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ISSUES IN DERMATOLOGY

Review of Pemphigus Foliaceus in Dogs and Cats

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Pemphigus foliaceus (PF) is the most common autoimmune skin disease in dogs and cats. It is also the most common variant of pemphigus diseases,[1,2] which are characterized by autoantibodies that target keratinocyte desmosomal proteins, leading to loss of cell-to-cell adhesion (acantholysis). Acantholysis of keratinocytes causes separation and loss of integrity of the epidermal cell layers, resulting in transient pustules and/or blisters that rapidly develop into erosions, crusts, scales, and alopecia on the skin and/or mucous membranes.[1-5] In dogs and cats, PF may occur spontaneously or, more rarely, may be associated with drugs,[6-12] environmental factors,[13] or concurrent thymoma[1] or autoimmune diseases.[3,14]

The stratified epithelia are made up of different layers that each contain keratinocytes and nonkeratinocyte cells (e.g., melanocytes, Langerhans cells, Merkel cells). Keratinocytes and the surrounding cells are held together predominantly by desmosomes, forming a type of cell adhesion with a complex network of many types of proteins. These desmosomal proteins are expressed with varying intensities among different epithelial layers and tissues (i.e., mucosa versus footpads).[15] The anatomic distribution and depth of lesions therefore depend on the targeted desmosomal protein. For instance, desmocollin-1 (DSC-1) is a desmosomal protein that is distributed primarily in the superficial layers of the follicular and interfollicular epidermis (stratum granulosum and stratum spinosum) but not in mucosae. DSC-1 is the major autoantigen in dogs with PF.[15] As such, disruption of DSC-1 by different mechanisms by pathogenic immunoglobulin G (IgG) autoantibodies results in superficial blister formation in the skin but not the mucosae. Conversely, when desmoglein-3 (DSG-3), another type of desmosomal protein expressed at higher intensity in the deeper layers of the epidermis and mucosae, is targeted by these autoantibodies, a deep erosive form of pemphigus develops (pemphigus vulgaris).[16] A recent study confirmed the presence of anti-keratinocyte IgG in cats with PF and different substrate immunoreactivity compared with that in the dog, suggesting the role of a different

SKIN IN THE GAME
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autoantigen target in cats with PF.[17] These studies seem to demonstrate variation in IgG autoantibody targets between dogs and cats and different forms of pemphigus within each species.

SIGNALMENT

For dogs and cats, predisposition to PF does not seem to be associated with the animal's sex; although PF can occur at any age, median age of onset is around 6 years (middle-aged).[2,18] Canine PF can affect dogs of any breed or crossbreed; however, certain breeds (e.g., Akitas and chow chows) are overrepresented,[19] suggesting a genetic predisposition.[3] Other dog breeds in which PF incidence may be higher include Labrador retrievers, cocker spaniels, German shepherds, and English bulldogs.[5] No strong cat breed predisposition to PF has been reported; however, the most represented breeds are domestic shorthair, medium-hair, and longhair cats.[18]

ASSOCIATED DISEASES AND RISK FACTORS

Data purporting possible associations between PF and drugs, environmental factors, or concurrent disease remain scant; most cases of PF are presumed to be idiopathic. However, a possible association between allergic skin diseases (e.g., atopic dermatitis and flea allergy dermatitis) and development of PF has been reported.[3] The high prevalence of allergic dermatitis and long-term drug use for chronically affected animals with allergies, and thus possible drug-related PF, complicates the etiology of PF. Reported drugs associated with PF include antibiotics (e.g., sulfonamides, penicillins, cephalosporins),[6,8] cimetidine in cats,[1] and 3 topical ectoparasitic preparations containing metaflumizone, fipronil, amitraz, S-methoprene, dinotefuran, pyriproxyfen, or permethrin.[9-11] In studies of dogs, clinical lesions resolved spontaneously after withdrawal of the suspected drug for some, but others required immunosuppressive therapy to achieve clinical remission.[8,11] Of note, after the adverse drug reaction probability scale was applied, the scores obtained indicated that drug-related cases of canine PF were possible.[19] Other potential associations include concurrent systemic disease (e.g., cutaneous polyautoimmunity reported for 2 dogs with concurrent PF and generalized discoid lupus erythematous),[14] sunlight exposure,[13] and exposure to drugs in other groups (isolated cases).[12]

CLINICAL PRESENTATION

The primary lesion in patients with PF is the pustule

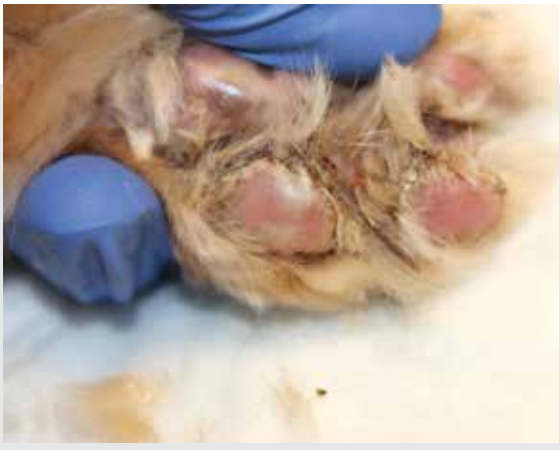

FIGURE 1. Pustules and margin crusting on digital pad of a cat.

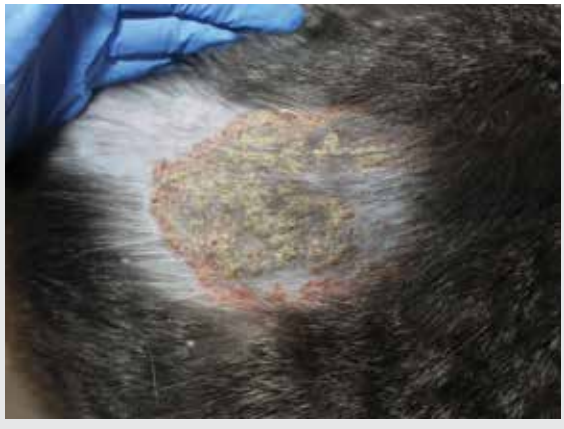

FIGURE 2. Coalescing, thick, yellowish crusts forming a large alopecic lesion with peripheral erythema and erosions on the dorsum of a cat.

(**FIGURE 1**). However, because most PF pustules are superficial, small, and therefore transient, identification of pustules can be a rare and challenging opportunity. Most pustules will instead rapidly evolve into erosions and crusts. Indeed, crusts are the most common clinical presentation of PF in dogs and cats and will often appear thick (multilayered) with a yellowish coloration due to the cyclic nature of PF (**FIGURE 2**).[1-3,5,18] Other lesions include pustules of variable size, erosions, and alopecia. The lesions can coalesce or cluster and organize into different patterns. Pruritus is variable but commonly reported and can be severe in patients of both species.[1-3,5,18] A relatively high number of dogs and cats also exhibit systemic signs of lethargy, pyrexia, hyporexia or anorexia, weight loss, and pain.[1-3,5,18]

Lesions typically first appear on the face and for most patients progress to other body sites such as the trunk and feet/footpads (**FIGURE 3**). Lesions may also become generalized or appear commonly in other parts of the body as well (**TABLE 1**). However, in dogs some PF variants can affect only a few localized areas of the body without appearing anywhere else (e.g., lesions may be restricted to the face or footpads only or to the trunk only).[18,20] A notable distinction between PF in dogs and in cats is the presence of lesions in the ungual

FIGURE 3. (A) Diffuse alopecia with crusting and erosions on the face of a cat. **(B)** Multifocal erythematous patches, crusts, small pustules, and erosions on the face of a French bulldog. **(C)** Severe crusting on footpads with plantar erythema, erosions, and pustules on the left hind foot of the same French bulldog. **(D)** Multifocal crusts with peripheral erythema and alopecia on the trunk of a dog.

TABLE 1 Location and Frequency of Commonly Reported Skin Lesions on Dogs and Cats with Pemphigus Foliaceus[18,20]

LESION DISTRIBUTION	% DOGS (N = 40)[20]	% CATS (N = 49)[18]
Face	82.5	90
Pinnae (convex and concave)	85	92
Dorsum	90	41
Ventrum	60	35
Periareolar (cat)	Not applicable	27
Ungual folds (cat)	Not applicable	47
Feet (dorsal aspect, interdigital area)	55	33
Footpad	27.5	37

folds (47%) and periareolar region (27%) of cats;[18] however, it is uncommon (11%) for cats to display skin lesions affecting only the ungual folds or footpads (**FIGURE 4**).[1] Periareolar lesions lead to high suspicion for feline PF, although lesions are less commonly found in this area than in other areas.[1,18]

DIAGNOSTIC INVESTIGATIONS

In brief, diagnosis of PF is based on compatible clinical presentation, confirmation of superficial pustular acantholysis, and exclusion of relevant differential diagnoses (**FIGURE 5**).[3] Diagnostic investigation of PF begins, as for every disease, with consideration of the patient's signalment (e.g., breed predisposition) and a detailed history. Careful questioning should reveal the patient's therapeutic history (responses to antibiotics, ectoparasiticides, immunosuppressants) and exposure to other drugs or toxins. PF typically affects the face first. The course of the disease might be progressive with either a rapid (within days or weeks) or slow (within months or years) onset of clinical signs, or it may wax and wane with waves of pustule formation.[5,21,22] Clinical suspicion of PF can be elicited by the classic clinical features of superficial pustular dermatitis (pustules, erosions, crusts, alopecia, scales) affecting the face, pinnae, and feet/footpads (including ungual folds and/or periareolar regions in cats) and sparing the mucosae (**TABLE 1**). For patients, the next recommended diagnostic step is cytology to detect acantholytic keratinocytes (**FIGURE 6**). Also helpful is evaluating whether the patient has concurrent pyoderma.

Cytology

The best lesions to sample are pustules. However, when pustules are not evident, the authors use a dermatoscope (a handheld device that emits high-quality light coupled with a magnifying lens), which increases the sensitivity for identifying small or early-forming pustules. If an intact pustule is found, it can be sampled by puncturing the pustule with a needle and lifting the keratinocyte scaffold to expose the pus and gently placing a slide against it.[23] Most clinicians,

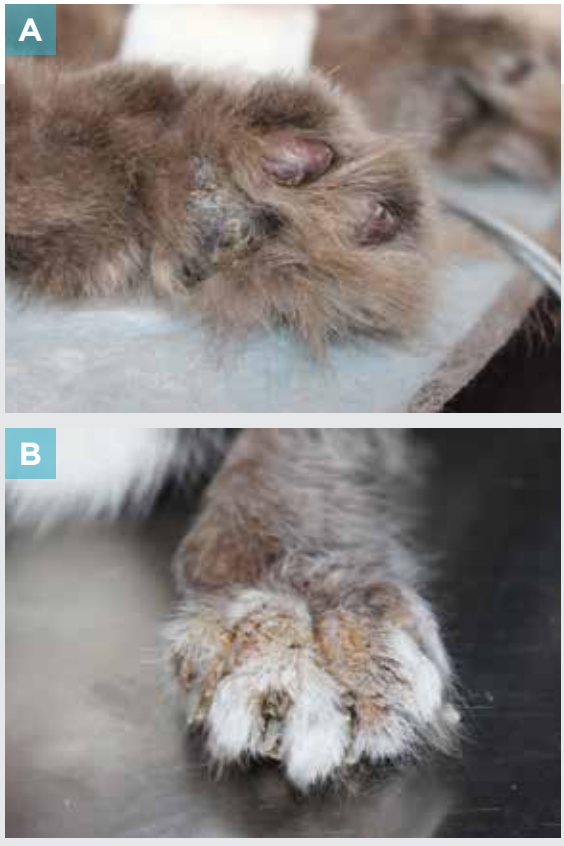

FIGURE 4. (A) Crusting and hyperkeratosis on footpad as the sole clinical presentation in an adult cat with pemphigus foliaceus. **(B)** Severe crusting on ungual folds in a cat.

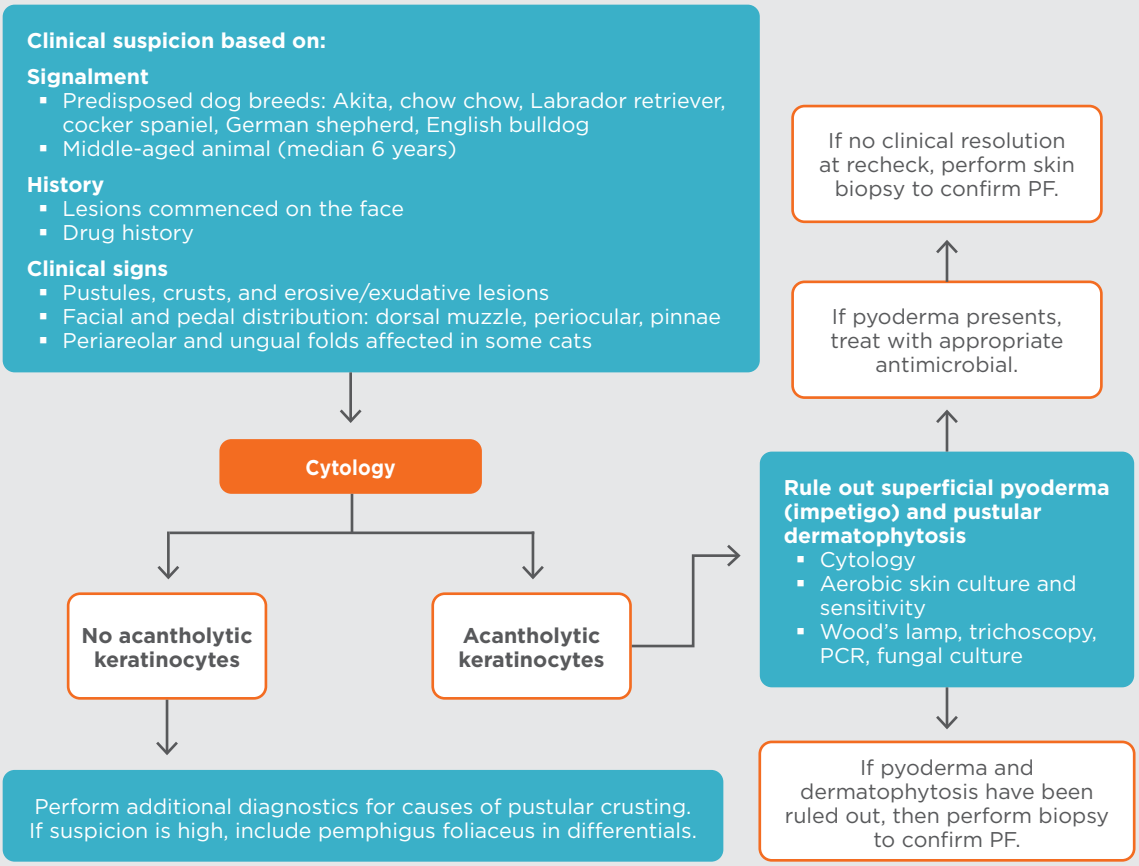

FIGURE 5. Algorithm for diagnosis of pemphigus foliaceus. Note: In *Leishmania*-endemic or in high- or emergent-risk geographic areas, canine leishmaniasis should be ruled out. PCR=polymerase chain reaction, PF=pemphigus foliaceus.

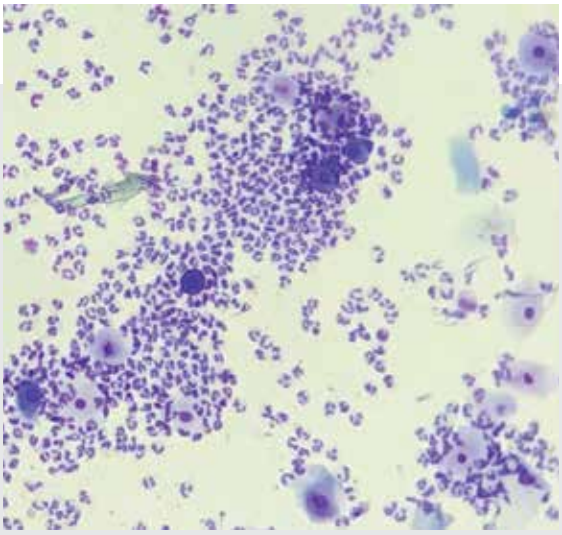

FIGURE 6. Microscopic view of an impression smear made from under a crust from a dog with suspected pemphigus foliaceus. Several acantholytic keratinocytes (round, deep basophilic, large nucleus) are surrounded by numerous nondegenerate neutrophils.

however, will see patients with secondary crust formation; for these patients, sampling can be performed by removing the superficial crust and obtaining an impression smear of the exudative lesion underneath.[23] Once the sample on the slide is dried, it is then fixed and stained using the 3-solution Diff-Quik method. The sample should then be examined in 100× high-powered oil field using immersion oil. Acantholytic keratinocytes have reportedly been captured in cytologic samples from approximately 77% of dogs[2] and 74% of cats.[18] However, presence of acantholytic keratinocytes is not pathognomonic for PF because pyoderma (particularly that caused by some strains of *Staphylococcus pseudintermedius*), dermatophytosis caused by *Trichophyton* species, and canine leishmaniasis can also produce acantholytic keratinocytes.[1-3,24] Impression smears may reveal nondegenerate neutrophils and, to a lesser extent, eosinophils and bacteria (intracellular and extracellular).[1-3]

BOX 1 Tips for Performing a Skin Biopsy for Suspected Pemphigus Foliaceus Cases

- Search thoroughly for pustules to sample. Pustules are the most informative lesions in patients with pemphigus foliaceus. Thoroughly check commonly affected areas (i.e., face, pinnae, footpads). If pustules are not seen, then select thick crusts. In many cases, acantholytic keratinocytes can be seen embedded in crusts or in exudate from ungual folds in cats (**FIGURE 7**).
- Do not scrub the lesions before collecting biopsy samples. Hair can be clipped for better visualization of the lesion, but care should be taken to not rupture intact pustules.
- If during the procedure a crust falls off from the underlying skin, include it with submissions to the pathologist.
- Collect biopsy samples with a punch (6–8 mm whenever possible) or by excising.
- Select multiple sample sites that appear to be the most representative of affected lesions. In most cases, 3 or 4 samples are enough. If acantholysis was visualized on cytologic examination, include that sampled area.

Ancillary Testing

In dogs and cats for which PF is clinically suspected and acantholytic keratinocytes have been identified, superficial pyoderma (impetigo) and pustular dermatophytosis should be ruled out. Useful for ruling out superficial pyoderma is ancillary testing via cytology and/or aerobic bacterial culture with sensitivity testing. Pustular dermatophytosis is rarer than impetigo but still warrants investigation. Ancillary tests to rule out pustular dermatophytosis include Wood's lamp examination, trichoscopy, molecular testing (e.g., polymerase chain reaction), and fungal culture. When PF and concurrent superficial pyoderma are suspected, it should be determined whether the superficial pyoderma is secondary to PF or causal to the clinicopathologic features. Regardless, a systemic antibiotic should be given, either guided by sensitivity testing or empirically chosen if no test is done. If there is partial or no clinical improvement, then superficial pyoderma is not likely to be the final diagnosis. In areas where *Leishmania* are endemic or emerging, the authors also recommend ruling out canine leishmaniasis.

Histopathology

After superficial pyoderma and pustular dermatophytosis have been ruled out, the next diagnostic step is skin biopsy.[3] A representative biopsy sample is critical for an accurate diagnosis of PF (**BOX 1**). Classic histopathologic features of PF are superficial epidermal and follicular (subcorneal or intragranular) pustules with acantholytic keratinocytes in the absence of infectious pathogens (**FIGURE 8**). These pustules are often large and span several hair follicles.[3] Special stains (i.e., Gram, periodic acid–Schiff) can be used to detect bacteria or fungi and thus increase the sensitivity of histopathology. Neutrophils are usually found in large numbers within pustules, and most appear intact (nondegenerate or nontoxic). Eosinophils can also be a prominent cytologic and histopathologic finding.[3,20] Eosinophilic infiltration is

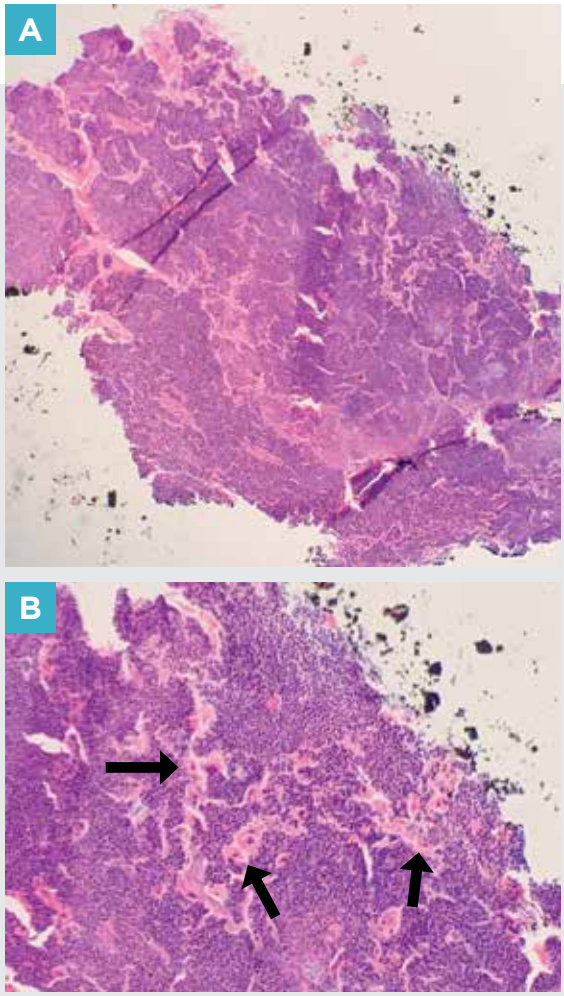

FIGURE 7. (A) Histologic image of submitted purulent exudate from an ungual fold in a cat, using hematoxylin and eosin stain at 40×. **(B)** Detailed photomicrograph at 100× from **(A)** showing acantholytic keratocytes **(black arrows)**.

TABLE 2 Differential Diagnoses for Pemphigus Foliaceus (PF) in Dogs and Cats

DIFFERENTIALS FOR PF WITH ACANTHOLYSIS[1,3]	DIFFERENTIALS FOR PF WITHOUT ACANTHOLYSIS*[21,22]

▪ Superficial impetigo
▪ Pustular dermatophytosis (*Trichophyton* species)
▪ Leishmaniasis
	▪ Autoimmune dermatoses
	  ▪ Systemic lupus erythematosus
	  ▪ Cutaneous lupus erythematosus
	  ▪ Pemphigus erythematosus
	▪ Parasitic and infectious diseases
	  ▪ Demodicosis
	  ▪ Feline scabies
	  ▪ Feline herpesvirus
	  ▪ Feline mosquito hypersensitivity
	  ▪ Leishmaniasis
	▪ Neoplasia
	  ▪ Cutaneous epitheliotropic lymphoma
	▪ Metabolic diseases
	  ▪ Zinc-responsive dermatitis
	▪ Iatrogenic/drug-related causes
	  ▪ Cutaneous drug reaction

*Diseases that are not associated with acantholysis but have a clinical phenotype similar to that of PF.

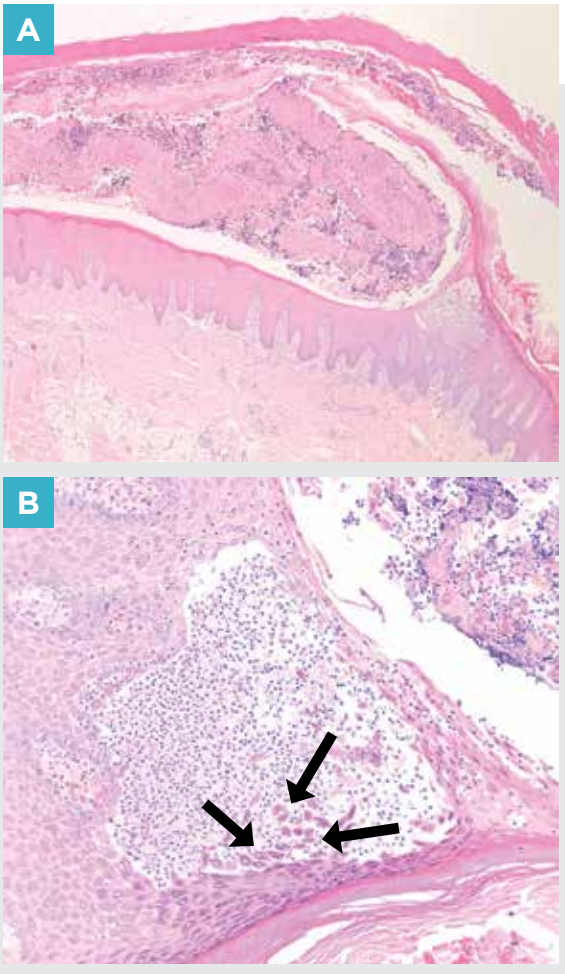

FIGURE 8. (A) Photomicrograph showing a large subcorneal pustule, using hematoxylin and eosin stain at 40×. **(B)** Numerous acantholytic keratinocytes **(black arrows)** with nondegenerate neutrophils in an early pustule, shown at 200×.

reportedly more likely in patients with concurrent systemic disease.[20] In a recent study, histopathologic findings consistent with vasculopathy or vasculitis were more likely to occur in dogs with systemic signs and for which clinical remission took longer to achieve.[25]

DIFFERENTIAL DIAGNOSIS

Differential diagnoses for PF (**TABLE 2**) include conditions that can cause superficial pustular dermatitis with acantholysis. Diseases that can mimic PF clinically and histopathologically are superficial pyoderma

FIGURE 9. Severe facial scaling and crusting with erythema in a dog with leishmaniasis resembling pemphigus foliaceus.

TABLE 3 Treatment Protocols for Pemphigus Foliaceus (PF) in Dogs and Cats

THERAPY	INDUCTION DOSE*	NOTES
TOPICAL THERAPY		
Antimicrobial, keratolytic shampoo	Every other day until crusts and concurrent pyoderma resolve	
Tacrolimus 0.1%	Q12h and taper	
Corticosteroid (e.g., hydrocortisone)	Q12h and taper	
Sun avoidance		
CORTICOSTEROID MONOTHERAPY		
Prednisolone (cats)	2 mg/kg q24h (median)[26]	Complete remission seen in 97% of client-owned cats (n = 37) within 8 weeks.[26] Maintenance dose was 1.2 mg/kg/week.[26] Other studies show variable response rates of 35%–97%.
Prednisone (dogs)	2–6.6 mg/kg q24h[3]	Varied response; general success rate <50%. Others require concomitant immunosuppressive therapy.[3]
Prednisone (dogs)	Pulse therapy at 10 mg/kg q24h for 3 consecutive days, followed by a reduced dose (<2 mg/kg q24h)[27]	61% achieved complete remission with pulse therapy over 6.9 days (median) compared with 15% of dogs receiving a traditional course of prednisone.[27]
Triamcinolone (cats)	0.6–2 mg/kg q24h[28]	100% (15/15) achieved complete remission in 1 study, and remission rate was significantly higher than that for cats receiving prednisone monotherapy (61.5%, 8/13).[28]
Dexamethasone	0.1–0.2 mg/kg q24h[2,18]	
ADJUVANT IMMUNOSUPPRESSIVE DRUGS		
Azathioprine (dogs only)	1.5–2.6 mg/kg q24h (with or without prednisone)	Has slow onset of action[4]
Cyclophosphamide	25 mg/m q24h to q48h[2,3]	Main adverse effect is hemorrhagic chemical cystitis
Chlorambucil	0.1–0.2 mg/kg q24h to q48h[3,18]	
Cyclosporine	5–10 mg/kg q24h (with prednisolone)[1,3]	Can be used as a steroid-sparing agent and be combined with other immunosuppressants
Mycophenolate mofetil (dog)	10–20 mg/kg q12h	Most dogs require concurrent glucocorticoid therapy but at tapering doses[29]
Tetracycline and niacinamide	250–500 mg of each q8h[3]	Has slow onset of action[4]
High-dose human intravenous immunoglobulins	One dog received multiple doses (0.5 mg/kg) infused IV over several hours[30]	Evidence for use for PF is very limited
Polysulfated glycosaminoglycan	A recent report described use as adjunctive therapy to control the cutaneous clinical signs of PF in 3 dogs[31]	Evidence for use for PF is very limited
NOVEL THERAPIES		
Oral oclacitinib (cats)	1 mg/kg q24h[32]	According to 1 case report, reduction in pruritus and severity of lesions was <50%.[32]
Bruton's tyrosine kinase inhibitor (PRN-473) (dogs)		All 9 dogs with PF in a pilot study showed reduced lesions after treatment with PRN-473 monotherapy.[33,34]

**Maintenance doses may include further tapering.*

(impetigo), pustular dermatophytosis (*Trichophyton* species infection), and leishmaniasis (**FIGURE 9**).[1-3,24] Patients with PF can have secondary pyoderma, but treating the infection will not eliminate further pustule formation. In addition, superficial pyoderma does not typically spread from the face and involve the footpads.[22] However, superficial pyoderma as a differential diagnosis is more relevant for patients in which the main region affected is the trunk. Another clinical clue is the bilateral and symmetrical distribution of lesions that is commonly seen in patients with PF (**FIGURE 10**) but far less commonly seen in patients with clinical PF–mimicking non–autoimmune-driven conditions.

TREATMENT

The standard of care for patients with PF is immunosuppression.[3,4] Several immunosuppressive drugs can be used for dogs and cats either as monotherapy or combined (**TABLE 3**).

Glucocorticoids

The overall treatment strategy of PF is to initially induce remission and control the disease as soon as possible. For dogs, cats, and people, the most common initial treatment is glucocorticoids, either alone or combined with a second immunosuppressive drug (adjuvant) until remission is achieved,[4,35] and the preferred route of administration is oral. The main advantages of glucocorticoids are their fast onset of action and broad effects.[4] For long-term management, glucocorticoids should be tapered with the goal of maintaining complete remission while preventing relapses and avoiding potential adverse effects.[4,5] To taper glucocorticoids, the initial dose is gradually reduced to a minimum dose still able to control disease; the final goal is long-term disease management with treatment on alternate or fewer days, using less than 1 mg/kg q24h.[4,5] Prednisone and prednisolone (for cats) are typically dosed at 2 to 4 mg/kg q24h, although an induction dose of 2 mg/kg q24h may achieve complete remission in cats[26] and a lower dose (approximately 1.5 mg/kg q24h) has been reported to successfully induce remission in dogs.[25] The authors currently tend to use 2 mg/kg q24h as the induction dose in dogs and typically 2 to 3 mg/kg q24h in cats. The percentage of dogs achieving complete remission with prednisone monotherapy varies among studies. In a recent study comparing pulse treatment versus traditional treatment with glucocorticoid monotherapy, 61% of dogs that received pulse treatment achieved clinical remission after 3 months compared with 15% that received traditional treatment; however, the numbers of dogs with severe adverse effects and the times to remission were similar.[27] In a large canine PF case series, remission was achieved by 38% of dogs that received prednisolone alone compared with 42% that received a combination of prednisolone and azathioprine.[2] However, in a different pilot study, 4 of 5 dogs with PF achieved complete remission after receiving a combination of prednisolone and cyclosporine.[36] For cats, it has been reported that

FIGURE 10. (A) Skin lesions on the face compatible with pemphigus foliaceus in a Siberian husky and **(B)** bilateral symmetric distribution of lesions on an Australian shepherd.

Clinical remission is likely for most patients (50% of dogs and 90% of cats), and the time to remission is generally 4 to 7 weeks.[1-3,18,20]

glucocorticoid pulse therapy did not offer a superior clinical benefit compared with standard glucocorticoid treatments.[1] Other glucocorticoids include triamcinolone, dexamethasone, and methylprednisolone.[4] For patients with localized cases, topical glucocorticoids can be combined with systemic treatments as adjuvants to spare the effects of systemics.

Nonsteroidal Immunosuppressants

Adjuvant use of nonsteroidal immunosuppressive drugs is common in any of the following circumstances:

- Little to no response to glucocorticoid monotherapy is seen during the first weeks of treatment.
- An acceptable and safe long-term protocol with glucocorticoids is not possible due to relapsing.
- The steroid-sparing effects of these drugs are desirable when trying to avoid the adverse effects of steroids or when glucocorticoids are contraindicated.

Novel Therapies

Novel therapies that have been investigated include the off-label use of oclacitinib (1 mg/kg PO q12h) in a 13-year-old domestic shorthair cat with PF and concurrent cardiac and renal disease. The PF had not previously responded to oral and injectable corticosteroids, but after a 7-day course of oclacitinib, pruritus and severity of the cat's lesions decreased by more than 50%.[32] In another pilot study of 4 dogs with PF that were given only oclacitinib at 1 mg/kg q12h (approved labeled dose range is 0.4 to 0.6 mg/kg q24h or q12h), 2 showed improvement with a clinical score decrease of 65% after 1 month; however, 2 were excluded from the study due to neoplasia.[37] One of the benefits of oclacitinib is its higher margin of safety compared with that of corticosteroids. Another open-trial pilot study concluded that monotherapy with a Bruton's tyrosine kinase inhibitor may have beneficial effects for some dogs with PF,[33] and this finding led to a second open trial.[34] However, further studies with a larger number of dogs of various breeds within a controlled protocol are warranted.[32]

Sun Avoidance

Because of suggestions that sun exposure can worsen PF,[13] the authors usually recommend sun avoidance during peak hours whenever possible and use of sunscreens for facial lesions.

PROGNOSIS

The prognosis for patients with PF remains fair (dogs) to good (cats) depending on comorbidities, response to therapy, and adverse response to treatment. Clinical remission is likely for most patients (50% of dogs and 90% of cats), and the time to remission is generally 4 to 7 weeks.[1-3,18,20] Although it is possible that PF for some dogs and cats will remain in remission long after therapy is discontinued, rates of clinical relapse remain high (61% to 73% of cats);[1-4,18,38] relapse has been associated with tapering or discontinuing medications.[18,38] Larger, randomized controlled studies are needed to determine the benefits of combined versus single-agent treatment regimens in terms of prognosis and outcome.[1-4]

Adverse effects to immunosuppressive therapies have been experienced by about half of all dogs and cats with PF and include iatrogenic hyperadrenocorticism, skin fragility syndrome, hepatotoxicity, secondary infections, gastrointestinal upset, and diabetes mellitus.[18,20]

CLIENT COMMUNICATION

Approximately 10% to 18% of dogs and cats are euthanized after PF is diagnosed.[2,20,38] Reasons for euthanasia include patients' lack of response to treatments, adverse effects to medications, poor quality of life due to PF, or clients' financial limitations of continuing management.[38] It is therefore wise to inform clients that although treatment for PF is generally successful, recurrence is common and many patients require lifelong therapy. A 2019 survey study found that 95% of cat owners complained about the time investment required for the care of feline PF patients and that more than 70% experienced negative effects on their financial stability and emotional wellbeing.[18]

SUMMARY

In dogs and cats, PF may occur spontaneously or, more

rarely, may be associated with drugs, environmental factors, or concurrent autoimmune disease.[1-3] Lesions commonly include pustules, erosions, crusts, alopecia, and scales distributed along the trunk, pinnae, dorsal muzzle, foot pads, periocular area, and nasal planum.[1-3] Diagnostic suspicion is based on medical history, clinical findings, and acantholytic keratinocytes demonstrated on cytologic examination of skin lesions. Histopathology and ruling out other acantholytic pustular diseases by different means will further support suspicions. Definitive diagnosis may follow advanced immunologic testing such as direct and indirect immunofluorescence.[3] The treatment of choice is use of immunosuppressive drugs. Reported immunosuppressives used to treat PF in dogs and cats include glucocorticoids (oral, topical), azathioprine, cyclosporine, mycophenolate mofetil, chlorambucil, topical glucocorticoids, or calcineurin inhibitors, and less commonly a combination of tetracycline and niacinamide.[4] The prognosis for dogs with PF is fair and for cats is good; however, recurrence is common. TVP

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