PODODERMATITIS – a pattern based approach to diagnosis and treatment.

Including Canine interdigital furunculosis (interdigital cyst syndrome) , a 2025 perspective

Dr Robert Hilton *BVSc(Hons) MANZCVS (Canine Medicine) Cert.VD*
Yarrambat , Vic Australia
Mobile : +61 (0) 433-853560 , Email : drrobhilton@gmail.com

Introduction

Pododermatitis is defined as inflammation of the interdigital skin. That is to say that skin, the skin located between the toes and the footpads. Diseases principally restricted to the nails/claw folds or the footpads are outside the scope of this article.

The dermis and epidermis of the interdigital skin does not differ markedly from that of the rest of the body. A layer of subcutis separates the dermis of the dorsal and ventral skin (figure1). Adnexal structures, compound hair follicles and adnexal glands, are located within the two dermis's and separated form the ground substance, vascular and connective tissue elements by a basement membrane.

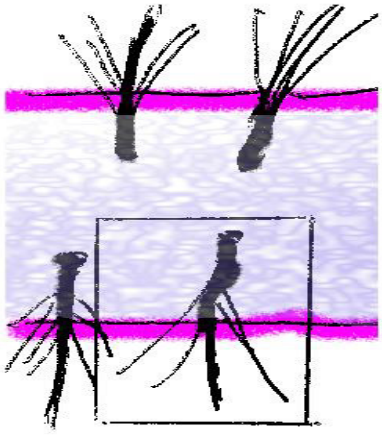


Figure 1. Representation of compound hair follicles in the interdigital skin

Special features of the interdigital skin that predispose to disease include:

- Contact with soil based potential pathogens
- A moist environment with may support microbial growth and alter the barrier function of the local skin

- Contact with potential allergens
- Microtrauma of hair follicles, predisposing to follicular rupture

Foreign body reactions to free keratin from follicular rupture and secondary bacterial infection perpetuate the process of ododermatitis (figure 2)

The interdigital skin may be involved as part of a generalised dermatitis. This article will focus on diseases that present with ododermatitis as the primary complaint

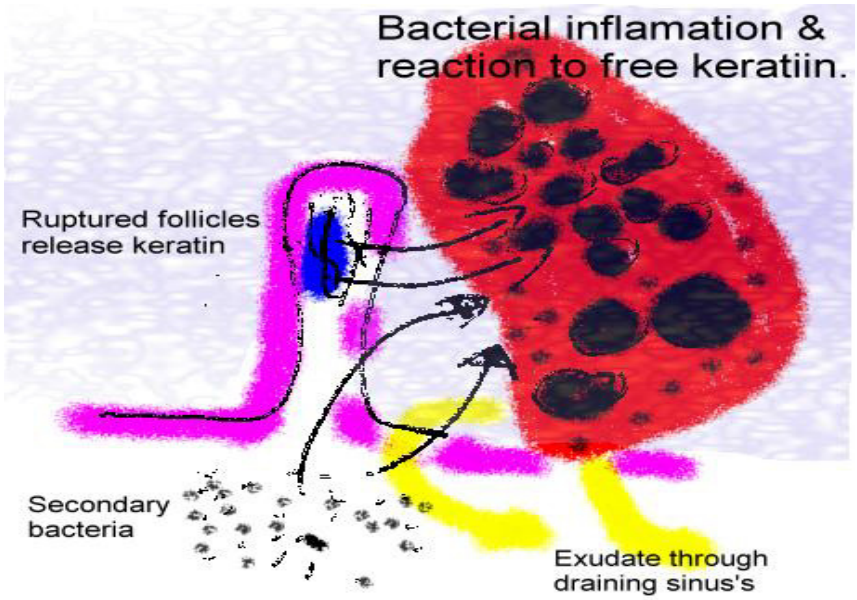


Figure 2 Foreign body reactions to free keratin from follicular rupture and secondary bacterial infection perpetuate the process of ododermatitis

Reaction patterns of the interdigital skin

1. **Inflammatory alopecia, erythema with variable levels of pruritus – leading to thickening (lichenification) and hyperpigmentation**
2. **Erosions, ulcers, nodules and draining tracts**
3. **Diseases of the footpads that may extend into the interdigital skin**

Type I Pododermatitis

- Inflammatory alopecia and erythema
- Variable levels of pruritus, may be intense
- Chronic cases : lichenification +/- hyperpigmentation
- Secondary superficial bacterial and / or Malassezia yeast infection.

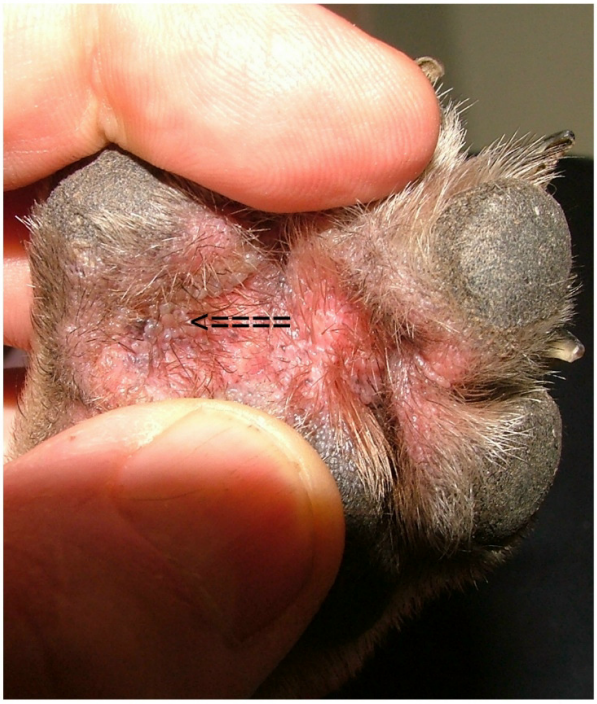
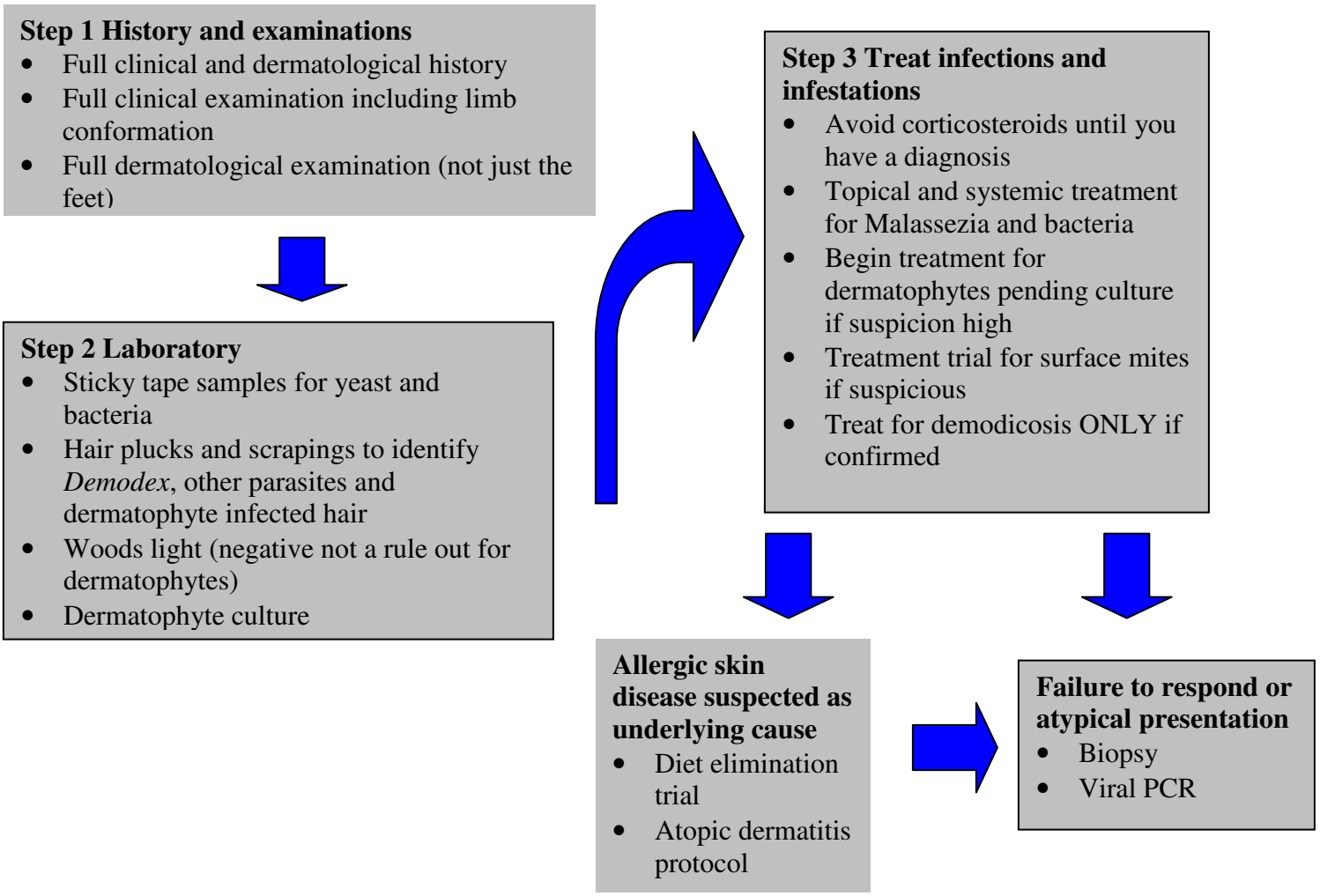


Figure 3 Erythema and mild lichenification and focal hyperpigmentation in case of Malassezia ododermatitis secondary to atopic dermatitis. Dietary reactions can result in identical lesions.

More Common	**Less Common**
Atopic dermatitis	Contact allergy
dverse cutaneous food reactions "dietary allergy"	Contact irritant dermatitis
Defects of conformation / lameness	
Dermatophytosis	Viral infections : Papilloma, Feline Herpes, Calicivirus, FIV, FeLV Poxvirus
Pododemodicosis	Miscellaneous diseases: • Cutaneous larva migrans • Surface mites (*Notoedres*, *Sarcoptes*, *Trombicula* spp) • *Trauma and burn* • *Scaling disorders.*

Table 1 – Diseases associated with Type I pododermatitis

Clinical Approach to Type 1 Pododermatitis



Type II Pododermatitis

- Erosions and ulcers
- Nodules
- Draining tracts

Deep bacterial infections – *Staphylococcus pseudointermedius* – Anaerobes – Gram negatives – Atypical bacteria including mycobacteria *Actinomyces*, and *Nocardia* spp May be secondary, especially demodicosis	**Neoplastic and paraneoplastic diseases** – German Shepherd metacarpal/tarsal fistulae – Epidermal dysplasia (mixed tissue) – Squamous cell carcinoma – Fibrosarcoma – Cutaneous lymphoma – Miscellaneous other skin neoplasms – Metastatic carcinoma – Bowenoid carcinoma (Papilloma virus)
Deep / subcutaneous fungal infections	**Miscellaneous diseases** – Interdigital cyst syndrome – Comedone syndrome – Lick granuloma – Eosinophilic granuloma – Xanthomatosis (cat: small nodules) – Foreign body reactions (grass seeds)
Immune mediated diseases Generalised – Pemphigus complex – Vasculitis and vasculopathies – Bullous pemphigoid complex – Drug eruptions	**CANINE "INTERDIGITAL CYST" SYNDROME (see below)**

Table 2 – Diseases associated with Type II pododermatitis

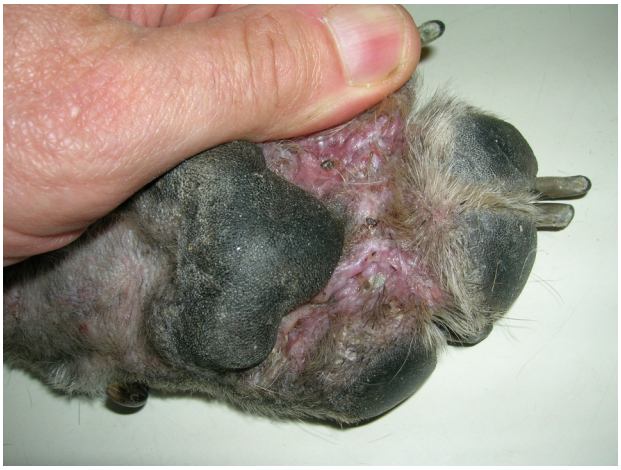


Figure 4. Chronic inflammation and deep pyoderma and furunculosis in a case of pododemodicosis

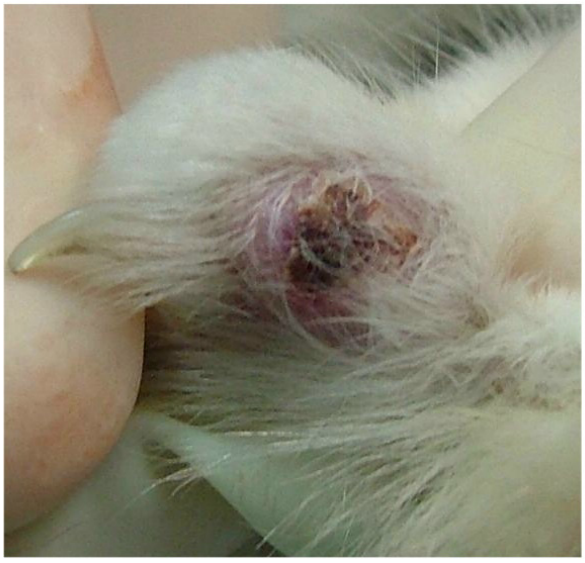


Figure 5 Nodule and draining sinus in a cat with fungal infection due to Alternaria spp

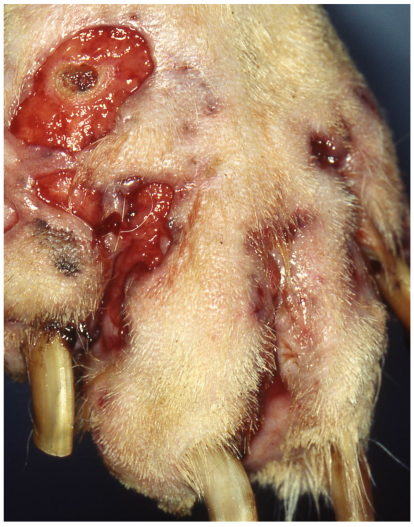


Figure 6 Ulceration with tissue necrosis and demarcated borders suggestive of vasculitis. Photo courtesy of Dr Michelle Rosenbaum

Pattern 3 Diseases of the footpads that may extend into the interdigital skin

- Hepatocutaneous syndrome (metabolic epidermal necrolysis)
- Generalised immune mediated disease (esp. pemphigus foliaceus and vasculitis)
- Zinc responsive dermatosis
- Epidermal dysplasia
- Idiopathic hyperkeratosis
- Papilloma virus

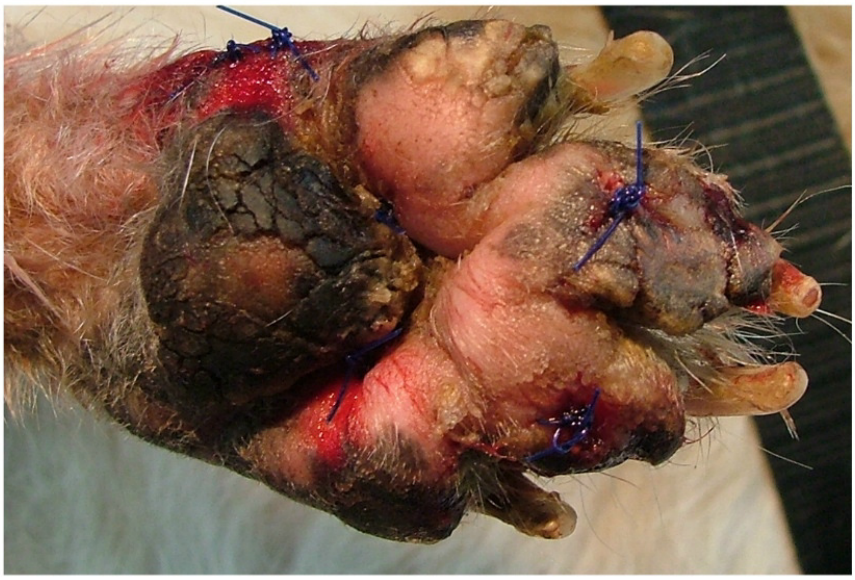


Figure 7 Hepatocutaneous syndrome. Pad hyperkeratosis, with inflammation, erosion and ulceration of pads and interdigital skin

Canine interdigital furunculosis (interdigital cyst syndrome) , a 2025 perspective

Pathogenesis

- Follicular rupture due to microtrauma is thought to a significant the primary cause. In particular any conformational deformity that permits the dog to **WALK ON HAIRED AREAS OF THE LOWER FEET**
- Other factors include body size and coat type (short haired dogs over 20kg predisposed) and other forms of dermatitis that lead to follicular rupture (Korvacs et al 2005, Duclos et al 2008).
- The syndrome is seen more commonly (but NOT limited to) short-haired hard-coated breeds.
- Free keratin results in sterile granulomas **that then become** secondarily infected.
- **The process begins on the ventral and then may rupture on to the dorsal surface** (Duclos et al 2008). See figures 9 and 10.

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TAKE HOME: As the condition progresses, inflammation and scarring result in increasing ventral food deformity with more follicular rupture in a vicious cycle.

Presentation

- Interdigital Nodules
- Ulceration

- Draining sinus's on the dorsal surface
- Swelling, inflammation and possibly comedones on the ventral pressure absorbing haired areas of the feet
- Many dogs may develop lesions before 3 years of age.

Diagnosis

- Elimination of other conditions by history, physical examination and culture.
- Rule out demodicosis by scraping and hair plucks
- Cytology is non specific pyogranulomatous inflammation. Bacteria may be difficult to visualise and their absence is NOT a rule out for secondary infection
- Histopathology. Lesions with severe secondary infection are very difficult for pathologists to interpret. Best done after 3 weeks of appropriate antibiotic therapy. Histopathology is an aid to diagnosis only as the findings of pyogranulomatous inflammation with free keratin are not necessarily specific. Histopathology however may be useful in ruling out atypical bacterial, fungal and neoplastic diseases

Treatment

Treatment of this disease can be frustrating, even for dermatologists.

1. **Identify and treat the secondary infection.**
2. **Once the infection is eliminated, address the sterile keratin-induced inflammatory process**
3. **Identify and try to correct the underlying conformational issues**

Antimicrobials

- Most cases will improve with antimicrobial treatment. Some cases respond dramatically to antimicrobials, other just improve.
- As is the case with all deep pyoderma, treatment will need to be continued for a period of time after visible clinical cure. This period was recommended to be 2-4 weeks after visible cure but current antimicrobial stewardship guidelines recommend the shorter end of this period In any case, treatment may be several weeks
- The most common organism involved is *Staphylococcus pseudintermedius*. Given the high numbers of resistant infections, **culture is regarded as essential.**
- Culture based on tissue samples collected by biopsy is the gold standard. The following protocol often yields good results:
    - Withhold topical treatment (essential) and preferably systemic antibiotics for at least 3 days
    - Wipe with saline to clean
    - Collect multiple samples by gel swab ideally from prick of an intact bulla or deep as possible inside draining sinus's. Ask the laboratory to combine multiple samples and warn them about the site of collection.

    - Interpret the results with care, E coli, Enterococcus and Proteus spp are more likely to be contaminants but in some cases , faecal organisms including Pseudomonas may be pathogens
  - Cytology is a useful tool to detect micro-organisms but is not as sensitive as culture. Organisms may always not be visible on cytology smears (Hillier 2006)
  - Topical treatment alone is NOT effective for deep pyoderma but ESSENTIAL in conjunction with systemic therapy to minimise resistance.
  - Maintenance with topical antimicrobials often is needed and may assist in preventing relapses. Foot dips in 4% hypochlorite bleach , 4-6 teaspoons/litre or 2% chlorhexidine solutions are useful

Topical and systemic immunosuppressive therapy

If the lesions have not resolved after appropriate antimicrobials AND re-culture identifies only a light growth of non-specific organisms then a trial course of prednisolone is indicated. 1.5mg/kg for 10-14 (max) days should provide a dramatic improvement in cases of sterile keratin induced inflammation

Long term options:

- **Cyclosporine at similar protocols as described for atopic dermatis (5mg/kg daily) is the most common systemic agent used to maintain cases requiring constant medication.** Some cases may be able to be tapered to 2-3x weekly
- Topical steroids may assist by both deceasing the reaction to free keratin and, by their side effect of skin atrophy, reduce keratin production. Potency = Mometasone -> Betamethasone 17-valerate -> Triamcinolone -> Prednisolone -> Hydrocortisone. Long term frequency of use is limited by skin atrophy risks. 2-3 times a week is the usual maximum
- Some cases benefit from the off label use of topical calcineurin inhibitors. Tacrolimus 0.1% (off label and compounded in Australia) or 1% pimecrolimus (Elidel, Novartis. Clients should wear gloves when applying these agents.
- Some cases will require maintenance therapy with intermittent tapering doses of systemic prednisolone.
- Oclacitinib (Apoquel R) is not indicated in this condition.

Surgery

- Carbon dioxide laser ablation from the **ventral surface** has been described in case series's with good results (Duclos et al 2008) (Frey and Varjonen 2023). **This is emerging as standard therapy where self perpetuation through pressure on haired skin results n chronic inflammation and may be preferable to long term cyclosporine use.**
- Fusion podoplasty in refractory cases (Swain et al 1991)
- Fluorescent light energy (FLE) can shorten the time to clinical resolution and minimise systemic antimicrobial use. (Lange et al 2025)

Idiopathic lymphocytic – plasmacytic pododermatitis of dogs is a syndrome identified by Breathnach et al in 2005 as an immunosuppressive responsive interdigital furunculosis syndrome with lymphocytic-plasmacytic histopathology. There is some doubt now whether this is indeed a separate entity.

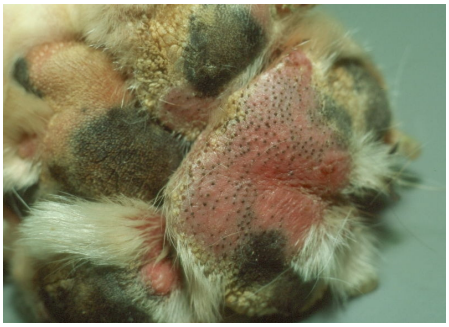

Figure 8 Ventral view of comedone formation combined with pad fusion deformity (Horse-shoe pads). Follicular plugging and comedones. No significant deep secondary infection (yet) Photo courtesy of Dr Massimo Beccati

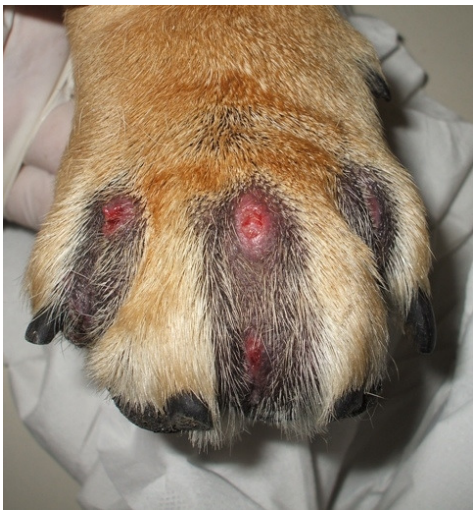

Figure 9 Dorsal draining sinus's from a similar case of ventral origin Photo courtesy of Dr Massimo Beccati

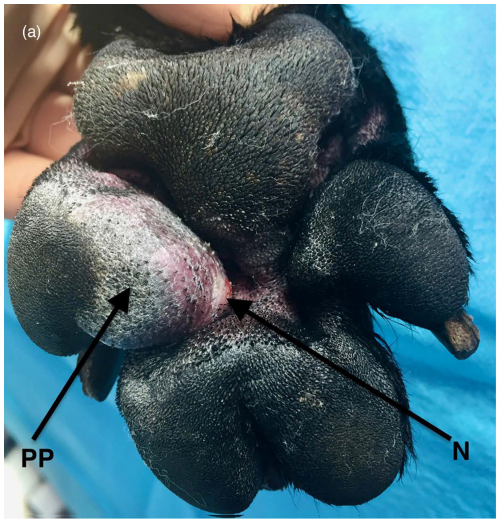

Figure 10 Pseudopad (PP) and inflammatory nodule (N) resulting in comedones -> follicular rupture and inflammation. Frey and Varjonen (2023)

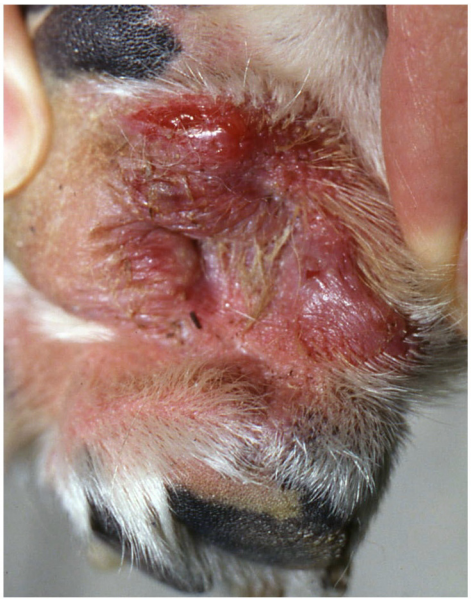

Figure 11 Bulla formation and draining sinus's. Interdigital furunculisis acute stage . Photo courtesy of Dr Michelle Rosenbaum

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