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Challenging Dermatology Presentations Mini Series

Session 1: The Dog with Sore Paws

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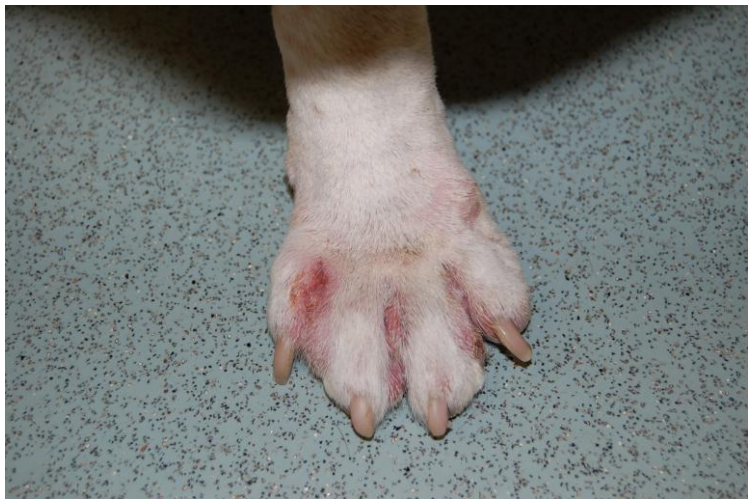

Introduction

Canine pododermatitis is a common clinical presentation in small animal practice. It usually presents as either paw licking or lameness with variable additional, non-specific clinical signs. There are many causes for pododermatitis and a careful systematic approach and definitive diagnosis are essential as it can be a cause of significant suffering for the patient if misdiagnosed and poorly managed. This lecture will review the more common causes of pododermatitis and outline the clinical approach to be taken.

Approach to pododermatitis

Unless a diagnosis is immediately obvious, cases of pododermatitis require a methodical, systematic approach. This should include detailed history taking including age of onset, seasonality, presence or absence of signs suggestive of systemic involvement, and whether other areas of the integument are involved. Aspects of management should be explored. For example, if the dog is kenneled, are there rough surfaces, are strong disinfectants used to clean floors, are conditions potentially unsanitary?

A full physical examination should be performed along with careful examination of not just the paws but also the entire integument. The history combined with physical examination should allow the clinician to compile the differential diagnosis.

Diagnostic tests

Minimum baseline diagnostic tests would include hair plucks, skin scrapes and trichograms to look for evidence of demodicosis and cytology to determine whether bacterial and Malassezia infections are involved. Culture and sensitivity testing would be indicated in cases of deep pyoderma. Histopathological examination is indicated in cases of suspected metabolic and auto-immune disease, and neoplasia and may also be useful to identify the presence of plantar hair follicle cysts[10]. The diagnosis of atopic dermatitis is made on the combination of history, clinical signs and ruling out other pruritic skin diseases but diet trials and intradermal / serological testing would be indicated to identify putative causative allergens for avoidance and/or allergen immunotherapy.

Causes of pododermatitis

A wide range of conditions can result in pododermatitis. Disease may be confined to the paws, it may be part of a generalised skin condition or pododermatitis may be a manifestation of a systemic disease. Then there are conditions such as demodicosis and atopic dermatitis that most commonly present as generalised skin diseases but occasionally present with disease confined to the paws, which can catch out the unwary. Diseases may be further subdivided into those conditions that tend to affect the haired skin versus those that affect the footpads. Parasitic diseases, infections, allergies, autoimmune conditions, congenital disorders, and neoplasia can all result in pedal disease. Table 1 lists the main conditions involved. It is beyond the scope of this lecture to review every disease in detail.

Demodicosis

Demodicosis caused by *Demodex canis* frequently results in pedal involvement and is most commonly a generalised multifocal disease characterised by patchy alopecia, comedones, scaling, erythema, hyperpigmentation and secondary pyoderma. It is classified as localised or generalised, and juvenile or adult onset. Pododemodicosis is a recognised clinical entity[1]. The paws alone may be involved or there may be more generalised disease. On occasion, pododemodicosis may be a sequelae to generalised demodicosis. The history should determine whether the dog has previously suffered from generalised demodectic mange or whether the paws alone have been affected. Typically, these cases will present with a history of foot licking and in more severe cases pain and reluctance to walk, especially on hard ground. Regional lymphadenopathy may be marked and patients may be systemically unwell. Alopecia, comedones, swelling, erythema, crusting and scaling and secondary deep pyoderma are all common presenting signs. This condition may be misdiagnosed as atopic dermatitis due to the presence of pedal pruritus. Demodicosis should always be the first rule

out in any case of pododermatitis involving haired skin. In adult onset cases, a diagnosis of pododemodicosis should prompt a hunt for underlying immunosuppressive disease. Pododemodicosis is recognised to be more refractory to therapy compared to disease affecting other areas of the integument and in some cases it can be difficult to achieve negative skin scrapes. In the UK, there are two licensed treatments for demodicosis: topical moxidectin and imidacloprid (Advocate® spot on) and weekly amitraz dips (ALudex®) used at a concentration of 500ppm. Experience has shown that even when used weekly, Advocate® does not appear to be an effective treatment in cases of severe, generalised demodicosis and especially in pododemodicosis. The author's standard (unlicensed) treatment for pododemodicosis is to use weekly amitraz dips at 500ppm and mid way between each dip the paws are painted with a mix of undiluted amitraz and propylene at a ratio of 1:10. Treatment is continued until two negative sets of scrapings are obtained at an interval of a month. It is difficult to skin scrape the paws and hair plucks may be used to monitor therapy but skin scrapings are more sensitive than hair plucks for the detection of mites and should be used when the dog is nearing remission[2]. The owner should be warned it may require months of treatment to reach this point and in some cases it is difficult to achieve a parasitological cure. Failure to respond to amitraz would be an indication to treat with daily oral ivermectin and the reader is referred to publications on the use of this drug in dogs[3].

Malassezia dermatitis[4]

Malassezia dermatitis is a common cause of pedal pruritus and pododermatitis. As already mentioned it is usually secondary to underlying atopic dermatitis but in some breeds such as the Bassett hound and cocker spaniel it can apparently present as a primary disease. In atopic dogs it may be a major contributor to pruritus and management of the yeast infection may reduce pruritus to an acceptable level.

Typically skin folds are affected and disease may be generalised or limited to particular body sites including the paws. Clinical signs consist of erythema, brown greasy exudation, scaling and alopecia over the dorsal interdigital webs and between the footpads. Brown staining over the proximal nails is often recognised in *Malassezia* infections affecting the nail folds.
There are a number of topical therapies available for the treatment of Malassezia dermatitis including 2% miconazole and 2% chlorhexidine shampoos (Malaseb®; Dechra) and a 3% chlorhexidine shampoo (Microbex®; Virbac). Efficacy against Malassezia has been demonstrated by in vitro studies[5] and in the case of 2% chlorhexidine and 2% miconazole by *in vivo* studies as well[6,7] Cases should be shampooed at least twice weekly and the owner should be instructed to work the shampoo in well between the toes and footpads when the feet are being treated. Acetic acid and boric acid or climbazole and chlorhexidine wipes are very useful for the longer term management of Malassezia pododermatitis.
On occasion, topical therapy may be ineffective on the owner may be unwilling or unable to do the treatment. Systemic therapy would be indicated and the author's current treatment of choice is itraconazole at a dosage of 5mg/kg sid for up to three weeks. Unfortunately, ketoconazole is no longer available in the UK.

Pyoderma

Bacterial pododermatitis is also a common clinical presentation with variable signs depending on the depth of infection. More superficial infections affecting the interdigital webs can be a cause of interdigital erythema and pruritus. These cases do not present with pustules, papules and collarettes but are analogous to bacterial overgrowth with more diffuse erythema and greasy exudation. Deep pyoderma on the other hand most commonly affects the footpad margins and results in lameness, draining tracts and crusting. Typically, there is a sanguinous purulent exudate from these lesions. Deep pyoderma is also likely to be involved in draining tracts affecting the dorsal interdigital webs, so called "interdigital cysts". These will be discussed in more detail later.

Pyoderma is a secondary disease and is always recurrent unless the underlying cause can be identified and corrected.

Atopic dermatitis

Canine atopic dermatitis frequently presents with pedal pruritus. In white haired dogs saliva staining may be the presenting sign. This is a complex pruritic and inflammatory skin disorder of young dogs with well-recognised breed predilections. It is beyond the scope of this lecture to review this disease in detail but a detailed history is essential to make this diagnosis. Atopic dermatitis starts in dogs between 6 months and 3 years of age. Initial clinical signs are of non-lesional pruritus, typically affecting the face, ears, paws, limbs, ventrum and perineum although pruritus may be confined to one body area such as the paws. The pruritus is usually glucocorticoid responsive. It is common for cases to develop secondary pyoderma and *Malassezia* dermatitis.

In the early stages, pruritus may be non-lesional, or there may be saliva staining and interdigital erythema. As disease progresses self trauma can lead to significant alopecia which needs to be differentiated from that caused by demodicosis. More chronic lesions may present with significant soft tissue swelling, excoriation and secondary deep pyoderma.

"Idiopathic" pododermatitis

There are cases of pododermatitis where despite extensive work up, a definitive underlying cause cannot be identified. These cases have been classified as having idiopathic pododermatitis[8]. There are also cases where there may for example be indicators that the dog has underlying atopic dermatitis but despite aggressive treatment for this condition, the pododermatitis persists. All four feet may be affected or disease may be confined to the forefeet. Disease is often chronic and severe. Poor foot conformation, underlying immunosuppressive disease, and bacterial infections with furunculosis and granuloma formation probably all contribute to disease in these cases. It has been suggested that poor foot conformation leads to weight bearing on haired skin and results in in-driven hair shafts, bacterial folliculitis and furunculosis, release of hair fragments into the dermis and resultant granuloma formation. A form of lymphoplasmacytic pododermatitis that responded to immunomodulatory therapy has been reported although the author's conceded that there was insufficient evidence to make this a defined clinical entity-it is more likely to be a reaction pattern[9].

Frequently, these cases present with interdigital draining sinus tracts and repeated interdigital abscessation. These are commonly referred to as "interdigital cysts" but these are inflammatory, granulomatous lesions not cystic. However, a subtype of this group of cases has been reported associated with the formation of comedones and cysts over the *plantar* interdigital skin which periodically rupture and lead to repeated episodes of pyogranulomatous dermatitis and draining tracts onto the dorsal interdigital web[10]. Clinically, these cases present with an area of alopecia, thickening and comedone formation over the plantar interdigital skin with an overlying interdigital nodule or tract.

This can be one of the most challenging conditions in dermatology. Dogs presenting with chronic pododermatitis particularly those with repeated episodes of abscessation and draining interdigital tracts should be carefully evaluated as already described. Prolonged antibacterial therapy is indicated based on the results of cytology, and bacterial culture and sensitivity testing. Clearly, every attempt should be made to address any potential underlying diseases. Whilst antibacterial therapy may produce an initial improvement it is common for lesions to recur whilst the dog is receiving treatment presumably due to the presence of sterile pyogranulomatous disease. Some of these cases may respond to immunomodulatory therapy using systemic or topical glucocorticoids, ciclosporin or tacrolimus.

A surgical technique has been described to treat dogs presenting with interdigital lesions due to palmar/plantar interdigital comedo and follicular cyst syndrome. CO2 laser was used to ablate the cystic lesions and resulted in a cure in 25/27 dogs presenting with this syndrome[10]. In cases where the CO2 laser technique is not appropriate or may not be available and where medical management has not been successful, the only remaining option would be a partial or complete fusion podoplasty to restore the dog's quality of life.

Pemphigus foliaceus

Pemphigus foliaceus is an uncommon, sterile, auto-immune, pustular skin disease resulting from the production of autoantibodies targeting desmosomal proteins. This results in the process of acantholysis within the stratum spinosum, or separation of keratinocytes forming clefts within the epidermis. Exocytosis of neutrophils or eosinophils results in formation of sub corneal pustules, which rupture leaving extensive crusting overlying shallow erosions. The nasal planum, face, pinnae and footpads may all be affected although lesions may be more generalised. The disease typically affects middle aged to older dogs but it can start at any age. The Japanese Akita, Chow Chow, cocker spaniel, Labrador retriever and Dachshund appear to be over represented[11]. The disease may be localised or generalised and can affect the footpads. Typically footpad involvement results in erosions, crusting and hyperkeratosis. Occasionally disease is confined to the footpads[12]. The clinical appearance of this disease is fairly characteristic but differential diagnoses include superficial necrolytic dermatitis, zinc responsive dermatosis, dermatophytosis and cutaneous lymphoma.

Other auto-immune diseases[13]

A number of other autoimmune diseases can cause footpad ulceration which may be focal or involve the entire footpad epidermis. Diseases include pemphigus vulgaris, lupus erythematosus, erythema multiforme, toxic epidermal necrolysis, mucous membrane pemphigoid, epidermolysis bullosa, and vasculitis. These are complex and challenging cases. They may be secondary to underlying systemic disease or drug eruptions and severe presentations may be potentially life threatening.

The treatment of this group of diseases would fill a book chapter if not a whole book but the mainstay of therapy is likely to be glucocorticoid therapy which may be administered topically or may be required to be administered systemically in immunosuppressive dosages. In addition, there are numerous other therapies that may be used as glucocorticoid sparing agents.

Necrolytic migratory erythema (hepatocutaneous syndrome, superficial necrolytic dermatitis, metabolic epidermal necrosis)[14]

Superficial necrolytic dermatitis is a necrotising skin condition of dogs associated with internal disease, most commonly a hepatopathy but glucagonoma, feeding mycotozin contaminated foodstuffs and anticonvulsant therapy have been reported as causes.

The disease results in keratinocyte degeneration and necrosis. The specific cause is unclear but hypoaminoacidaemia is a frequent finding and it is hypothesized that the keratinocytes damage results from cellular starvation or nutritional imbalance.

This is a disease of older dogs that present with progressive skin lesions of 3 weeks to many months duration. Lesions include erosions, ulcerations, erythema, alopecia, crusting and scaling over the muzzle, periocular skin, elbows, hocks, ventrum, scrotum and footpads. The footpads are commonly affecting with fissuring and exudation resulting in unwillingness to walk, particularly on hard ground. Lethargy, inappetance, weight loss and signs of diabetes mellitus with polyuria and polydipsia are frequently seen.
NME carries a very guarded prognosis. Most dogs are eventually euthanased particularly if they have severe pedal lesions.

Surgical removal or debulking would be indicated if the underlying cause is a glucagonoma but hepatic metastasis seem to hepatic metastases occur readily.

Palliative therapy can improve quality of life. With dedicated nursing care and nutritional support, life expectancy can be extended but the majority of cases have died or are euthanased within five months of diagnosis. Nutritional supplementation consisting of egg yolk, zinc and fatty acid supplementation can he helpful. Intravenous amino acid infusions can produce a remarkable although generally short lived improvement.

Zinc responsive dermatosis

Two syndromes have been described

Syndrome 1

Siberian huskies, Malamutes and other Nordic breeds fed well balanced diets.
Familial therefore genetic problem leading to poor zinc absorption?

Syndrome 2

Usually young growing dogs fed high cereal diets –phytates in cereals bind zinc. Uncommon nowadays with improved quality of diet.

Clinical signs

Clinical signs in both cases result in adherent scale and sometimes crusting and erythema over the peri-orbital skin, nasal planum, elbows, hocks and footpads. Pruritus may be evidence and secondary bacterial infection is a potential complication. Histopathological examination shows a superficial perivascular dermatitis with marked, confluent parakeratotic hyperkeratosis. Measurement of serum zinc concentrations can be done but problems with zinc contamination from rubber stoppers / tubes and difficulties with interpretation of results mean that in practice this is usually not done.

Affected (syndrome 1) Nordic breeds are likely to require lifelong zinc supplementation with either zinc sulphate at 10mg/kg q 24 or zinc methionine 1.7mg/kg q24. A good quality diet high in essential fatty acids is also important. Most dogs respond to this treatment but occasionally oral Zn supplementation is not effective and anecdotally, concurrent administration of anti-inflammatory doses of prednisolone may aid zinc absorption in refractory cases.

Dogs affected by syndrome 2 respond well to dietary improvement and short term zinc supplementation.

Congenital footpad hyperkeratosis and fissuring

Congenital disorders of keratinisation are recognised in several breeds that can lead to footpad hyperkeratosis and fissuring.

Congenital footpad hyperkeratosis is recognised in the Dogue De Bordeaux[15], and Irish[16] and Kerry Blue terriers. There may be concurrent involvement of the nasal planum. A congenital nasal parakeratosis has been reported in the Labrador retriever[17] which results in thick hyperkeratosis of the dorsal aspect of the nasal planum but in severe cases, the footpads may also be involved.

These conditions are genodermatoses and so tend to present in young dogs. Fissuring and secondary infection leads to pain on walking and reluctance to exercise on hard ground. There is no specific treatment for this group of conditions. These genodermatoses require lifelong care. Soaking of the footpads in water softens the hard keratin and facilitates gentle trimming to remove excess keratin. Emollients and moisturisers such as petroleum jelly and propylene glycol may help to keep the keratin soft. Keratolytic preparations such as salicylic acid creams may help to reduce excess keratin. Topical tacrolimus has been reported to be beneficial for the management of hereditary nasal parakeratosis[18].

Corns are a focal hyperkeratotic lesion that may be treated by gentle elevation and removal of the hard keratin.

Feline plasma cell pododermatitis

Aetiopathogenisis

- Cause unknown but suspected immune mediated disease because of hypergammaglobulinaemia / tissue plasmacytosis and response to immune modulating drugs. .
- Sometimes seasonal so may be a hypersensitivity component.
- 50% cats with PCP are FIV+ve.

Clinical signs

- No age, breed or sex predilections
- Initially soft swelling of central carpal or tarsal pads. Footpads may develop a violaceous hue with white coloured striae.
- May ulcerate and cases may present as footpad haemorrhage.
- May develop a massive granulating response.
- May occasionally present with a stomatitis.

Diagnosis

- History, clinical signs
- Cytology-numerous plasma cells
- May be FIV +ve.
- Histopathology

Treatment

- Treatment of choice is doxycycline at a dosage of 10mg/kg sid.
- Prednisolone at 2-4mg/kg may also be effective.
- May require surgery to excise granulating tissue and resolve haemorrhage.

Acral mutilation syndrome

This is a rare genodermatosis reported in German short hair pointers, English pointers and English springer spaniels. The disease presents in dogs a few months old that lick and chew aggressively at their paws, particularly the hind feet and cause severe self trauma. Eventually, this can result in auto-amputation of the paws. The cause is thought to be due to a sensory neuropathy and abnormalities have been demonstrated in the spinal cord, spinal roots, ganglia and peripheral nerves. This is not the only cause of severe self trauma as the author has seen cases that have been referred with suspected acral mutilation syndrome but in fact responded to treatment for atopic dermatitis.

Nails diseases

Clinical presentations involving the claws

There are a number cutaneous and systemic diseases that may result in claw and claw fold involvement in both dogs and cats. Claw disease as a sole entity is relatively uncommon with a reported incidence of only 1-2% of referral dermatology cases.

The following terms are used to describe various clinical presentations of claw disease.

Anonychia	absence of claws
Onychodystrophy	malformation of the claw
Onychomadesis	complete loss of claw
Onycholysis	breakdown of claw matrix
Onychomycosis	fungal infection of claw
Onychorrhexis	brittleness, splitting of claw
Onychoschizia	longitudinal splitting of the claw from bed
Paronychia	inflammation of the folds surrounding the claw

The main presentations encountered in practice are paronychia, onychomadesis and onychorrhexis.

Diagnostic approach to paronychia

Inflammation of the nail folds is a fairly common clinical presentation. One or more nail folds may be affected with swelling, erythema, scaling, crusting and purulent exudation. There may be evidence of more generalized skin or systemic involvement.

Causes of paronychia

Self trauma

Pruritic skin diseases, particularly atopic dermatitis, can lead to foot licking and chewing with resulting trauma to the extremities of the digits. There may be secondary bacterial or yeast infection. The investigation of a primary pruritic skin disease is indicated in these cases.

Bacterial infection or Malassezia *dermatitis*

Bacterial and yeast infections are commonly involved in paronychia. Cytological examination of discharge from nail folds will indicate the presence of infection. Infection should be treated appropriately. If there is complete and permanent resolution then no further investigation is required. However, as bacterial and yeast infections tend to be secondary diseases, if there is only partial resolution, or resolution followed by relapse, then further investigation would be required to investigate the underlying cause.

Demodicosis and dermatophytosis

Pododemodicosis and dermatophytosis can both result in a variably erythematous, alopecic, swollen and crusting paronychia. Skin scrapes and hair plucks are mandatory in the investigation of this disease presentation. Fungal culture would be indicated if there is reason to suspect dermatophyte involvement.

Pemphigus foliaceus

Paronychia with a caseous purulent discharge is a frequent finding in cats with pemphigus foliaceus. There will usually be evidence of more generalized pustular skin disease (see notes on pustular diseases). Cytological examination of the discharge from the nail folds should reveal the presence of non-degenerate neutrophils and acantholytic keratinocytes. Histopathological examination is necessary to confirm the diagnosis.

Metastatic bronchial carcinoma

There is a rare syndrome of digital metastasis of bronchial neoplasia in cats resulting in an ulcerative and crusting paronychia. These cats would present as cachectic and with respiratory involvement.

Onychomadesis

Mechanical trauma

Loss of one or perhaps more than one nail may result from trauma, particularly in young active dogs. The aetiology is usually clear from the history and clinical signs. Wound management treatment is appropriate. Partially avulsed claws should be removed.

Symmetrical lupoid onychodystrophy (SLO)

This is a presumed auto-immune disease although aetiology is not clear. Affects any breed but German shepherds appear to be predisposed. Initially there is loss of one nail followed by loss of most of the nails over the following weeks. Pain, swelling and discharge accompany the nail loss. No systemic involvement and no other skin lesions. Claws regrow but tend to be soft, crumbly and dystrophic.

Cytological examination of discharge usually reveals secondary bacterial infection. Some dermatologists are happy to make the diagnosis on the basis of history and clinical signs alone. Histopathological examination of an amputated digit confirms the diagnosis but poses problems. If a dew claw(s) is affected then this is amputated and submitted for histopathological examination. The option of referral to a dermatology specialist should be considered before amputation of a weight bearing digit to make the diagnosis. A technique for biopsy of the nails and nail matrix involves the use of an 8mm punch biopsy to “shave” a sample of claw with the underlying bone of the distal phalanx and through normal skin on the lateral aspect of the claw fold.

Treatment involves removal of any partially separated, painful, claw plates. Topical antibacterial/antifungal soaks and systemic antibacterial therapy are indicated when there is secondary bacterial infection.

Various anti-inflammatory and immunosuppressive therapies have been described for treatment of SLO.

Some cases respond to good nail care, treatment of secondary bacterial infection and high doses of essential fatty acids.

More aggressive treatment includes tetracycline and nicotinamide given at 250mg tid of each for dogs under 15kg and 500mg tid of each for dogs over 15kg.

Pentoxifylline has also been reposted to be of benefit at a dosage of 10-15mg/kg tid.
A technique of total onychectomy has been described in cases of nail disease which are refractory to treatment and result in continuous pain.

Other autoimmune diseases

A number of rare autoimmune diseases can result in onychomadesis including pemphigus vulgaris, epidermolysis bullosa, vasculitis, erythema multiforme and drug eruptions.

Summary
In short, canine pododermatitis is a complex multifactorial condition. A careful, systematic work up is required including detailed history taking, thorough examination and various diagnostic procedures. The first step in any case involving haired skin is to rule out the involvement of demodicosis. Cytology, cultures, histopathology, and blood work may all be indicated.
Treatment should be directed at specific disease causes. Some cases such as those presenting with recurrent Malassezia dermatitis may require ongoing treatment to prevent recurrence. In cases where a specific underlying cause cannot be identified, then infections should be thoroughly treated before considering the use of immunomodulatory therapy. Surgery may be required in cases that cannot be managed with medical therapy.

Infectious causes	**Parasitic**	**Allergies**	**Auto-immune disease**
Bacterial	Demodicosis	Atopic dermatitis	Pemphigus foliaceus
Malassezia dermatitis	Harvest mites	Adverse food reactions	Lupus erythematosus
Dermatophytosis	Pelodera	Contact dermatitis	Bullous pemphigoid
Deep mycoses	Hookworm dermatitis	Drug reactions	Epidermolysis bullosa
Leishmaniasis			Vasculitis
Metabolic	**Congenital / hereditary**	**Neoplastic**	
Superficial necrolytic dermatitis	Nasodigital hyperkeratosis	Melanoma	Corns
Zinc responsive dermatosis	Acral mutilation syndrome	Squamous cell carcinoma	
	Nodular dermatofibrosis	Cutaneous lymphoma	

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