

Pododermatitis

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Introduction

Pododermatitis - inflammation of the paws - may affect the nail fold (paronychia), interdigital spaces, foot pads, claws and/or remainder of the skin of the paws. When presented with a case of pododermatitis, familiarity with the list of potential differential diagnoses for various presentations may prove beneficial to limit the most likely etiology. Listed below are differentials to consider based on species, presence or absence of symmetry and single or multiple digits or feet affected:

Canine Asymmetric Pododermatitis: Trauma, Irritant, Foreign body, Infection (Bacterial : *Staph. intermedius*, *Nocardia*, *Actinomyces*, *Pseudomonas*, *Proteus*; Fungal: dermatophytes, *Malassezia*, blastomycosis, cryptococcosis, eumycotic mycetoma, candida), Parasitic-demodex , Neoplasia , Miscellaneous: acral lick dermatitis (multitude of causes), calcinosis circumscripta, arteriovenous fistula, osteomyelitis

Canine Symmetric Pododermatitis: Irritant contact, Allergies - atopy, food, contact, Infection (bacterial - *Staph. Intermedius*; Fungal-*Malassezia*), Parasitic: (demodex, pelodera, hookworms, leishmaniasis), Autoimmune (Pemphigus foliaceus, P. vulgaris, bullous pemphigoid, SLE). Immune Mediated (sterile granuloma/pyogranuloma, cold agglutinins, vasculitis, Dermatomyositis, drug eruption, EM-TEN), Metabolic (Superficial necrolytic dermatitis, calcinosis circumscripta), Nutritional (Zinc responsive dermatosis), Immune suppression (congenital or acquired)(bacterial, dermatophytes, candidiasis, demodicosis), Psychogenic/neurogenic (Acral mutilation of GSH pointers), Congenital (Familial vasculopathy of GSD, Vasculitis of Jack Russell Terriers, idiopathic footpad hyperkeratosis, Familial hyperkeratosis in Irish terriers, Kerry blue terriers and dogues de Bordeaux, acrodermatitis of bull terriers, tyrosinemia), Miscellaneous (Dermatofibrosis, neoplasia, distemper).

Feline Pododermatitis: Immune mediated (Pemphigus foliaceus, plasma cell pododermatitis, drug eruption, EM-TEN), Allergic (Atopy, food sensitivity), Eosinophilic granuloma (may or may not be allergy related)

Diseases Restricted to One or Two Claws: Trauma, Bacterial Infection, Dermatophytosis/fungal infection, Neoplasia

Canine Symmetric Onychomadesis (Nail Sloughing:) Autoimmune/Immune Mediated (Pemphigus/pemphigoid, SLE, "Lupoid" onychodystrophy, Vasculitis, Cold agglutinin disease, drug eruption), Superficial necrolytic dermatitis, Hypothyroidism?, Neoplasia, Food sensitivity, Bacterial infection, Idiopathic

Canine Onychodystrophy (Deformed/Friable Nails: Lupoid onychodystrophy, Primary seborrhea (e.g., cocker spaniel), Senile change, Idiopathic, Acrodermatitis in bull terriers

Feline Paronychia: Pemphigus foliaceus, Bacterial, Dermatophytosis, Malassezia

Feline Nail Disease: Onychorrhexis (trauma; post inflammatory; dermatophytosis),
Onychomadesis (cold agglutinin disease, drug eruption, vasculitis), Neoplasia (Pulmonary
Bronchial Adenocarcinoma)

Pododermatitis - Specific Diseases

Demodicosis

Demodicosis is a mandatory differential diagnosis for any canine pododermatitis, whether symmetrical or involving only one paw. Demodex infections may be restricted to only the paws in some cases. Diagnosis is made by hair plucking or scraping. In patients with very chronic paw changes with excessive scarring, biopsies may be necessary to rule out this diagnosis. Some breeds, (Chinese Shar pei) may also require a biopsy for demodex diagnosis. Patients with only one paw or all feet involved should typically be classified as generalized demodicosis and worked up and treated as such. Pododemodicosis cases are often difficult to treat topically with localized or spot therapy (i.e., Goodwinol), and most require systemic therapy (e.g., 0.3-0.6 mg/kg oral ivermectin or moxidectin daily or every other day in non herding breeds or milbemycin oxime, 1-2 mg/kg daily) More recently a topical amitraz/metaflumizone product (Promeris®) has been used successfully in the treatment of demodicosis. Typical protocol includes topical application every 2 weeks until 2 negative skin scrapes then once monthly. Regardless of the type of therapy treatment should be for 2 months beyond remission and may be needed for life. For adult onset demodicosis one should always look for underlying immunocompromising diseases ie, cushings, diabetes, neoplasia, etc, as a predisposition to the development of this problem.

Allergic Dermatitis

Both atopy and food sensitivity in the dog commonly cause pododermatitis usually in association with pruritus. Salivary staining is common. The dermatitis produced is often diffuse in the interdigital and ventral interpad spaces and may also involve the flexor surface of the front paws and extensor surface of the rear paws just proximal to the carpal and tarsal pads. Claw fold involvement (paronychia) may predominate in some. This dermatitis is commonly complicated by secondary bacterial and/or *Malassezia* dermatitis (especially interdigital spaces, claw folds). These secondary infections should be identified and treated. Milder *Malassezia* cases may be treated with an antifungal/antibacterial topical shampoo or wipe (Containing miconazole, ketoconazole, climbazole and chlorhexidine). Many cases will require systemic therapy with oral antifungals (ketoconazole, fluconazole itraconazole) followed by maintenance oral and/or topical therapy.

Recurrent Bacterial Interdigital Pododermatitis

Bacterial pododermatitis in the canine is usually associated with *Staphylococcus intermedius*. Lesions are usually more focal, crusted, papular or pustular and may produce draining tracts. Lesions are often incorrectly referred to as "interdigital cysts." Differential diagnoses include other bacterial and fungal infections, foreign bodies and sterile nodular granuloma/granulomas. Short-coated breeds (e.g., Doberman, Mastiff, English Bulldog) appear to be prone to idiopathic (no systemic disease documented) recurrent bacterial infections in this area. In the English Bulldog conformational defects are also a major predisposing factor. In the author's practice, common therapies for such cases most commonly include higher doses of antibiotics such as cephalexin or fluorquinolones until at least 2 weeks beyond remission. Some cases need culture and susceptibility testing to identify appropriate antimicrobial choices. Resolution may

be facilitated by the use of topical mupirocin (Bactoderm, Pfizer) q 12h and germicidal shampoos (especially chlorhexidine or benzoyl peroxide containing). Pentoxifylline (20 -30 mg/kg q 8-12h) may also be of benefit to enhance perfusion, reduce fibrosis and reduce inflammation in chronic, fibrotic lesions. Some routinely use metronidazole (10 mg/kg q 12h) as an adjunctive therapy to help reduce inflammation. Very recurrent problems are treated with long term, lower dose daily cephalexin or pulse cephalexin therapy (3 days per week) or with bacterins (Staphage Lysate, Delmont Laboratories).

Vasculitis

Vasculitis often involves the extremities and not uncommonly the footpads in both dogs and cats. Lesions are often focal, erosive to ulcerated or have well-delineated, pale or "punched out" lesions and interestingly may often appear in the more central portions of the pad (pressure points). Patients may or may not have other skin lesions (e.g., purpura, erosions/ulcerations, dermatitis, edema) or claw dystrophies. The diagnosis is supported by skin biopsy, of deep samples from the margins of lesions. Although many cases of vasculitis are idiopathic, emphasis must be placed on looking for other underlying causes (e.g., local bacterial infection, systemic diseases such as Rickettsia or systemic lupus erythematosus). Treatment for idiopathic vasculitis often includes pentoxifylline (15-20 mg/kg q 8h) as a first choice with cyclosporine (Atopica) or immunosuppressive dosages of glucocorticoids (e.g., 2.2-4.4 mg/kg q 24h or prednisone/prednisolone to start) in more severe cases. Other therapies that may be beneficial include glucocorticoids combined with azathioprine, tetracycline and niacinamide, (or doxycycline and niacinamide), dapsone and sulfasalazine Tacrolimus topically (ProTopic®) may also be useful for localized lesions.

Pemphigus Complex

Inflammation/crusting at the pad/skin junction is common on the paws of dogs with pemphigus foliaceus and pemphigus vulgaris (Rosenkrantz WS, 2004). There is usually interdigital involvement and paronychia is not uncommon. In some patients, hyperkeratosis, crusting, inflammation and erosion will involve the pads of all four feet. In cases of Pemphigus foliaceus, these lesions can occasionally be , the only areas involved at initial presentation. Pain is variable, but may be the primary reason for presentation. Some dogs will be reluctant or unable to walk because of the discomfort. Severe lesions may also have pustule formation, often seen as a greenish/yellowish discoloration of the pad. Significant epithelial loss of the pads may be encountered. Purulent material, if present, should have a cytologic examination performed. The presence of large numbers of neutrophils and/or eosinophils and acantholytic keratinocytes would be highly suggestive of pemphigus foliaceus or pemphigus vulgaris. Bacteria are usually not present in the cytology of unruptured pustules, but may contaminate ruptured lesions. In cats, with pemphigus foliaceus, the paws (especially clawfolds) are the most common area for the initial development of skin lesions (Preziosi, Goldschmidt, Greek, 2003). In one study (Greek, 1993) initial affected sites in the cat were clawfolds (78%), paws (43%), face (39%), ears (30%). Pad changes include swelling, hyperkeratosis, scaling, crusting, hardening and fissuring. Pain is variable, and may be severe. Pain may be disproportionately severe for the degree of pad change present. Diagnosis is supported by cytology (look for greenish/yellowish discoloration at pad/skin junction) and confirmed by biopsy.

Therapy: Treat secondary bacterial and *Malassezia* infections and utilize classic immunosuppressive/anti-inflammatory drugs such as glucocorticoids, azathioprine, +/- tetracycline/niacinamide, cyclosporine. Cyclosporine can be very effective in cases of feline pemphigus but typically not as a sole therapy in the dog.

Superficial Necrolytic Dermatitis (Hepatocutaneous Syndrome, Metabolic Dermatitis, Metabolic Epidermal Necro(ly)sis)

SND is an uncommon dermatosis seen in dogs and recently has been described in the cat. In dogs, SND has been associated most commonly with idiopathic hepatocellular collapse, cirrhosis, glucagon producing pancreatic adenocarcinoma, hyperglucagonemia and glucagon secreting liver metastases (primary tumor not found, hepatopathy secondary to phenobarbital/dilantin administration, hepatopathy possibly associated with primidone or phenobarbital and hepatopathy secondary to ingestion of mycotoxins (Campbell, Matousek, Lightensteiger, 2000) (March, Hillier, Weisbrode,). In the author's practice a few cases associated with inflammatory liver disease (chronic active hepatitis, etc.) have also been seen. The disease is generally seen in middle aged to older dogs. and may wax and wane. Concurrent diabetes mellitus is relatively common (especially later in the course of the disease). Pad and/or pad/skin junction lesions are common early associations with this disease and often consist of inflammation, crusting and hyperkeratosis progress to fissuring. All pads are usually involved. The interdigital spaces and nail folds are also typically inflamed and crusty. With progressive involvement and fissuring, the paws often become very painful and lameness and reluctance to walk may present significant problems. Other areas of skin involvement are often seen (muzzle, periocular, perioral, perianal, perivulvar, scrotal, pressure points over elbows and hocks). In severe cases, the claws may be dystrophic and may slough. Secondary infections of paw lesions are common and may contribute significantly to pain, lameness or pruritus. These include bacteria (usually *Staphylococcus*), *Malassezia*, and occasionally candida or dermatophytes.

Diagnosis is by skin biopsy (characteristic parakeratosis, superficial epidermal vacuolation, epidermal hyperplasia). Once the diagnosis is made the author also prefers to evaluate the liver via abdominal ultrasound and in most cases ultrasound guided liver biopsy.

Treatment for SND primarily revolves around the finding that these patients have significant deficiencies in amino acids. Potential options may include the following: 1. Diet high in good quality protein (e.g., supplement with Hill's a/d). Enteral or parenteral feeding may be necessary 2. Management of diabetes mellitus if present. Response to insulin may be erratic. 4. Symptomatic treatment for secondary infections. 5. Amino acid supplementation: Egg yolk supplementation (3-6 yolks per day) has been noted to be of some benefit, perhaps because of the amino acid profile provided. Others have used protein supplements favored by body builders, Intravenous amino acid therapy has been noted to benefit people with hepatopathy related SND. A number of clinicians have also seen significant benefit from canine cases treated with products such as FreAmine III or Aminosyn 10% Crystalline Amino Acid Solution (Abbott Laboratories). Protocols include 500 ml per dog (or 25 ml/kg) administered slowly over about 8-12 hours in a large central vein (jugular vein). Consideration should be given to measuring blood ammonia prior to this infusion in that it is possible to contribute the hepatic encephalopathy with this therapy. The patient is re-examined in 7-10 days. If significant improvement is noted, no further infusions are given. Prolonged remissions have been noted after only one infusion. If minimal to no response is noted, the infusions are repeated every 7-10 days for 4 treatments. If an individual does not improve in this time, it is unlikely that they will respond. The infusion is repeated with each exacerbation of the skin lesions. Some patients go several months between infusions but others who require monthly infusions to maintain remission. As the disease progresses, the need for infusions will likely increase. However, there are anecdotal reports of indefinite remissions following the first treatment. 6. Essential fatty acid

supplementation - we have, at present been supplementing primarily with omega 3 fatty acids at twice the bottle dosage of a high potency omega 3 fatty acid supplement (3 V caps - DVM pharmaceuticals). 7. Zinc supplementation is also instituted in all individuals. We have used 2mg/kg/day of zinc methionine. 8. Niacinamide therapy may also be considered - 250-500 mg/dog q 8 – 12h. 9. For focal inflammatory lesions (e.g., pododermatitis), when infection problems have been as well as possible controlled, consider the use of topical glucocorticoids - e.g., generic triamcinolone acetonide BID initially. Gradually reduce this frequency and, if possible, switch to hydrocortisone for long term, maintenance therapy. 10. Systemic steroids (e.g., prednisone starting at 1 mg/kg/day) have been noted to benefit some individuals. Effects are usually transient (will eventually become refractory to these dosages of therapy) and there is concern for exacerbating diabetes mellitus. 11. Ketoconazole as been used with some benefit in this disease (5-10mg/kg q 12h perhaps because of its effects on secondary Malassezia infections or perhaps for its antiinflammatory and antipruritic effects

Idiopathic Digital Hyperkeratosis (Also see Localized Keratinization Defects)

Hyperkeratosis of the footpads, with or without involvement of the planum nasale is seen as an idiopathic disorder, usually in older dogs. However, it may occur in any aged individual. Certain breeds, such as the cocker spaniel, may be predisposed. Hyperkeratotic "feathers" or "fronds" are most commonly noted at the margins of the pads. In some instances, the hyperkeratotic tissues may be very hard, resulting in fissures. This likely causes the pain sometimes associated with this disease. If depigmentation, inflammation, erosion or ulceration is present in association with these lesions, consideration should be given to differentiating this disorder from SLE, pemphigus complex, SND and cutaneous lymphoma. The diagnosis is generally based on history and physical examination, and, if necessary, skin biopsy. Histologically, idiopathic pad

hyperkeratosis is characterized by epidermal hyperplasia and marked orthokeratotic to parakeratotic hyperkeratosis. Therapy is considered only if the hyperkeratosis is problematic. Excess keratin may be removed with scissors or a blade. When the keratin is not hard/dry, keratolytics may be applied (e.g., KeraSolv gel, DVM Pharmaceuticals - 6.6% salicylic acid, 5% sodium lactate, 5% urea in propylene glycol or Retin A (tretinoin, Ortho). When the keratin is hard, rehydrating the pad by soaking the paw for 5-10 minutes, then applying an occlusive agent (petrolatum or 50:50 propylene glycol in water) may be of benefit. This is done daily initially. Debris is removed by shampooing every 3-7 days. Owners must be warned of the potential "mess factor" associated with these topical therapies. Consideration should also be given to looking for and/or treating secondary bacterial infections. Another topical product tried by the author combines an occlusive agent along with germicidal effects (Bag Balm by Dairy Assoc. CO, Inc. Lyndville VT 05851. This contains 8 hydroxy-quinoline sulfate 0.3% (germicidal) in a petrolatum and lanolin base. The product is applied once or twice daily initially, and then the frequency is gradually reduced to maintenance. Oral Vitamin A therapy may also be of benefit. Dosage is 8,000-20,000 units q 12 h. The author generally starts with 8,000 to 10,000 units q 12 h. Retinoid therapy (e.g., isotretinoin at 1 mg/kg/day or acitretin 0.5 mg/kg/day) may have some efficacy but the high cost and need for continued therapy are prohibitive.

Zinc Responsive Dermatitis

The footpads of dogs with zinc responsive dermatosis may become hyperkeratotic (as noted in the Malamute, Siberian Husky, and occasionally in other breeds such as the Doberman pinscher and Great Dane) (White S, et al 2001). This condition can also be seen in dogs that are on diets that are deficient in zinc. This was a problem in California years ago associated with certain generic dry dog foods. Zinc responsive dermatosis pad lesions are usually

relatively mild, compared to other skin lesions. Pad hyperkeratosis is most commonly noted in conjunction with skin lesions involving other areas of the body. Diagnosis is by skin biopsy (widespread parakeratotic hyperkeratosis) and response to zinc supplementation (2 mg/kg/day zinc methionine, 5 mg/kg/day zinc gluconate or 10 mg/kg/day zinc sulfate). The potential multifactorial nature (nutritional?) of this problem is supported by the fact that some individuals respond better when they are treated with essential fatty acids, or low every other day dosages of glucocorticoids (improved absorption?) or hypoallergenic or restrictive diets. Very refractory cases have been treated with IV zinc sulfate (10-15 mg/kg zinc sulfate; diluted 1:1 with saline and given very slowly IV. Panting or possible anaphylaxis, seizures have been anecdotally reported. Perivascular leakage will produce severe local necrosis, if leakage occurs, recommend intralesional steroid and topical DMSO. IV therapy is repeated once weekly for 4-6 weeks, then every 1-6 months for maintenance. Note: this dose is for zinc sulfate (the salt) and not elemental zinc. 50 mg/ml of zinc sulfate contains 0.2 mg/ml elemental zinc.

Feline Eosinophilic Granuloma Complex

Eosinophilic plaques and eosinophilic granulomas may occasionally be noted to affect the pad or pad/skin junction of cats. Although underlying hypersensitivity disorders must be explored (e.g., atopy, food and insect sensitivity), many of the cases appear to be idiopathic. Diagnosis is by skin biopsy. Response to glucocorticoid therapy has generally been good (methylprednisolone - 1.1-2.2 mg/kg q 24h initially, then tapered to q 48-72h). Some cases may require alternative glucocorticoids, (i.e. triamcinolone) or other immunosuppressive therapies such as chlorambucil, gold salts or cyclosporine. Spontaneous resolution may be noted.

Feline Plasma Cell Pododermatitis

The underlying cause of this problem is not known, but likely relates to an immunologic disorder

(Guaguere E, Hubert B, Delabre C, 1992). In some cases, seasonal changes suggest an allergic basis. Some cases may be associated with underlying viral disease (i.e. FIV, FeLV). The earliest change seen is usually the development of a white, scaly, cross hatch pattern and scaling of the pads. Occasionally, the pads may progress to erosions or ulcerations and often appear swollen or “inflated”. There may be significant hemorrhage. These changes vary from being asymptomatic to involving significant pain and lameness. Systemic signs are also variable (pyrexia, lethargy, anorexia, peripheral lymphadenopathy). General laboratory screening usually reveals a hyperglobulinemia. Pad biopsies show a perivascular accumulation of plasma cells, with lesser numbers of lymphocytes and neutrophils. Leukocytoclastic vasculitis has been described. It has been suggested that the early lesions may involve significant infiltration with eosinophils. Some cases will spontaneously resolve with no therapy. In one study glucocorticoids cleared 4/6 cases (Pereira, PD and Faustino, AMR 2003). Therapeutic options include doxycycline (5 mg/kg q 12h; for antiinflammatory effect) (Bettenay SV, Mueller RS, Dow K, Friend S, 2003), glucocorticoids (prednisolone at 4.4 mg/kg q 24h), methylprednisolone acetate, oral triamcinolone acetonide beginning at 2-4 mg/cat q 24h, glucocorticoids and gold salts or glucocorticoids and chlorambucil or surgical excision. Cyclosporine has also produced favorable results at 5 mg/kg q 24h. Some treated patients may be able to eventually weaned off all medications, while others require ongoing therapy at lower dose or less frequent administration.

Paraneoplastic Alopecia and Internal Malignancies in the Cat

Cats with pancreatic adenocarcinoma or bile duct carcinomas may present with a symmetric alopecia involving the ventrum and extremities (Turek, 2003). Hairs are usually readily epilated. The footpads or footpad/skin junctions may be dry and scaly/crusty and occasionally fissured

and painful. Diagnosis is by skin biopsy from haired areas of the body (severe follicular and adnexal atrophy with follicular miniaturization and mild perivascular inflammation). Footpad biopsies may not be as rewarding and primarily show chronic inflammatory and secondary infections. It is also recommended to do workups to document visceral neoplasia. The prognosis is grave.

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