

**CANINE AND FELINE PODODERMATITIS:
ACCEPTING THE CHALLENGE - PART II
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CANINE “PADODERMATITIS” (Inflammatory disease targeting the pads)

Vasculitis

Vasculitis often involves the extremities and not uncommonly the footpads in both dogs and cats. Lesions are often focal, erosive to ulcerated or "punched out" and often appear in the more central portions of the pad (pressure points?). Patients may or may not have other skin lesion (e.g. purpura, erosions/ulcerations, dermatitis, edema) or claw dystrophies.

The diagnosis is supported by skin biopsies (recommend deep skin biopsies 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). A familial tendency towards the development of a such lesions has been seen in the German Shepherd dog (vasculopathy in the GSD) and in the Jack Russell Terrier.

Treatment for idiopathic vasculitis include immunosuppressive dosages of glucocorticoids (e.g. 1.5 - 2.2mg/kg/day of prednisone/prednisolone to start), pentoxifylline (10 - 25 mg/kg BID or TID) with or without glucocorticoids (appears to work better if used in conjunction with glucocorticoids), tetracycline and niacinamide, or doxycycline and niacinamide, cyclosporine (starting at 5 mg/kg/day), glucocorticoids and azathioprine or sulfasalazine (20 mg/kg TID).

Pemphigus Complex

It is common to see inflammation/crusting at the pad/skin junction of dogs with pemphigus foliaceus and pemphigus vulgaris. There is usually interdigital involvement and paronychia is relatively common. With chronicity, the pads themselves may go on to become symmetrically involved. The pads may become crusted, hyperkeratotic, hard, inflamed and fissured or eroded. In some dogs with pemphigus foliaceus, the feet may be the only area of involvement at initial presentation. Pain is variable, but may be the initial reason for presentation. Some dogs will be reluctant or unable to walk because of the discomfort.

Pustule formation, often seen as a greenish/yellowish discoloration of the pad may be seen. Significant epithelial loss may be encountered.

The diagnosis is suggested by cytologic examination (impression smears of debris taken from unbroken pustules, or from moist areas under crusts). The presence of large numbers of neutrophils, variable numbers of eosinophils and large numbers of acantholytic keratinocytes would be highly suggestive of pemphigus foliaceus or pemphigus vulgaris. Bacteria are not present in the cytology of unbroken pustules, but may contaminate broken lesions. The diagnosis is confirmed by biopsy. If reasonable, consider placing the patient on a systemic antibiotic for 2-3 weeks prior to biopsy to minimize potentially confusing changes due to secondary bacterial infections.

Therapy: Consideration should be given to looking for and resolving secondary bacterial and *Malassezia* infections. Therapy is otherwise with classic immunosuppressive/anti-inflammatory drugs including glucocorticoids, glucocorticoids and azathioprine, tetracycline or doxycycline and niacinamide (usually not effective), mycophenolate or oral cyclosporine. Topical therapy with tacrolimus (Protopic, Fugisawa) initiated at BID may be advantageous as an adjunctive treatment.

Superficial Necrolytic Dermatitis (Hepatocutaneous Syndrome, Metabolic Dermatosi, Metabolic Epidermal Necrosis)

SND is an uncommon dermatosis seen in dogs and rarely described in the cat. In dogs, SND has been associated most commonly with idiopathic hepatocellular collapse, (most common), 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. The disease is generally seen in middle aged to older dogs. The skin disease 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. Inflammation, crusting and hyperkeratosis progress to fissuring. All pads are usually involved. The interdigital spaces and nail folds are also inflamed and crusty. With progressive involvement and fissuring, the feet often become very painful resulting in lameness and reluctance to walk. In severe cases, the claws may be dystrophic and may

slough. Other areas of skin involvement are usually seen (muzzle, periocular, perioral, perianal, perivulvar, scrotal, pressure points over elbows and hocks). Secondary infections of foot lesions are common and may contribute significantly to the symptomatology. These include bacteria (usually *Staphylococcus*), *Malassezia*, and occasionally candida or dermatophytes. Diagnosis is by skin biopsy (characteristic parakeratosis, superficial epidermal vacuolation, epidermal hyperplasia). The most effective means of improving the foot lesions is to work at resolution of the underlying cause and/or systemic supportive care (IV amino acids most predictably beneficial; high, good protein diet, oral egg yolks, oral essential fatty acids, zinc supplementation, oral niacinamide, oral glucocorticoids (to be used very judiciously in light of concurrent problems). Emphasis is placed on resolving secondary infections (bacterial and fungal) of the feet. Once this has been achieved, symptomatic reduction of the inflammatory changes may be achieved with a topical steroid (triamcinolone in the form of Genesis spray or a betamethasone preparation).

CLAW DISEASES

Diseases that specifically target the claws are uncommon to rare in the dog. Those diseases that involve the claw folds (paronychia – meaning inflammation of the claw fold) are quite common and are usually an extension from a more generalized pododermatitis. Most of these diseases do not significantly affect claw plate morphology. Diseases that affect one claw or multiple claws on one foot are defined as asymmetric and tend to be traumatic, due to infections (bacterial or fungal) or neoplasia. Those that involve several or all claws on all feet are defined as symmetric and are usually associated with systemic disease, infection secondary to systemic disease, autoimmune or immune mediated disease, nutritional or congenital diseases.

The major clinical manifestations of claw diseases include onychodystrophy/ onychodysplasia (abnormal claw formation), onychogryphosis (hypertrophy and abnormal curvature of claws), onychomalacia (softening of claw plates), onychorrhaxis (friable, brittle claws) and/or onychomadesis (soughing of the claw plate; separation of the claw plate from the dermis).

Damage to the claw matrix (that area of the claw plate that extends under the crescent shaped dorsal ungula process at the proximal end of P3) will result in abnormal rates of growth and/or the production of abnormal keratin which is clinically manifest as softer, drier, more friable claw plate. Abnormal curvatures may be produced by asymmetric growth patterns.

Permanent damage to the matrix will result in permanent morphologic changes. Inflammatory diseases that involve the dermo-epidermal junction (junction of claw plate and underlying dermis) may weaken this junction and be manifest as sloughing of the claw plate (onychomadesis). Chronic, deep seated inflammation of the nail fold and adjacent matrix (e.g. demodex, hookworm pododermatitis, leishmaniasis, neoplasia) may stimulate excessive matrix activity and claw growth, producing overgrown, larger claws that often have abnormal curvatures (onychogryphosis). This may also be seen as a senile change.

Fungal Infection (Dermatophytosis)

Unlike in humans, where fungal infection of the nail is relatively common, fungal infections targeting the claws of dogs is very rare. When noted, dermatophytosis is usually an extension from adjacent haired skin. Affected claws may be dystrophic, friable, and malacic. Onychomadesis is rare. Diagnosis is by fungal culture (shavings of the claw plate or pieces of claw plate). Systemic anti-fungal therapy is warranted (e.g. ketoconazole, fluconazole, terbinafine or itraconazole) and is long term (until good evidence of claw plate regrowth is noted). This can be supplemented by topical azole therapy (daily or BID).

Bacterial Infection

Bacterial infections of the claw are usually secondary to underlying local or systemic disease. Trauma (fracture, excessively short nail clipping etc) affecting only one or a few claws is most common. Osteomyelitis may develop in chronically affected claws. Although *Staphylococcus pseudintermedius* is most commonly incriminated, gram negative bacterial may also be contributory. Diagnosis is based on cytology (culture for refractory cases). Therapy for traumatized claws with secondary infections include removal of partially avulsed or broken nail plate. Topical anti-microbial therapy may be sufficient to resolve the problem: dilute bleach (5 – 15 ml/quart of water), dilute chlorhexidine (25 ml 2% chlorhexidine/pint water) or chlorexidine and an astringent (25 ml chlorhexidine / pint of aluminum acetate as Domeboro® or Bluboro® or in Epsome salts). The affected area is soaked for 5 minutes BID.

Symmetric Lupoid Onychodystrophy (SLO)

SLO is the most commonly encountered disease causing symmetric onychomadesis in dogs. Initially there is an acute loss of the claw plate from one or two toes. Within 2 -12 weeks, all toes are involved. Claw folds are typically not involved with the acute

presentation. Pain and lameness are variable. Secondary bacterial infections, often associated with the development of paronychia, are relatively common and suggested by serohemorrhagic or purulent exudation. This contributes to the development of pruritus and pain. Following acute claw plate loss there is claw regrowth, but regrowth is often abnormal. Claw plates are short, soft, brittle, crumbling and may be misshapen. If presented in the more chronic stages of the disease, the reason for presentation may only be claw dystrophy. The natural course of the disease is to periodically slough regrown, dystrophic claw plate material. Spontaneous remissions are possible.

The histologic changes associated with this disease are lupus-like (hence the name SLO). Characteristic histologic changes are most consistently noted in the claw matrix and dermo-epidermal junction of the dorsal ridge of the claw. SLO is thought to be an idiopathic, immune mediated disease in most individuals. This pathogenesis has been supported by studies done in Norwegian Gordon setters, Giant Schnauzers and Bearded Collies. A genetic predisposition has been noted in Norwegian Gordon Setters. This clinical presentation and histopathology has also been associated with other disease processes, including food sensitivity and bacterial infection- but rarely. The histologic pattern is therefore more aptly considered a reaction pattern, associated with multiple underlying causes. The incidence of hypothyroidism in dogs with immune mediated SLO appears to be increased (17% in one study). It does not appear that hypothyroidism is part of the pathogenesis. There may be a genetic predilection for the development of concurrent diseases. In one study, the majority of dogs experienced their onset of disease during the summer, raising the question of a possible link to atopy. There have been anecdotal reports of “flares” of SLO in association with “flares” of atopy, with affected individuals having their SLO remain in remission with successful immunotherapy. It has also been suggested that the mechanical trauma of claw contact with the ground may predispose to or perpetuate the problem. This observation is supported by the fact that, in some individuals, keeping nails trimmed back may significantly improve the problem.

Breeds predisposed include the German Shepherd dog, Giant, standard and miniature schnauzer, Rottweiler, greyhound, Bearded Collie and Norwegian Gordon and English Setter. The disease has also been seen in a number of other breeds. Age at onset is from 6 months to 11 years (mean and median 4-5 years).

The diagnosis of SLO is made on the basis of history, physical examination, claw biopsy (P3 amputation) and rule out. Food sensitivity has been associated with this presentation (2/24 cases in one study). Because this association appears to be rare, many do not routinely

initiate restrictive diet trials to rule out this possibility, saving this diagnostic for more refractory cases. Because SLO is the overwhelmingly most common cause of symmetric onychomadesis seen in the dog and because several therapeutic alternatives for this disease are relatively innocuous, therapy for this disease is often instituted without histologic confirmation.

Therapy

1. Recognition (cytology) and treatment of secondary bacterial infections; often significantly reduces pain and pruritus.
2. Removal of loosened claw plates – reduced pain; more rapid response to medications.
3. Keep the nails trimmed back to prevent contact with the ground.
4. Fatty acids – variably effective (author does not find them an effective monotherapy, but will use them as an adjunctive treatment; combined omega 3 and 6 products may be more effective (bottle dosages); consider omega 3s, 60 – 70 mg/kg/day of combined EFA and DHA; improvement noted within 3-4 months, maximal response 8 – 12 months.
Successfully treated individuals may still have some degree of dystrophic nails
5. Pentoxifylline is noted to benefit 50 – 60% of cases; 15 – 25 mg/kg BID; 3 month trial; for patients experiencing normal regrowth, after 6 – 9 months of therapy, consider stopping medication (looking for spontaneous remission). For recurrent disease, use long term maintenance therapy.
6. Tetracycline (500 mg/dog ; < 10 kg body weight – 250 mg/dog; TID) or doxycycline (5 – 10 mg/kg BID) and niacinamide (dose as for tetracycline); trial period 3 months; once significant regrowth noted (4-6 months), can decrease the frequency of administration of both drugs to once daily. If good response is maintained for 4-6 months on once per day therapy, treatment is stopped. Recurrence of signs would warrant the re-institution of the above regimen and indefinite maintenance therapy (usually once daily therapy; occasionally twice daily). About 30 – 40% of responders will regrow normal claws; others will regrow claws that are dystrophic (short, friable, misshapen).
7. Glucocorticoids are effective; prednisone/prednisolone starting at 1-2 mg/kg/day; often used at the initiation of therapy (e.g. first 3 or 4 weeks), along with other drugs such as fatty acids or pentoxifylline or doxycycline/niacinamide – all of which have a slower onset of benefit (especially if partial). It has been suggested that this more aggressive therapy, early in the disease may improve the overall prognosis. Others only use

glucocorticoids in patients that have failed other better tolerated therapies such as doxycycline/niacinamide or pentoxifylline.

8. The combination of a glucocorticoid and azathioprine (standard dosage regimens) have been used for more conservative treatment failures and to reduce glucocorticoid dosages for longer term, maintenance therapies.
9. Cyclosporine therapy, starting at 5 mg/kg/day has been noted to benefit the problem.
10. Refractory cases or patients who are intolerant of the above therapies can be managed with 20 nail, P3 amputations. Individuals generally do well without claws.

It has been suggested that SLO may be a “hit and run” disease associated with an acute immunologic attack that subsequently “burns out”. The growth of abnormal nails is then due to permanent injury /loss of germinal epithelium rather than ongoing disease. Using this rational, it has therefore been suggested that more aggressive therapy be instituted at the onset of disease (e.g. glucocorticoids and cyclosporine for small dogs, glucocorticoids and azathioprine for large dogs) and that this has produced a higher percentage of normal claw regrowth and the ability to stop all medications within 4 – 6 months, with prolonged remissions.

Prognosis: Cases of spontaneous remission and regrowth of normal claw plates or regrowth of dystrophic claw plates, but without the tendency to slough, have been noted (with no therapy). With successful therapy, the majority of patients regrow dystrophic claw plates that are often short (may not need to be clipped). A smaller percentage of individuals may regrow normal claw plates. Medications can be discontinued in some, with subsequent spontaneous remissions (claw plate normal or dystrophic; no further onychomadesis). Irregardless of the therapy employed, patients with excellent responses, characterized by the re-growth of normal claw plate, should have their medication discontinued at some point. These patients appear to have a greater incidence of being able to stop all medications and remain in remission. Others may require long term, indefinite therapy to maximize normalcy of claw plates and to prevent the recurrence of onychomadesis.

Other causes of canine onychomadesis: Food sensitivity (see previous). Vaccine reactions - although suggested by some, have also not been recognized in other studies. Several diseases associated with the presence of secondary bacterial paronychia (and usually more diffuse pododermatitis) may produce sufficiently deep and severe inflammation to result in

onychomadesis. These include demodicosis, or immunocompromising diseases such as hypothyroidism or hyperadrenocorticism. Other diseases may weaken the dermo-epidermal junction, including vasculitis/vasculopathy, idiopathic ischemic dermatitis, leishmaniosis, pemphigus vulgaris, pemphigus foliaceus, systemic lupus erythematosus, bullous pemphigoid, congenital epidermolysis bullosa, erythema multiforme, TEN, and drug eruption. In the vast majority of these cases, there will be other dermatologic changes to suggest these diseases.

Other diseases associated with onychomalacia/onychorrhaxis: primary seborrhea (most commonly reported in cocker spaniels; more generalized signs of seborrhea present); ichthyosis; idiopathic nasodigital hyperkeratosis; senile change in old dogs; Idiopathic (symmetric; seen in young dogs, 2-6 years, more common in dachshund, Siberian husky, Rhodesian ridgeback, German shepherd dog); Acrodermatitis in bull terriers ; Zinc responsive dermatitis (reported in two young malamutes with paronychia and onychorrhaxis of all digits; histopathology suggestive of this diagnosis; responded to zinc sulfate therapy); linear epidermal nevus (present since birth; extended from the groin to two digits of the paw)

Therapies for onychodystrophies due to primary keratinizing disorders or those that are idiopathic include: frequent nail clipping and filing; painting the claw plates with nail glue (to prevent fragmenting and “catching” on materials); nail caps (Soft Paws®). Although the use of biotin (5 mg/kg/day) or gelatin (10 grains every 12 hours / 10kg body weight; one packet of Knox gelatin per 7 kg q 24 hrs), there is no data to substantiate their benefit.

Neoplasia: may cause onychogryphosis (large claw) or onychomadesis /onychomalacia/ onychorrhaxis. Squamous cell carcinoma may involve multiple claws over 2-4 years in genetically predisposed breeds: large breeds with black coats; Labrador retriever, Standard Poodle.

FELINE PODODERMATITIS

Feline Paronychia

The list of differential diagnoses to consider for paronychia (nail fold inflammation) in the cat is pemphigus foliaceus, dermatophytosis, bacterial pyoderma, Malassezia and metastatic bronchogenic carcinoma. Cytology should be performed on all exudates from nail fold inflammatory diseases. The cytology that is suggestive of pemphigus consists of large numbers of neutrophils, variable numbers of eosinophils and large numbers of acantholytic keratinocytes. Bacteria are usually not present but if they are present, they are secondary. In

one study, paronychia was the first site to be affected with the development of pemphigus foliaceus in the cat.

FELINE “PADODERMATITIS”

Pemphigus foliaceus

In cats, the initial and most commonly affected areas of the body are the head/face, ears, paws (frequently the pads), nail folds, dorsum and ventrum, legs, chin and tail (in decreasing order of incidence). In another study initial sites of involvement included paronychia (78%), feet (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. The diagnosis is suggested by cytology (acantholytic keratinocytes, neutrophils with variable numbers of eosinophils) and confirmed by biopsy. Therapies have included prednisolone, triamcinolone (in one report, suggested to produce the most consistent responses with the least side effects compared to prednisone), dexamethasone, prednisolone and chlorambucil and gold salts. Achieving a good response to therapy and maintenance of remission with lesser side effects appears to be more common than in the dog.

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 sensitivity), the few cases the author has seen appear to have been idiopathic. Diagnosis is by skin biopsy. Response to glucocorticoid therapy has generally been good (prednisolone - 1.1 - 2.2 mg/kg/day). Spontaneous resolution may be noted.

Feline Plasma Cell Pododermatitis

Most cases are thought to represent an immune mediated disease. There may be some seasonal waxing and waning. In European studies, 45% - 60% of cases were FIV positive. We do not appear to see this association in the USA. There has been a recent suggestion that bartonella may play a role. The earliest change seen is usually the development of a white, scaly, cross hatch pattern and swelling of the pads. Swelling may be dramatic. With chronicity, the swollen pad may ‘deflate’ (appearing almost like a deflated balloon). Occasionally, the pads may go on to erode or ulcerate. There may be significant hemorrhage. In one study of 26 affected cats, the relative incidence of these changes included swelling (35 footpads), softening (36 footpads), exfoliation (19 footpads), ulcers (nine footpads) and

abscesses (nine footpads). These clinical changes vary from being asymptomatic to involving significant pain and lameness (lameness in 22/26 cats in the above study). Systemic signs are also variable (pyrexia, lethargy, anorexia, peripheral lymphadenopathy). General laboratory screening usually reveals a hyperglobulinemia. In one retrospective study of 10 cats, thrombocytopenia was noted in 70%, a leucocytosis in 40% and lymphopenia in 30%. Pad biopsies show a perivascular accumulation of plasma cells, with lesser numbers of lymphocytes and neutrophils. Leukocytoclastic vasculitis has been described. Early lesions may involve significant infiltration with eosinophils. Some cases will spontaneously resolve with no therapy. The initial therapy of choice for the author is doxycycline (10 mg/kg/day; we use 5 mg/kg BID). In one retrospective study looking at 10 affected cats, one cat went in to remission within 30 days, 4 cats in 60 days and 4 cats improved more than 50% within 60 days. Only one cat failed to respond. Treated patients may be eventually weaned off therapy. Therapies for 'doxycycline failures' include glucocorticoids (prednisolone, triamcinolone or dexamethasone), oral cyclosporine (5.0 - 7.5 mg/kg/day; authors alternative of choice), glucocorticoids and chlorambucil or surgical excision (usually to ameliorate hemorrhage problems). Again, successfully treated patients may be able to eventually weaned off all medications.