyperpigmentation (bluish discoloration of skin with sharp demarcation between normal and abnormal skin), lichenification, and/or draining tracts. A dog is diagnosed with GD if it has 5 or more localized lesions, involvement of an entire body region, or involvement of 2 or more feet. The prognosis is usually related to the age of onset of the disease. In dogs that develop disease at less than 18 months of age (juvenile onset) there is a 30-50 % chance of self-cure. The possibility of a spontaneous cure is virtually zero in

the dogs that develop GD as adults. Generalized demodicosis occasionally can be found affecting only the paws, and is the first rule out in any dog presenting with pododermatitis. Secondary deep pyoderma and furunculosis are common and occasionally a dog can develop sepsis. Certain breeds of dogs have been shown to have a statistically higher risk of developing juvenile-onset GD, including brachycephalic breeds such as boxers, pugs, and English bulldogs; Chinese shar peis, American cocker spaniels, collies, Doberman pinschers, German shepherd dogs, Great Danes, Old English sheepdogs, pit bull terriers, and Staffordshire terriers. Since there is evidence for a hereditary predisposition to develop GD, affected animals should not be bred.

In dogs with adult-onset GD (disease starting after 4 years of age), a thorough work-up should be done to identify any possible underlying cause, such as hyperadrenocorticism (most common), hypothyroidism, and underlying neoplasia or chronic corticosteroid use, although approximately 50 % of cases are idiopathic.

The diagnosis of demodicosis usually is easily made. A deep skin scraping should be performed from several areas making sure that red blood cells are visible on the slide. A skin biopsy may be necessary to make the diagnosis in the Chinese shar pei, some bulldogs, dogs with chronic, fibrosing, granulomatous lesions, and dogs with pododemodicosis. It has recently been shown that plucking hairs from affected sites and examining the area around the hair bulbs in mineral oil will also demonstrate the mites, although the numbers may be fewer than are seen on a scraping. This is useful in delicate areas such as the periocular skin.

Microbial Caused Pruritus

<u>STEP 2:</u> Identify and treat bacterial and yeast infections

Staphylococcal Pyoderma

The word pyoderma is commonly used to refer to a bacterial colonization or infection of the skin and/or hair follicle. An infection is the combination of the invasion of the body by a pathogenic microorganism and the reaction of the tissue to their presence and to the toxins generated by them. Bacterial pyoderma is second only to flea allergy dermatitis as the most commonly diagnosed dermatosis of dogs, but this statistic is probably changing with the advent of newer flea-control products.

Staphylococcus spp can be identified in about 94% of the cases of canine bacterial pyoderma and *S pseud intermedius* comprises approximately 84% of these pyodermas. *S schleiferi* subsp *coagulans* and *S aureus* have been isolated from approximately 8 % and 1 % of cases of staphylococcal pyoderma in dogs, respectively. Successful management of bacterial pyoderma requires recognition of the type of pyoderma, the causative or underlying factors present, correct antibiotic treatment and appropriate topical therapy.

Canine bacterial pyodermas are classified by depth of involvement, pattern, location or clinical outcome. A surface infection or colonization involves the stratum corneum. These include such conditions as intertrigo (skin-fold pyoderma) and pyotraumatic dermatitis (“hot spots”). Superficial pyoderma is the most commonly diagnosed canine bacterial skin disease. The infection involves the epidermis below the stratum corneum and/or extends into the hair follicle.

Impetigo, superficial folliculitis, and superficial spreading pyoderma are examples of this type of infection. A deep pyoderma occurs when the infection extends through the epidermis or hair follicle and involves a pyogenic inflammation of the dermis or subcutis. Often there is evidence of hair follicle rupture. In addition to *Staphylococcus*, gram-negative bacteria such as *Proteus, Pseudomonas, Klebsiella* or *Escherichia coli* can be cultured from deep pyodermas.

The presence of bacterial pyoderma is always secondary to an underlying cause. Recurrent pyoderma is defined as a bacterial infection of the skin that responds entirely to appropriate systemic and / or topical therapy, but recurs shortly after cessation of therapy, usually within a month. It is the obligation of the veterinarian to try to determine the precipitating cause and to treat or eliminate it.. Unfortunately, this may be easier said than done. The most common disorders complicated by secondary pyoderma are the allergic diseases such as flea allergy dermatitis, food allergy, or atopic dermatitis. The pyoderma will generally worsen the pruritus. These allergic diseases may also have *Malassezia* overcolonization.

Uncommon causes of pyoderma are diseases of cornification/keratinization such as idiopathic "seborrhea", icthyosis, and sebaceous adenitis which alter the normal surface microenvironment and allow the overgrowth of bacteria. Endocrine disorders: hypothyroidism, hyperadrenocorticism (either iatrogenic or naturally-occurring) cause changes in the cornified layer predisposing to secondary bacterial pyoderma. Genodermatoses that cause cutaneous anatomic abnormalities (color dilution alopecia, black hair follicular dysplasia, and follicular dysplasia) typically have secondary bacterial overgrowth.

The diagnosis of pyoderma involves evaluation of physical exam findings and cytologic examination of skin surface or lesion exudates. In some situations such as previous antibiotic therapy or lack of response to rational therapy additional diagnostic steps such as culture and sensitivity or skin biopsy may be needed. . If a dog with pyoderma is not showing clinical improvement while being treated with an empirically selected antibiotic a bacterial culture and susceptibility test should be performed. If a dog with pyoderma is not showing clinical improvement while being treated with an empirically selected antibiotic a bacterial culture and susceptibility test should be performed.

Characteristic clinical lesions are erythema, alopecia, pustules, papules, crusts, and epidermal collarettes. With a deep pyoderma, there are often nodules, erosions, ulcers, and draining tracts. If cytologic examination of exudate from skin lesions shows large cocci, usually in pairs, tetrads or clumps, it is highly suggestive of pyoderma caused by *S pseudintermedius*. The concurrent presence of rods is indicative of a mixed infection. When bacteria are seen within the cytoplasm of a neutrophil it confirms that they are being actively phagocytized and are not just contaminants.

Yeast (Malassezia) Dermatitis

Malassezia sp. are lipophilic, non-mycelial, saprophytic yeast that can be found on the skin (esp. axilla, groin and interdigitally), in the ears, anal sac, rectum, and vagina of normal dogs and cats. In the dog, disease is caused by colonization or infection with Malassezia pachydermatis. In the normal animal, commensal bacteria and yeast and the animal's immune system keep the

Malassezia from overgrowing and causing disease. If the skin barrier is "disrupted" as in allergic disease, the immune system compromised, or there is a change in the microclimate (i.e. skin folds), the yeast can increase in numbers and cause disease. The most common underlying disorder is allergiy but conditions such as skin folds, seborrhea, or endocrine imbalances may rarely be identified. If yeast found think allergy. Breeds of dogs that are predisposed to develop yeast overcolonization include the West Highland white terrier, German shepherd, shih tzu, basset hound, cocker and English springer spaniel, maltese and dachshund.

Clinical signs of Malassezia dermatitis can include moderate to severe non-steroid-responsive pruritus, erythema, greasiness, waxiness, scaling, hyperpigmentation, lichenification, and rancid odor. Lesions can be focal, multifocal or generalized. Common sites of involvement include the lip folds, ear canals, ventral neck, abdomen, interdigital and ventral paws, perianal area, and skin folds. The affected areas may be sharply demarcated.

The diagnosis is best made using cytology. Direct impression smears are the method of choice. The glass slide is pressed or rubbed firmly several times onto the skin surface to collect loose surface material. A cotton swab is useful for collecting material from the ear canals and interdigital spaces/ nailbeds. A skin scraping can be used to collect material from the skin and base of the nails (brown discoloration). The slide is stained with Dif Quik®. Malassezia are oval to peanut-shaped budding organisms that stain dark blue. It is ideal to record the numbers of yeast by examining 10 oil immersion fields. As this is a hypersensitivity and samples are taken from active lesions finding any malassezia is diagnostic.

Allergic Causes of Pruritus

Continue to Steps 3 and 4 if pruritus not resolved with Steps 1 and 2

STEP 3- Rule out an Adverse Reaction to Foods

Food Allergy

A **cutaneous adverse reaction to food (CARF)** or "**food allergy**" is a non-seasonal, pruritic skin disorder associated with a hypersensitivity reaction to a variety of antigenic materials (usually proteins or carbohydrates) in the diet. Food allergy accounts for about 20 % of allergic dogs, but may act as a flare factor for dogs with atopic dermatitis in 20-30 % of cases. Approximatley 50 % of food sensitive pruritic dogs are poorly responsive to corticosteroids. No age, breed, or sex predilection in dogs or cats has been proven. About 70% of the animals have been eating the offending diet or ingredient for over 2 years prior to the development of symptoms. Most dermatologists believe that all pruritic dogs should have the diagnosis of food allergy ruled out before testing for atopic dermatitis, as food allergy is believed to contribute to pruritus in a significant number of atopic dogs. In humans food allergens are generally glycoproteins with a molecular weight greater than 10,000 kD and are usually proteins. The nature of food allergens in dogs is largely unknown. The most common food allergens in dogs are beef, dairy products and wheat, with lesser number of reactions reported to lamb, chicken, egg, corn, rice and gluten.[31] Dogs of any age can be affected, including dogs under 6 months old

or over 7 years old, and is a more common allergy than atopic dermatitis in this population. Predisposed breeds include: Labrador and golden retrievers, cocker spaniels, German shepherd dogs, Chinese shar peis, and poodles.

The clinical signs are extremely variable but include non-seasonal moderate to severe pruritus that may be poorly responsive to glucocorticoids. Papules, pustules, wheals, erythema, excoriations, scales, and crusts are often seen secondary to pruritus and resulting infection. Dogs can present with dermatologic disease that resembles atopic dermatitis (face, ears, axilla, paws- "food-induced atopic dermatitis"), flea allergy dermatitis, recurrent pruritic or non-pruritic pyoderma, urticaria, recurrent otitis externa with or without other signs, chronic anal pruritus: "Ears and Rears". With careful questioning concurrent gastrointestinal signs are typically found.

A diagnosis of food allergy is made using the history and physical examination and evaluating the animal's response to a hypoallergenic elimination diet trial. Serologic testing for foods is not useful and is best avoided. The diet should be composed of food substances to which the dog has not been commonly exposed. Simply switching to another brand or form of commercial dog food is not a valid test as many of the so-called "hypoallergenic" over-the-counter pet store diets have been found to be contaminated with other proteins during the manufacturing process.[32] Dogs are preferably fed a novel home-cooked diet (for example ostrich, emu, rabbit, alligator, kangaroo, or kidney beans combined with sweet potatoes, yams, quinoa, oats or barley) for a minimum of 30 days and preferably for 8-12 weeks.[33] Sources for these meats include: mypetgrocer.com, exoticmeats.com, blackwing.com, www.dinomeat.com, www.redoakfarm.com, www.heartlandemu.com. Go to balanceit.com for easy recipes with supplements to make diet balanced or raynenutrition.com for prepared limited ingredient diets and treats.

If the owner is unable or unwilling to prepare a home-cooked elimination diet perform the diet trial with s a novel protein and carbohydrate or hydrolysed protein commercial diet.

The owners need to be reminded not to give the pet any treats, rawhides, pig's ears, flavored pill pockets, flavored toothpastes or chewable medications such as heartworm preventatives, antibiotics (gelatin capsules included) and/or NSAID's during the diet trial.

While many food allergic dogs will show significant improvement in clinical signs after 4-6 weeks on the diet, some dogs will need 7-12 weeks of the diet to reach maximum improvement. If the animal's symptoms and disease improve while being fed the test diet, a challenge with the original diet should be performed to confirm the diagnosis. Symptoms of pruritus usually return within 48 hours to rarely after 14 days of feeding the offending diet.

STEP 4: It must be Atopic Dermatitis Atopic Dermatitis

Atopic dermatitis (AD) is a common genetically-based pruritic dermatosis of dogs that is believed to be related to the development of allergen-specific IgE antibodies. According to various investigators, the incidence has been reported to vary from 10 to15% of the canine population. Most dogs will first show evidence of the disease between 1 and 3 years. Clinical

signs may be seasonal or non-seasonal. In many dogs the signs began seasonally and progress to year round.

Atopy is the inherited predisposition to form allergen-specific IgE antibodies. Allergen-specific IgE antibodies bind to tissue mast cells, especially those in the skin. When mast cell-bound IgE reacts with its specific allergen, mast cells degranulate and release pharmacologically active compounds (histamine, serotonin, bradykinin, inflammatory cytokines, etc.). While mast cells and IgE are involved in AD and are important, new research has shown that it is not that simple. More recent research has shown that dogs with AD likely have both an abnormal skin barrier function[38] that allows excessive transcutaneous absorption of allergens. In addition, atopic dogs have an imbalanced cutaneous immune system which favors a T-lymphocyte helper type-2 pro-inflammatory reaction to allergens and may be less efficient at fighting infection.[39] The release of many pro-inflammatory cytokines after allergen exposure is thought to be the key to the allergic response seen. A video summarizing the current pathogenesis of the canine AD itch cycle is available at excellenceindermatology.com, and an excellent summary article on our current understanding of the pathophysiologic mechanisms of canine atopic dermatitis has recently been published.[40]

Atopic-like Dermatitis (ALD) is a newly described variant of atopic dermatitis similar to intrinsic AD in humans in which affected dogs show all of the clinical signs of AD without evidence of hypersensitivity to environmental allergens.[41] Affected dogs have negative intradermal skin test results, low to negative levels of allergen-specific serum IgE, and no response to home-cooked and commercially prepared hypoallergenic diets.

Atopic dermatitis can occur in any breed of dog but there is an increased risk reported in Golden and Labrador retrievers, Pit Bull terriers, German shepherd dogs, English bulldogs, boxers, pugs, Irish setters, dalmatians, West Highland white terriers, Scottish terriers, wirehair fox terriers, Welsh terriers, Boston terriers, cairn terriers, Lhasa apsos, shih tzus, and miniature schnauzers. Some authors report a slightly increased incidence in females. A recent study supported the genetic predisposition of atopic dermatitis in Labrador and Golden retrievers (breeding 2 atopic parents resulted in 65% of the offspring being atopic, breeding 1 atopic parent-21-57 % affected, breeding 2 non- atopics- 11 % affected).[36]

In dogs with AD, pruritus is the overwhelming owner complaint. Pruritus and the resultant secondary lesions of self-trauma usually involve the face, particularly the periocular and perioral areas, inner surface of the pinnae, ear canals, ventral neck, paws- especially the caudal metacarpal and tarsal regions and interdigital skin; the flexural and medial aspects of the legs, the axillae, groin, ventral abdomen, perineum, ventral tail area or some combination of these. There are generalized cutaneous signs in about 40% of the cases. Otitis externa is present in about 50% of the cases. Bacterial pyoderma (i.e. folliculitis, furunculosis, acute moist dermatitis) is present in approximately 33% of the cases.

Atopic dermatitis is one of the many possible underlying causes of recurrent pyoderma. Many dogs with AD will experience secondary overcolonization of their skin with both Staphylococcus pseudintermedius and Malassezia pachydermatis. Reasons for this predisposition to infection

include: staphylococci adhere to and multiply more easily on the skin of atopics[42], dogs with AD are thought to have a defective cutaneous protective lipid barrier function, and the secondary seborrheic skin disease, hyperhidrosis and self-trauma seen with AD create a micro-environment more conducive to bacterial and yeast overcolonization.

The diagnosis of AD in dogs is made by the history, physical examination, and by ruling out ALL other causes of pruritic dermatoses. Questions to be addressed in the history include the signalment; age of onset of the pruritus; seasonality, distribution of the pruritus; and known responsiveness of the disease to corticosteroid therapy. Routine labwork is normal in most dogs with AD.

Successful management of AD requires a multi-modal approach for success- be proactive: correcting underlying immune dysregulation and improve barrier function, individual maintenance plan of medications for gradual continued improvement and long term control. Client education key: set realistic expectations for control not cure! Cases need to be managed aggressively and proactively early in the course of disease to prevent irreversible skin barrier function abnormalities and further immune dysregulation..

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CANINE DERMATOLOGY HISTORY FORM

Your Name _____ Your Dog's Name _____
Dog's Age _____ Breed _____ Sex _____
Primary concerns about your dog's skin: _____
_____
When was this first noticed? _____
Onset rapid or gradual? _____
Does your dog itch? Yes No When? Constant Sporadic Night only
Rate your dog's itching on a scale of 1-10 (10-constant severe itching all day and night, 0-no itching) _____
What time of year most itchy? Spring Summer Autumn Winter Year around
What part(s) of your dog most itchy? _____
Where does your dog spend time? _____ % indoor _____ % outdoor
What other pets live in your household? Do any have skin problems or itching? _____
_____
Do any people in the house have skin problems or itching?_____
What is the name of your dog's food? _____
What treats or table food does your dog eat? _____
_____
What flea control do you use and how often? Year round? _____
Do all the pets receive the same flea control at the same intervals? _____
How often to you bathe your dog? _____
How often does your dog swim? _____
What medications is your dog taking at this time? _____
_____
_____
What previously prescribed medications have been of benefit? _____ _____
_____
What other health problems does your dog have? _____

Please share any additional information that you think is important:
_____
_____
_____

BE SURE TO BRING THE PREVIOUS MEDICATIONS, PILLS, OINTEMENTS, EAR CLEANERS, SHAMPOOS, EVEN IF EMPTY TO THE CONSULTATION. BRING FOOD AND TREAT INGREDIENT LABELS.
DO NOT BATHE YOUR DOG WITHIN 5 DAYS, DO NOT CLEAN OR TREAT EARS WITHIN 2 DAYS OF YOUR DOG'S APPOINTMENT.