

Pruritus

Peter J. Ihrke

Pruritus can be defined as the sensation that elicits the desire to scratch, chew, or initiate other self-trauma. Pruritus is assumed to occur in animals that have self-trauma or erythema, excoriations, alopecia, lichenification, or hyperpigmentation that results from inflammation and self-mutilation. Licking, chewing, rubbing, hair removal, irritability, and even personality change (lack of tolerance, aggressive behavior) can result from pruritus; it is the most common clinical manifestation of disease that causes owners to bring their pets in for treatment. Because skin changes from self-trauma of any cause are similar clinically, pruritic skin diseases often are diagnostically challenging and frustrating.

PATHOPHYSIOLOGY

The skin functions as an “external nervous system,” providing continuous sensory input to the central nervous system (CNS) through a finely arborized 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. This may be true of domestic animals as well because the face, distal extremities, and groin are common sites of self-trauma in many species. The sensations of pruritus and pain are carried centrally by nonmyelinated slow conducting C fibers and, to a lesser extent, along myelinated A delta fibers. Myelinated ganglion cell axons carry the message of itch from free nerve endings to neurons located in the posterior horn of the spinal cord. Axons of second-order neurons transmit the message across the midline to the lateral spinothalamic tract and upward to the thalamus. Thalamic neurons then carry the signal to the postcentral gyrus of the cerebral cortex, where the message is interpreted as the sensation of pruritus.

Pruritus commonly stimulates self-trauma. The mechanisms by which self-trauma relieves itching are unclear. Self-trauma may disturb the amplified, reverberating spinal pathways that perpetuate the sensation of itch. More severe self-trauma probably substitutes pain for pruritus. Severe self-trauma characterized by deep excoriations is more common in cats than in dogs.

Diffusible chemical mediators induce the sensation of itch. Implicated endogenous mediators include histamine, peptides (endopeptidases, bradykinin, substance P, vasoactive intestinal peptide, neurotensin, secretin, enkephalins, endorphins), proteases (trypsin, chymotrypsin, mast cell chymase, fibrinolysin, kallikrein, cathepsins, plasmin), prostaglandins, leukotrienes (especially LTB₄), monohydroxy fatty acids, and opioid peptides. Proteolytic enzymes are thought to be the most important mediators of pruritus in dogs, cats, and humans. Leukotrienes also play an important role. The hypothesized magnitude of the role of histamine has diminished as the importance of other mediators is recognized. Bacterial and fungal endopeptidases also can initiate pruritus. Chemical mediators present in arthropod saliva, venom, body fluids, and on

poisonous hairs or spines include proteolytic enzymes, histamine, cantharidin, apamin, mellitin, histidine decarboxylase, kinins, serotonin, endopeptidases, and proteinases.

A “gate control theory” has been hypothesized to explain how the CNS can amplify or reduce the sensation of pruritus. Stress or anxiety may amplify pruritus in humans by releasing opioid peptides. Boredom or other cutaneous sensations such as pain, heat, cold, or touch also can alter the perception of pruritus. Factors such as increased skin temperature, diminished skin hydration, and low humidity can heighten the sensitivity of the skin to pruritic stimuli.

The concepts of *threshold phenomenon* and *summation of effect* are paramount in understanding and managing pruritus. A certain pruritic load may be tolerated without initiating clinical signs, but a small increase in that load can provoke clinical signs. The itch threshold often is reduced at night in humans and animals when other sensory inputs are diminished. *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, physical 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 predilections may offer more direct clues to diagnosis than physical examination. Canine and feline scabies and cheyletiellosis may have increased in frequency during the past 5 years because of the popularity of newer, more narrow-spectrum flea control products that do not kill acarids. The effect of newer flea control products on the incidence of pruritic skin diseases is controversial. Although the frequency of flea allergy dermatitis may have declined in regions where newer products are used, flea allergy dermatitis is still common globally. Effective flea control also may have unmasked formerly undiagnosed cases of canine atopic dermatitis where the pruritus was assumed to be due only to flea allergy dermatitis rather than combined flea allergy and atopic dermatitis. The importance of pyoderma and *Malassezia* dermatitis as frequent causes of pruritus cannot be overemphasized.

Signalment Age

Age provides critical information for prioritizing differential diagnoses. Some skin diseases occur more commonly in young animals, whereas other dermatoses are seen more frequently in middle-aged or older animals. As examples, scabies and demodicosis are pruritic skin diseases seen more commonly in young dogs. Similarly, atopic dermatitis, food allergy, and pyoderma occur more commonly in adult animals.

Breed

Breed predilections for skin diseases are becoming increasingly available (see appendices). Further, some skin diseases are breed specific. As examples, golden retrievers, Dalmatians, and many small terrier breeds are at increased risk for the development of atopic dermatitis. The West Highland white terrier is at increased risk for secondary *Malassezia* dermatitis and the Chinese Shar Pei seems predisposed to atopic dermatitis, food allergy, pyoderma, and demodicosis.

Sex

Sex predilections are not common in pruritic skin diseases. However, pruritus may be seen with Sertoli cell tumors, male-feminizing syndromes, and canine female hyperestrogenism.

Historical Findings**General History**

General history should be sought referable to diet, environment, use, home skin care, recent exposures, other household pets, and the presence or absence of pruritus in other animals or people in the environment. These data are helpful in prioritizing differential diagnoses.

Diet. Food allergy or intolerance can cause pruritus in both dogs and cats. However, adverse reactions to food frequently coexist with other allergic skin diseases such as atopic dermatitis and flea allergy dermatitis. In addition, lipid-deficient diets may exacerbate cornification abnormalities (seborrhea).

Environment and exposure. The likelihood of contagious pruritic ectoparasitic skin diseases is affected by environmental exposure. Flea allergy, canine and feline scabies, and less common ectoparasitic skin diseases are all seen more frequently in animals permitted to roam free. Feline scabies is endemic to certain geographic urban areas. Recent exposures such as acquisition of a new pet or sheltering a stray animal increase the likelihood of contagious disease. Grooming establishments, kennels, and veterinary practices offer additional opportunities for contagion.

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 indoor or outdoor cat is often the source of flea acquisition 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 canine or feline scabies mites 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 (predictability), 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, if the disease has generalized before veterinary care is sought. For example, canine 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 atopic

dermatitis, food allergy, pyoderma, cornification abnormalities, and *Malassezia* dermatitis.

Intensity. Most animals do not exhibit pruritus in examination rooms. Canine and feline scabies, canine flea allergy dermatitis, and feline food allergy are 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 or pattern (predictability). Atopic dermatitis and flea allergy dermatitis are seasonal in many regions of the world. *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. 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 atopic dermatitis 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.

Physical Findings

A complete physical examination is extremely important when evaluating any animal with skin disease. Skin disease may be seen secondary to internal medical disorders (see Chapter 8). Proper lighting is of paramount importance. The clinician should observe the animal for general demeanor and signs of pruritus while taking the history. Examination of the skin, mucocutaneous 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 haircoat. In the dog, worn incisors (buccal surface) and canine teeth (mesial surface) most frequently indicate chronic flea allergy dermatitis.

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 (see Chapter 9). 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 atopic dermatitis 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 (Tables 10-1 and 10-2).

Diagnostic Plan

Diagnostic plans should be formulated based on prioritization of differential diagnoses using signalment, history, and physical findings. Diagnostic procedures are selected based on the most likely differential diagnoses. The algorithm in Figure 10-1 offers an overview of possible diagnostic plans.

Table • 10-1

Pruritic Canine Dermatoses

DISEASE	SITE	LESIONS
Flea allergy dermatitis A, E, F	Bilaterally symmetric, dorsal lumbosacral, caudal thighs, groin, axilla, caudal half of body	Papules, macules, alopecia, erythema, lichenification, hyperpigmentation, excoriations, fibropruritic nodules
Canine scabies A	Ventrum, pinnae margins, face, elbows, partially bilaterally symmetric	Macules, papules, erythema, alopecia, crusts, excoriations
Demodicosis A	Periorbital, commissures of mouth, forelegs, generalized	Alopecia, erythema, crusts, follicular plugging, hyperpigmentation, secondary pyoderma
Pyoderma A	Groin, axilla, ventrum, interdigital webs, generalized, pressure points	Pustules, crusted papules, erythema, alopecia, target lesions, coalescing collarettes, hyperpigmentation
Atopic dermatitis A, F	Face, periorbital, ears, caudal carpi and tarsi, feet (dorsum), otitis externa, axilla, generalized	Erythema, alopecia, excoriations, lack of primary lesions, lichenification, hyperpigmentation
<i>Malassezia</i> dermatitis A, E, F	Ventral neck, groin, skin folds, face, feet, ventrum	Erythema, exudative or dry, alopecia, hyperpigmentation, lichenification
Cornification defects A, E, F	Generalized, ears, preen body	Scales, crusts, alopecia, erythematous plaques
Acral lick dermatitis A	Anterior carpal, metacarpal, radial, metatarsal, tibial regions	Firm, alopecic plaque, central irregular ulcer, hyperpigmented halo
Food allergy B	Face, feet, ears, generalized	Erythema, alopecia, excoriations, lack of primary lesions
Contact dermatitis C	Hairless areas, feet (ventrum), genitals, groin, axilla, generalized	Erythema, exudation, lichenification, hyperpigmentation, papules
Drug eruptions C	Anywhere, localized or generalized, face, ears, scrotum	Pleomorphic, erythema, papules, coalescing target lesions
Endoparasitic migration in puppies C	Face, feet, generalized	Erythema, alopecia, excoriations, lack of primary lesions
Cheyletiellosis B, E	Dorsum of thorax, generalized	Large scales, crusts, alopecia, erythema
Chiggers B, E, F	Ventrum, legs, anywhere	Erythema, scales, crusts, papules, alopecia
Superficial necrolytic dermatitis C	Footpads, face, mucocutaneous junctions, genitals, groin	Adherent crusts, ulcers, excoriations, erythema, fissured pads
Psychogenic pruritus C	Carpi, tarsi, feet (especially forelegs), perianal, generalized	Erythema, alopecia, excoriations, lack of primary lesions
Pediculosis C, E, F	Dorsum, generalized	Scales, crusts, alopecia, papules
Tail-dock neuroma C	Previously docked tail	Erythema, excoriations, alopecia
Rhabditic dermatitis D, E, F	Ventrum, legs, groin	Erythema, papules, alopecia, crusts, scales

A, Common; B, less common; C, uncommon; D, rare or controversial; E, regional; F, seasonal.

Skin Scrapings

Multiple skin scrapings should be performed on all pruritic dogs and cats. Affected areas should be gently clipped, and then a no. 10 scalpel blade or spatula, dipped in mineral oil, should be scraped perpendicular to the skin surface in the direction of hair growth. The acquired debris should then be dispersed on a slide, a cover slip applied, and the specimen examined microscopically using low light.

Demodectic 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 pachydermatis*.

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* organisms, and inflammatory cells. Clear tape preparations

Table • 10-2

Pruritic Feline Dermatoses

DISEASE	SITE	LESIONS
Flea allergy dermatitis A, E, F	Neck, dorsum, lumbosacral, caudal and medial thighs, groin, ears	“Miliary dermatitis,” erythema, alopecia, eosinophilic plaques
Eosinophilic plaque A, E, F	Ventral abdomen, medial thighs, anywhere	Raised, ulcerated, erythematous alopecic plaques, secondary to allergy (primarily flea allergy dermatitis)
Otodectic acariasis A	Ears, head, neck, rarely generalized	Otitis externa, excoriations, “miliary dermatitis”
Food allergy A	Head, neck, ears, generalized	Erythema, excoriations, alopecia, lack of primary lesions, “miliary dermatitis”
Self-induced pruritic hair loss (atopic dermatitis, food allergy, flea allergy) B	Bilaterally symmetric, caudal and lateral thighs, ventral abdomen, perineum	Alopecia, hair stubble, erythema, papules, underlying skin may be normal
Self-induced psychogenic hair loss B	Bilaterally symmetric, stripe(s) on dorsal thorax, caudal and lateral thighs, ventral abdomen, perineum, forelegs	Alopecia, hair stubble, normal underlying skin
Cheyletiellosis B, E	Dorsum of thorax, generalized	Large scales, crusts, seborrhea, “miliary dermatitis”
Demodicosis B, E	Trunk, ventral, generalized	Alopecia, scaling
Mosquito-bite hypersensitivity C, E, F	Bilaterally symmetric, dorsal muzzle, planum nasale, periorbital, pinnae, paw pad margins	Papules, crusts, alopecia, erosion, exudation, fistulation
Pediculosis C, E, F	Dorsum, generalized	Scales, crusts, alopecia
Feline scabies C, E	Head, ears, neck, generalized, partially bilaterally symmetric	Erythema, papules, crusts, excoriations, alopecia
Pruritic dermatophytosis C	Head, neck, ears, generalized	Erythema, alopecia, hair stubble, “miliary dermatitis,” hyperpigmentation
Atopic dermatitis B, F	Head, neck, ears, generalized	“Miliary dermatitis,” erythema, excoriations, alopecia
Drug eruptions C	Anywhere, localized or generalized, pinnae, face	Pleomorphic, erythema, papules, coalescing target lesions
Pemphigus foliaceus D, E?, F?	Bilaterally symmetric, face, planum nasale, ears, interdigital webs, nipples, generalized	Crusts, vesicopustules, alopecia

A, Common; B, less common; C, uncommon; D, rare or controversial; E, regional; F, seasonal.

may demonstrate *Cheyletiella* mites, and stained tape preparations may demonstrate bacteria or *M. pachydermatis*.

Fecal Examination

Fecal examination may document endoparasite infestation in pruritic puppies and may reveal mites or other ectoparasites in any animal.

Skin Biopsy

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

Fungal Culture

Most dogs and cats with dermatophytosis are not pruritic. However, fungal culture may be warranted because many cases of dermatophytosis are not visually distinctive.

Elimination Diets

Animals suspected of having food allergy or food intolerance as a cause of pruritus should be fed a home-cooked diet consisting of one protein and one carbohydrate source for 8 to 12 weeks. Alternatively, newer “limited antigen” or “hydrolyzed” diets are available. Commercial restricted diets are recommended for long-term maintenance. Nothing is specifically “hypoallergenic” about any food source. Foods are selected based simply on lack of previous exposure. Whitefish, rabbit,

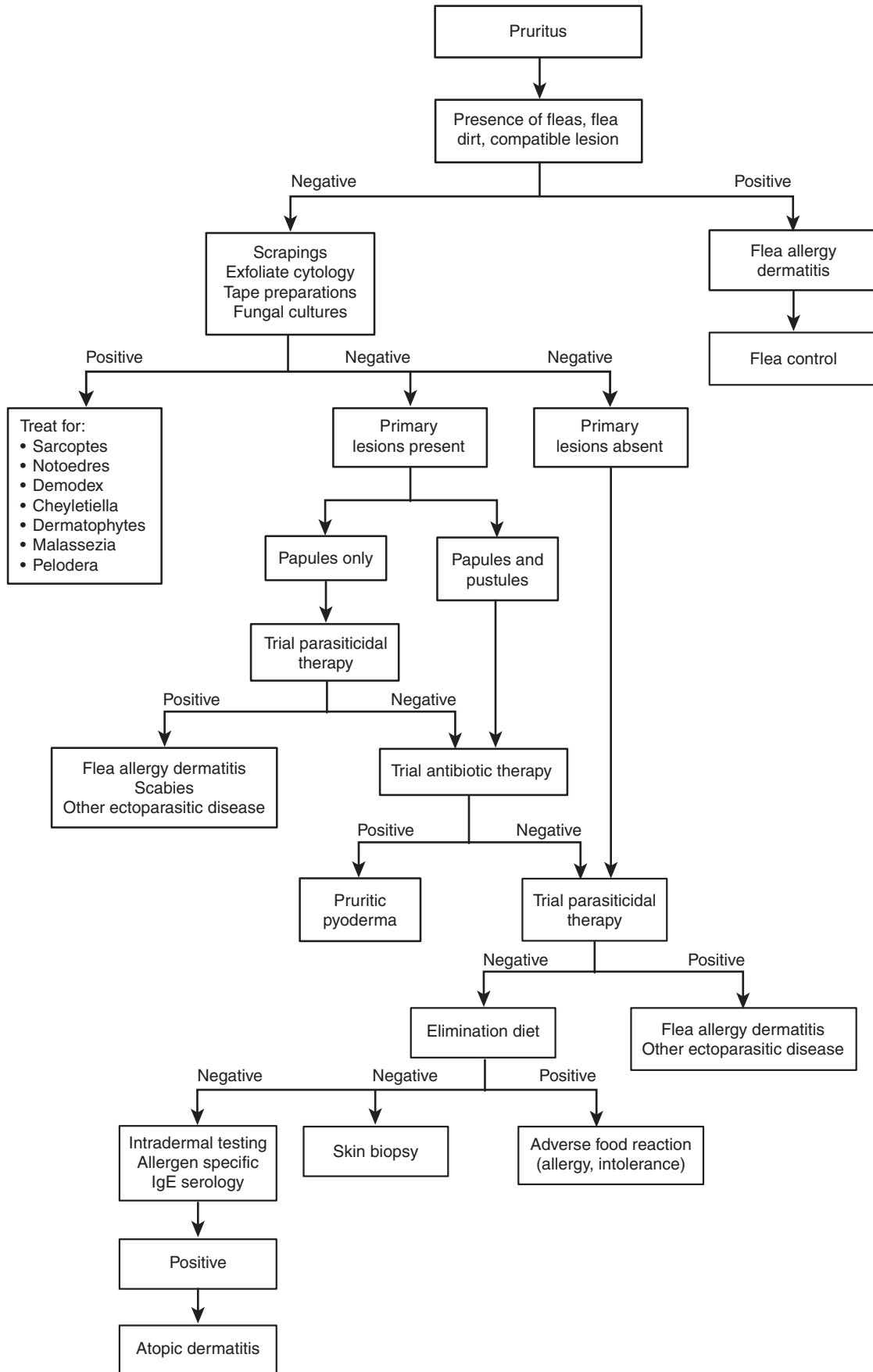


Figure 10-1 Algorithm for diagnosis and management of pruritus.

venison, pork, cottage cheese, or tofu mixed with either potatoes or other novel carbohydrate sources are commonly used in the dog. Mutton, lamb, and chicken are no longer as useful because they are frequently used in commercial dry dog foods. Cats may be fed rabbit, venison, pork, or mutton without an added carbohydrate source, or they may be fed commercial restricted or hydrolyzed diets.

Intradermal Testing

Substantial training is required to select appropriate candidates suspected of having atopic dermatitis, to choose antigens, to develop and maintain a reproducible technique, and to interpret results. Consequently, skin testing is most effective when practiced by dermatologists or other clinicians with a strong interest in dermatology.

Allergen-Specific IgE Serology (enzyme-linked immunosorbent assay [ELISA] or radioallergosorbent test [RAST])

In vitro testing for atopic dermatitis 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.

Response to Trial Therapy

Trial therapy with parasitocidal agents is used routinely in suspected cases of scabies or flea allergy dermatitis. Flea allergy dermatitis remains the most common cause of canine and

feline pruritus despite effective modern products. Because the lesions seen with canine superficial pyoderma may be pleomorphic, trial use of antibiotics may be indicated in undiagnosed pruritic crusted papular dermatoses. Although response to corticosteroids is suggestive of underlying allergic disease, superficial pyoderma will frequently respond partially to corticosteroid therapy.

Cost Containment

Skin scrapings, fungal culture, exfoliate cytology, trial therapy for ectoparasites, and, surprisingly, skin biopsy, are the most cost-effective diagnostic procedures for the pruritic animal.

GOALS OF THERAPY

Successful long-term management of a pruritic dog or cat usually requires definitive diagnosis. Repetitive parasitocidal therapy on a weekly basis for 3 or 4 weeks will rule out most contagious ectoparasitic diseases such as canine or feline scabies. However, management of flea allergy dermatitis is a lifelong endeavor encompassing control of fleas on the affected animal and all in-contact dogs and cats, as well as environmental control. Atopic dermatitis responds best to allergen-specific immunotherapy. Secondary pyoderma and *Malassezia* dermatitis are common sequelae to most pruritic skin diseases and must be assessed for and managed long-term. If corticosteroids are used adjunctively for the long-term management of allergic skin disease, short-acting oral corticosteroids such as prednisone, prednisolone, or methylprednisolone are recommended on an alternate-day basis. Corticosteroids are contraindicated in the treatment of canine demodicosis and pyoderma. Many pruritic animals require long-term adjunctive topical management with shampoos and emollients or antipruritic rinses.

CHAPTER • 11

Cutaneous and Subcutaneous Lumps, Bumps, and Masses

Didier N. Carlotti

Cutaneous and subcutaneous lumps, bumps, and masses include hematomas, abscesses, urticaria and angioedema, neoplasms and pseudoneoplasms.

CLINICAL AND HISTOPATHOLOGIC DEFINITIONS

A hematoma is a focal extravasation of blood with purpura (bruising) and pain, whereas an abscess is a localized collection of pus with pain, heat, and sometimes purpura. Urticaria is referred to as a group of wheals (sharply circumscribed, raised, edematous lesions) that appear and disappear rapidly. Angioedema is a large swelling in a distensible region such as the face and limbs.

Clinically, pseudoneoplasms and neoplasms appear as nodules, plaques, and tumors. Ulceration always indicates a severe pathologic process. Pseudoneoplasms include cysts,

nevi, keratoses, granulomas, and pyogranulomas and other lesions. *Pseudoneoplasm* is a better term than *pseudotumor* because the term *tumor* is clinical and should refer to a localized hypertrophy of a tissue or an organ, neoplastic or not. Cysts are epithelial lesions containing grayish keratinous material or serous material, such as apocrine cysts, which appear fluctuant, bluish, and well circumscribed. A hamartoma is a malformation formed by components of a normal organ arranged erroneously. A nevus is a cutaneous hamartoma that may arise from any skin component. Collagenous nevi are single or multiple nodules characterized histopathologically by large areas of collagen hyperplasia. In German shepherds, multiple collagenous nevi may appear, particularly on the limbs, in association with renal adenocarcinomas and uterine leiomyomas (nodular dermatofibrosis syndrome). Organoid (i.e., pilosebaceous) and epidermal nevi are variable in shape and may be linear. Vascular nevi are seen on the