



International Journal of Veterinary Sciences and Animal Husbandry



ISSN: 2456-2912

NAAS Rating (2025): 4.61

VET 2026; 11(3): 257-259

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www.veterinarypaper.com

Received: 02-12-2025

Accepted: 07-01-2026

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Pemphigus in dogs: An update

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DOI: <https://www.doi.org/10.22271/veterinary.2026.v11.i3e.3135>

Abstract

Pemphigus in dogs is an autoimmune vesicobullous to pustular dermatosis characterized by acantholysis and intraepidermal blister formation, most commonly manifesting as pemphigus foliaceus in middle-aged animals. Autoantibodies directed against desmosomal proteins (primarily desmoglein-1, desmoglein-3, and desmocollin-1) induce complement activation, neutrophilic inflammation, and progressive crusting, with clinical forms ranging from superficial (foliaceus, erythematosus, vegetans) to deep, mucosa-involving variants (vulgaris, paraneoplastic). Diagnosis is based on distribution and morphology of lesions, cytology demonstrating acantholytic keratinocytes, characteristic histopathology, and immunofluorescence, supplemented by newer tools such as disease activity indices, eosinophilic infiltrate scoring, C-reactive protein, and antidesmocollin assays. Management centers on staged immunosuppression with glucocorticoids and steroid-sparing agents, with emerging roles for calcineurin and Bruton's tyrosine kinase inhibitors, mycophenolate, oclacitinib combinations, chrysotherapy, and plasma exchange. Prognosis is generally fair for superficial forms but guarded for deep and paraneoplastic variants.

Keywords: Pemphigus, autoimmune disorder, desmoglein, immunosuppression

Introduction

Pemphigus in dogs is an autoimmune vesicobullous to pustular skin disease characterized by acantholysis (loss of adhesion between keratinocytes) with lesions restricted to the epidermis and typically located suprabasally or subcorneally depending on the variant. It most commonly presents as pemphigus foliaceus in middle-aged dogs, but several distinct clinicopathologic forms exist with differing target antigens, depth of acantholysis, lesion distribution, and prognosis.

Definition and Basic Concepts

- Pemphigus complex comprises autoimmune skin diseases in which IgG (sometimes IgA) autoantibodies target desmosomal adhesion molecules, leading to keratinocyte detachment and intraepidermal pustule/vesicle formation.
- Lesions are confined to the epidermis, and each variant is defined by the level of epidermal cleavage (subcorneal vs suprabasal) and typical clinical distribution (face, mucosa, trunk, etc.).

Etiology and Pathogenesis

- There is a strong genetic predisposition in certain breeds (Akita, Chow Chow, Dachshund, Collie, Doberman, Newfoundland), and MHC class II/DLA alleles are suspected to underlie autoimmune dysregulation.
- Environmental triggers include drugs (cephalosporins, sulfonamides), UV light, infections (molecular mimicry), stress, and neoplasia, which together with genetic factors promote immune dysregulation ($\downarrow$ Tregs, $\uparrow$ Th2/Th17).
- Autoantibodies are mainly directed against desmoglein-1 in pemphigus foliaceus, desmoglein-3 in pemphigus vulgaris, and in some cases desmocollin-1 or plakins (paraneoplastic pemphigus).

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- Binding of these antibodies activates complement and recruits neutrophils and eosinophils, whose proteases and reactive oxygen species cause acantholysis, subcorneal/suprabasal clefting, neutrophil-rich pustules, and progressive crusting and lesion spread from face/pinnae to feet, trunk, and groin.

Classification and Key Variants

- Superficial forms: pemphigus foliaceus (PF), pemphigus erythematosus (PE), and panepidermal pustular pemphigus/pemphigus vegetans.
- Deep forms: pemphigus vulgaris (PV) and paraneoplastic pemphigus (PNP), which show suprabasal acantholysis with prominent mucosal involvement and more severe systemic illness.
- PF is the most common form in dogs, with “leafy” scaling and crusting, especially on face, nasal planum, pinnae, and footpads; vesicles and bullae are transient, and severe cases may show anorexia, depression, and fever.
- PE is considered a benign PF variant with lesions confined to face and ears, often in Collies and German Shepherds, frequently associated with nasal depigmentation and photodermatitis, and histologically shows subcorneal pustules plus lichenoid mononuclear infiltrates.
- PV is rare (< 0.1% of pemphigus cases), targets antigens near the epidermal-dermal junction (Dsg3 ± Dsg1), and produces deep erosions and shallow ulcers of oral cavity, mucocutaneous junctions, axilla, groin, and footpads; it is considered the most severe form.
- PNP is associated with neoplasia (e.g. thymic lymphoma, mammary carcinoma, Sertoli cell tumor), targets plakins and multiple antigens, and presents with severe, extensive mucosal and cutaneous ulceration and poor prognosis.

Clinical Features and Disease Activity Index

- Common clinical patterns include pustules, erosions, crusts, erythema, alopecia, and in PF loss of nasal cobblestoning with “hard pad” (erythematous swollen margins, cracking, villous hyperkeratosis) of footpads.
- Mucosal involvement is rare in PF/PE but prominent in PV and PNP, where oral cavity (gingiva, palate, tongue) lesions are often severe and debilitating.
- A canine pemphigus disease activity index scores lesion extent, lesion severity, pruritus/discomfort, mucocutaneous involvement, and systemic signs (0-3 or 0-5 scales), with total scores categorizing disease as remission/mild (0-4), mild-moderate (5-9), moderate-severe (10-14), or severe/life-threatening (≥15).
- Rapid score increase indicates flare or inadequate control, while a > 50% decrease suggests an effective therapeutic response and can guide tapering decisions.

Diagnosis and Recent Diagnostic Updates

- Core workup includes history, physical examination of lesion type and distribution, skin scrapings, cytology (impression smears from pustules/crusts), fungal/bacterial cultures, and ultimately skin biopsies for histopathology plus immunofluorescence/immunohistochemistry.
- Cytology typically shows acantholytic keratinocytes (rounded, deeply stained cells) with neutrophils and sometimes eosinophils, while secondary bacterial infection may also be present.

- Histopathology for PF reveals intraepidermal and intrafollicular subcorneal acantholytic pustules in the corneal, granular, or upper spinous layers; PV shows suprabasal clefts with “tombstoning” of basal cells and vesicle/bulla formation.
- Direct immunofluorescence demonstrates intercellular IgG (and sometimes IgA/IgM) in a “fishnet” pattern in the epidermis, sometimes with ANA positivity in PE or complex patterns in PNP (ICS + BMZ + cytoplasmic reactivity).
- Updated cytological criteria distinguish PF from superficial pyoderma: round (AC1) and boat-shaped (AC2) acantholytic cells and raft cells plus eosinophils are significantly higher in PF, whereas bacteria are more abundant in superficial pyoderma.
- An eosinophilic infiltrate score > 3 in pustules/crusts, follicles, and dermis correlates with truncal PF, while facial PF tends to have lower eosinophil scores (1-2), helping subtype distribution.
- Dogs with vasculopathic lesions in PF have longer time to remission and more adverse effects from therapy than those without vasculopathy.
- Serum C-reactive protein is significantly higher in PF than superficial pyoderma, and values >10.6 µg/ml are highly suggestive of PF in the differential diagnosis.
- Anti-desmocollin-1 antibodies are detected in almost all facial PF cases and in about 58% of truncal PF, whereas anti-desmoglein-1 antibodies occur less frequently, suggesting Dsc1 as a major target in canine PF.
- Trunk-dominant PF tends to have fewer raft acantholytic cells than facial PF, though most other clinical and histologic findings are similar between these distributions.

Differential Diagnoses and Workup Strategy

- Important differentials include pustular dermatophytosis, superficial pyoderma, demodicosis, scabies, zinc-responsive dermatosis, cutaneous adverse drug reactions, and lupus erythematosus (systemic and cutaneous).
- A structured diagnostic workup usually combines Wood’s lamp/LPCB for dermatophytes, coat brushing, tape impressions, skin scrapings, cytology, bacterial/fungal cultures, endocrine/systemic evaluation, dietary trials when indicated, and finally biopsies with histopathology and immunologic tests.

Treatment: Conventional Immunosuppression

- First-line therapy relies on glucocorticoids: oral prednisolone at 1-2 mg/kg once daily for 10-14 days, followed by tapering to 1 mg/kg every 48 h or less as control is achieved; topical low-potency steroids (1-2% hydrocortisone) can supplement localized lesions.
- Alternatives/adjuncts include methylprednisolone (≈1 mg/kg PO q24h then EOD) or triamcinolone (0.1-0.3 mg/kg/day), which are more potent and may be used short-term to gain initial control.
- Steroid-sparing agents: azathioprine (≈1.5-2.5 mg/kg q24-48h) suppresses T and B cell proliferation via purine antagonism but has delayed onset and requires monitoring for hepatotoxicity and myelosuppression.
- Chlorambucil (0.1-0.2 mg/kg q24-48h) is an alkylating agent reserved especially for cats or dogs where azathioprine is contraindicated, functioning by DNA cross-linking and lymphocyte suppression.

- Tetracycline-niacinamide combinations (250-500 mg of each q8h depending on body weight) provide mild anti-inflammatory and immunomodulatory effects and can be used in mild cases or as part of maintenance.
- Throughout therapy, secondary bacterial infections must be controlled with appropriate systemic or topical antimicrobials based on culture and sensitivity when possible.

Newer and Alternative Therapies

- Calcineurin inhibitors (cyclosporine 5-10 mg/kg q24h and topical 0.1% tacrolimus BID) suppress T-cell activation by inhibiting IL-2 and can be effective in steroid-refractory cases or localized lesions like nasal planum or periocular skin.
- Mycophenolate mofetil (about 8-12 mg/kg BID or 22-39 mg/kg/day divided) inhibits *de novo* purine synthesis in lymphocytes, offering potent immunosuppression but with GI adverse effects and variable efficacy.
- Dapsone ($\approx$ 1 mg/kg BID-TID) reduces neutrophil myeloperoxidase activity and reactive oxygen species and is useful in neutrophilic dermatoses such as PF, but carries risk of hemolysis and methemoglobinemia.
- Oclacitinib (0.4-0.6 mg/kg BID then SID) as a JAK1 inhibitor has a limited direct role in pemphigus but may alleviate pruritus and inflammation, and in combination with glucocorticoids has shown slightly better lesion reduction than glucocorticoid-azathioprine protocols in PF dogs.
- Bruton's tyrosine kinase inhibitors (PRN-473 and PRN-1008) have reduced clinical signs and anti-Dsg1 antibody levels in canine PF, suggesting they may become glucocorticoid-sparing alternatives.
- Therapeutic plasma exchange has achieved around 50% reduction in lesional body surface area in PF dogs, but remains experimental and requires further study for routine clinical use.
- Chrysotherapy (gold salts such as oral auranofin 0.05-0.2 mg/kg q12h or injectable 1 mg/kg weekly) has immunomodulating and anti-inflammatory properties and can be used as adjunctive or alternative therapy in refractory cases.
- Pulse glucocorticoid therapy (*e.g.* 10 mg/kg for 3 days then $<$ 2 mg/kg with repeat pulses as needed) and use of methylprednisolone as first-line have been associated with faster remission than traditional constant-dose regimens.

Treatment Phases and Monitoring

- Induction phase aims for rapid suppression of inflammation and autoimmunity using higher immunosuppressive doses until clear remission (no new lesions, healing of existing lesions) is achieved.
- Transition phase entails slow tapering of drugs over weeks to months to identify the minimum effective dose while avoiding relapse; if flare occurs, doses are increased and the induction phase is essentially repeated.
- Maintenance phase uses the lowest dose and frequency that keep the patient symptom-free, often with EOD steroid dosing plus one or more steroid-sparing agents.
- A dog is considered "cured" only when it has achieved remission, remains controlled on maintenance therapy, and does not relapse after complete discontinuation of treatment.

Prognosis

- Overall prognosis for pemphigus foliaceus and erythematosus is usually fair to good, especially with early diagnosis, aggressive induction, and careful long-term monitoring.
- PV and PNP carry guarded to poor prognosis because of severe mucosal disease, systemic illness, tumor associations, and frequent refractoriness to therapy.
- Relapses are common across the pemphigus complex, necessitating client education about the chronic, often lifelong nature of immunosuppressive management.

Aggressive glucocorticoid therapy, exploring different glucocorticoid options, and integrating newer targeted or adjunctive therapies can improve long-term control and quality of life in affected dogs.

Conflict of Interest

Not available

Financial Support

Not available

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How to Cite This Article

Karunyakaran DH. Pemphigus in dogs: An update. *International Journal of Veterinary Sciences and Animal Husbandry*. 2026;11(3):257-259.

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