PODODERMATITIS – a pattern based approach to diagnosis and treatment.

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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](image)

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 pododermatitis (figure 2)
The interdigital skin may be involved as part of a generalised dermatitis. This article will focus on diseases that present with pododermatitis as the primary complaint.

![Diagram of bacterial inflammation and reaction to free keratin](image)

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

**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 pododermatitis secondary to atopic dermatitis. Dietary reactions can result in identical lesions.

<table>
<thead>
<tr>
<th>More Common</th>
<th>Less Common</th>
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<tbody>
<tr>
<td>Atopic dermatitis</td>
<td>Contact allergy</td>
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<tr>
<td>diverse cutaneous food reactions “dietary allergy”</td>
<td>Contact irritant dermatitis</td>
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<tr>
<td>Dermatophytosis</td>
<td>Viral infections: Papilloma, Feline Herpes, Calicivirus, FIV, FeLV, Poxvirus</td>
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<tr>
<td>Pododemodicosis</td>
<td>Miscellaneous diseases:</td>
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<tr>
<td></td>
<td>- Cutaneous larva migrans</td>
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<td></td>
<td>- Surface mites (<em>Notoedres, Sarcoptes, Trombicula spp</em>)</td>
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<td>- Trauma and burn</td>
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<td>- Scaling disorders.</td>
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Table 1 – Diseases associated with Type I pododermatitis
Clinical Approach to Type 1 Pododermatitis

Step 1 History and examinations
- Full clinical and dermatological history
- Full clinical examination
- Full dermatological examination (not just the feet)

Step 2 Laboratory
- Sticky tape samples for yeast and bacteria
- Hair plucks and scrapings to identify *Demodex*, other parasites and dermatophyte infected hair
- Woods light (negative not a rule out for dermatophytes)
- Dermatophyte culture

Step 3 Treat infections and infestations
- Avoid corticosteroids until you have a diagnosis
- Topical and systemic treatment for Malassezia and bacteria
- Begin treatment for dermatophytes pending culture if suspicion high
- Treatment trial for surface mites if suspicious
- Treat for demodicosis ONLY if confirmed

Allergic skin disease suspected as underlying cause
- Diet elimination trial
- Atopic dermatitis protocol

Failure to respond or atypical presentation
- Biopsy
- Viral PCR
Type II Pododermatitis

- Erosions and ulcers
- Nodules
- Draining tracts

<table>
<thead>
<tr>
<th>Deep bacterial infections</th>
<th>Neoplastic and paraneoplastic diseases</th>
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<tr>
<td>Staphylococcus pseudointermedius</td>
<td>German Shepherd metacarpal/tarsal fistulae</td>
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<tr>
<td>Anaerobes</td>
<td>Epidermal dysplasia (mixed tissue)</td>
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<td>Gram negatives</td>
<td>Squamous cell carcinoma</td>
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<tr>
<td>Atypical bacteria including mycobacteria Actinomyces, and Nocardia spp</td>
<td>Fibrosarcoma</td>
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<td>May be secondary, especially demodicosis</td>
<td>Cutaneous lymphoma</td>
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<td>Miscellaneous other skin neoplasms</td>
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<td>Metastatic carcinoma</td>
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<td>Bowenoid carcinoma (Papilloma virus)</td>
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<tr>
<th>Deep / subcutaneous fungal infections</th>
<th>Miscellaneous diseases</th>
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<tr>
<td></td>
<td>Interdigital cyst syndrome</td>
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<tr>
<td></td>
<td>Comedone syndrome</td>
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<td></td>
<td>Lick granuloma</td>
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<td>Eosinophilic granuloma</td>
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<td>Xanthomatosis (cat: small nodules)</td>
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<tr>
<th>Immune mediated diseases</th>
<th>Foreign body reactions</th>
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<tr>
<td>Generalised</td>
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<tr>
<td>Pemphigus complex</td>
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<td>Vasculitis and vasculopathies</td>
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<tr>
<td>Bullous pemphigoid complex</td>
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<td>Drug eruptions</td>
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<td>Lymphocytic plasmacytic canine pododermatitis</td>
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Table 2 – Diseases associated with Type II pododermatitis
Figure 4. Chronic inflammation and deep pyoderma and furunculosis in a case of pododermicosis

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 cyst syndrome (interdigital furunculosis)

Pathogenesis
- Follicular rupture due to microtrauma is thought to be the primary cause. Other factors include body size (dogs over 30kg 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 become secondarily infected.
- The process begins on the ventral surface of the foot as follicular cysts and plugging which may rupture onto the dorsal surface (Duclos et al 2008).

Presentation
- Interdigital Nodules
- Ulceration
- Draining sinus’s on the dorsal surface
- Many dogs may develop lesions before 3 years of age.

Diagnosis
- Histopathology, best done after 3 weeks of appropriate antibiotic therapy.
- Lesions with severe secondary infection are very difficult for pathologists to interpret.

Treatment
Treatment of this disease can be frustrating and needs to be tailored to the individual. The following is based on the authors' experience and collected responses from the Vetderm list server.

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 3-4 weeks after visible clinical cure. This often means months of treatment.
- Cultures from 3mm punch biopsy samples are recommended especially if drug resistant organisms are suspected.
- The most common organism involved is *Staphylococcus pseudintermedius*. At present in Australia most stains respond to cephalexin at 25mg/kg twice daily. Resistant strains have emerged overseas and culture is indicated if there is a failure to respond.
- Because of soil contact and the moist interdigital environment, many other organisms (including *Pseudomonas* spp) can be isolated from pododermatitis cases.
Cytology is a useful tool to detect micro-organisms but is not as sensitive as culture. *Pseudomonas* spp may not be visible on cytology smears (Hillier 2006).

Antibiotic courses often need to be repeated. Frequently relapsing cases can sometimes be maintained on weekend pulse therapy. In an era of emerging resistance, weekend pulse therapy is open to question.

Maintenance with topical antimicrobials often is needed and may assist in preventing relapses. (mupirocin, silver sulfadiazine, chlorhexidine).

**Topical and systemic immunosuppressive therapy**

- Topical steroids may assist by both decreasing the reaction to free keratin and, by their side effect of skin atrophy, reduce keratin production. Potency = Mometasone -> Betamethasone 17-valerate -> Triamcinolone -> Prednisolone -> Hydrocortisone
- Some cases benefit from the off label use of topical calcineurin inhibitors. Tacrolimus 0.1% (off label and compounded in Australia) is used by several clinicians with good effects. Topical tacrolimus has shown to benefit cases of atopic dermatitis and cutaneous (discoid) lupus erythematosus. There are fewer anecdotes to support the use of 1% pimecrolimus (Elidel, Novartis) and absorption of this compound into the deep dermis may be poorer (Getzwiller 2006). Clients should wear gloves when applying these agents.
- Some cases will require maintenance therapy with tapering doses of systemic prednisolone or may respond to cyclosporine at similar protocols as described for atopic dermatitis. Tetracycline/niacinamide or pentoxifylline therapy may maintain remission or lower corticosteroid use in some cases.

**Keratolytics**

- Benzyl peroxide has keratolytic, antiseptic and possible follicular flushing effects. It is very drying and requires moisturizing agents after use. Sulphur / Salicylic acid shampoos (Sebazole, Virbac) may be better tolerated long term.

**Surgery**

- Surgical resection of isolated localized lesions
- Carbon dioxide laser ablation from the ventral surface has been described in a case series with good results (Duclos et al 2008)
- Fusion podoplasty in refractory cases (Swain et al 1991)

**Comedone syndrome**

This is often associated with a horse-shoe pad deformity and leads to significant gait abnormalities. There is controversy as to what comes first; the gait abnormality or the comedones and pad deformity. The author feels more cases are primary and then lead to gait abnormalities.
Clinical appearance

- Ventraly, fused pads forming a horse shoe bridge and allowing walking on interdigital skin.
- Distinct ventral comedones (see fig 8)
- Secondary infection results in draining sinus’s to form on the DORSAL aspect of the foot. (see fig 9)
- Bulldogs and larger brachycephalic breeds seem predisposed.

Diagnosis is based on clinical appearance, exclusion of Demodex by scrapings and hair plucks and biopsy (if needed)

Treatment

In acute cases, management of infection and inflammation as per interdigital cyst syndrome

Chronic cases will benefit from:

- Topical higher potency corticosteroids for both their anti-inflammatory action and importantly their ATROPHAGENIC action in reducing comedone formation. The frequency of use and potency needs to be adjusted so as not to induce unwanted skin atrophy. There is a low but not negligible risk of inducing local calcinosis cutis.
- Use of keratolytic and keratoplastic shampoo locally to regularise keratinisation and unplug follicles. Sulphur/salicylic acid combinations are useful. The author uses Sebazole (®Virbac)
- The use of antimicrobial cream in any folds after shampoo. Miconazole or clotrimazole have good activity against both yeast and gram + cocci.

Figure 8 Ventral view of Comedone syndrome. Follicular plugging and comedones. No significant deep secondary infection (yet) Photo courtesy of Dr Massimo Beccati
Idiopathic lymphocytic – plasmacytic pododermatitis of dogs

Recently, a syndrome of chronic refractory pododermatitis has been identified in dogs. These dogs were negative to ectoparasites, failed to respond to appropriate prolonged courses of antibiotics, failed to respond to dietary elimination trials and failed to meet the criteria for atopic dermatitis. They however responded well to immunosuppressive therapy. The name idiopathic lymphocytic – plasmacytic pododermatitis of dogs refers to the histological findings and has a different presentation to the similar-named disease of the footpads in cats (mushy pad disease).

Pathogenesis
- The pathogenesis is unclear but the presence of large numbers of lymphocytes and plasma cells is suggestive of immune reactivity and antigenic drive. (Breathnach et al, 2005)
- There is no evidence to support a reaction to systemic disease or to suggest an immunodeficit like is found in demodicosis or German Shepherd pyoderma.
- There is evidence to support increased local dendritic cell numbers and enhanced antigen presenting activity together with an increased Th2 and a reduced Th1 response. (Breathnach et al 2006, Breathnach et al 2008)

Presentation
- There is no age, breed or sex predilection
o Consistent signs include alopecia, erythema, pain, pruritus and thickening of the skin.

o Combinations of erosions, ulcers, nodules and draining tracts are commonly seen on the dorsal and ventral surfaces of the feet

o The lesions may develop on one foot but then spread to mostly involve the other feet.

o Secondary infection may be identified cytologically and by culture but prolonged courses of appropriate antibiotics fail to provide clinical resolution.

**Diagnosis**

o The diagnosis is a clinical one based on exclusion and confirmed by typical histopathology and response to immunosuppression.

o Differentials including allergic skin disease (dietary, contact and atopic), infection and ectoparasites (especially pododemodicosis) need to ruled out by appropriate testing and response trials

o Histopathology is best done after at least 3 weeks of appropriate antibiotic therapy. Lesions with severe secondary infection are very difficult for pathologists to interpret.

**Treatment**

o Published studies indicate that tapering doses of systemic corticosteroids or cyclosporine is required to maintain remission. (Breathnach et al 2005).

o Topical immunosuppressants (as described above) may reduce the reliance on systemic medication.

Figure 10 Bulla formation and draining sinus's. Canine lymphocytic-plasmacytic pododermatitis. Photo courtesy of Dr Michelle Rosenbaum
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