WHEN IS PYODERMA NOT PYODERMA?

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Introduction

Staphylococcal pyoderma is the number one cause of folliculitis in the dog. Although reported less frequently, it is also a common cause of skin disease in the cat. Cats however manifest their pyoderma differently; as opposed to the normal folliculitis lesions noted in dogs (papules, pustules, crusts, epidermal collarettes), cats will manifest Staphylococcal pyoderma as one of the cutaneous reaction patterns (head, neck and pinnal pruritus; self induced alopecia; miliary dermatitis; eosinophilic skin lesions including granuloma/plaque/indolent ulcer) or as excessive scaling. However, it is generally a fair assumption that in a dog, folliculitis is most likely due to Staph infection; this of course can be confirmed with cytologic evaluation. What if however the presumed pyoderma is not pyoderma at all but rather something different? Or what if the bacterial infection confirmed by cytology is secondary to another underlying disease process? This is not uncommon as bacterial pyoderma can be a secondary complication of myriad dermatologic abnormalities. This discussion will focus on diseases that can mimic bacterial pyoderma and highlight differences in clinical appearance and distribution of lesions. Particularly, demodicosis, dermatophytosis, and pemphigus foliaceus will be discussed.

Demodicosis

Demodicosis is more of a problem in dogs than in cats, however both species can develop dermatologic abnormalities due to these external parasites.

Feline demodicosis

Cats can harbor two different species of Demodex mites: *Demodex cati* and *Demodex gatoi*. *Demodex cati* is the feline equivalent of *Demodex canis* in the dog. One of the main differences however is that there is no juvenile onset form of demodicosis in the cat; when *Demodex cati* is found, it is typically secondary to an underlying immunosuppressive or metabolic condition. Even in a young animal, systemic disease (e.g. FeLV, FIV, FIP) should be evaluated if *Demodex cati* are found on skin scraping. The disease typically presents similarly to canine demodicosis; multifocal patchy alopecia with varying pruritus, scaling, crusts, and/or erythema and hyperpigmentation will be present. The head, face, and neck are frequently affected, however lesions can be anywhere with generalized disease.

*Demodex gatoi* on the other hand is quite different; this is considered to be a contagious Demodex mite. The mite lives superficially in the skin and is typically associated with moderate to severe pruritus. Cats will frequently be reported to pull out large chunks of fur (self induced alopecia is the most common cutaneous reaction pattern associated with *Demodex gatoi* infestation) particularly along the ventrum, flanks, limbs, and/or head. This superficial parasite can be very difficult to find on skin scraping; in general, a treatment trial with lime sulfur is recommended when the condition is suspected.

Canine demodicosis

Demodicosis in the dog can be due to various mite species: *Demodex canis, Demodex cornei*, or *Demodex injai*. Most commonly, dogs with demodicosis are affected by *Demodex canis*. *Demodex cornei* is a superficial, short
bodied Demodex mite. It has not been reported to cause disease by itself; rather it is typically found in conjunction with Demodex canis infestation. Demodex injai is a bit different; this Demodex mite is VERY long (approximately twice the body length of Demodex canis) and resides in the hair follicle and sebaceous glands. It has been over-reported in wire-haired fox terriers and West Highland white terriers; these breeds may have a genetic predisposition towards the development of this condition. As opposed to more common demodicosis presentation (see below), dogs infested with Demodex injai will present with greasy seborrhea over the dorsum; pruritus appears to be variable but can be severe.

Demodicosis due to Demodex canis infestation comes in several flavors based on location as well as age of onset. With localized demodicosis, disease is limited to five or less lesions of patchy alopecia, affecting only one body region. Erythema, scaling and hyperpigmentation will variably be present. The face and head are often affected. This condition is seen only in young dogs (if “localized” demodicosis is diagnosed in an adult dog with consistent dermatological examination, it should be considered “generalized” demodicosis and treated as such; see below) and is generally self-limiting. Treatment is typically not necessary; I will often prescribe benzoyl peroxide shampoo for these cases for its “follicular flushing” activity and to give the owners something to do to “treat” the condition at home. In some patients with initially localized demodicosis, the disease will progress to the more generalized form.

Generalized demodicosis necessitates treatment. The disease will generally manifest as folliculitis with papules, pustules, crusts, epidermal collarettes and scaling present. Multifocal patchy asymmetrical alopecia is reported to involve more than five spots and more than one body region. When feet are involved, it is typically due to generalized demodicosis. Common areas affected include the face (particularly the periocular skin), head, limbs, and feet although the mites may be found diffusely. With the juvenile-onset form of demodicosis, this is a genetic condition associated with a defect in cell-mediated immunity; the body does not identify the Demodex mites as “not supposed to be there in large numbers and causing problems.” This does not however mean the dog is an “immunologic cripple” and will be destined to a life of illness. Still, breeding of these affected dogs is not recommended. Juvenile-onset demodicosis should be considered in animals less than 18 months of age (although technically this is variably based on when the growth plates close). In the adult onset form, the condition manifests later in life; the clinical appearance however is identical to the juvenile form. With adult onset demodicosis, the mites over-proliferate secondary to an underlying systemic illness. This can be due to any metabolic, endocrine, or neoplastic disease. In some cases, at the time adult-onset demodicosis is diagnosed an underlying systemic condition is not noted even with additional diagnostic work-up. In those cases, I typically warn owners of two things: 1) that the demodicosis may be more difficult to resolve if the underlying process is not known and therefore not effectively managed, and 2) that they should monitor that dog fairly closely; usually within about two years of Demodex diagnosis, something in the overall health of the animal will change.

There are several options for treatment of generalized demodicosis, however with all of them the same principles remain: goal for treatment is two to three consecutive negative skin scrapes, scraped from the same sites at each visit. Negative skin scraping means NO MITES of any life stage, alive or dead. On average, the time frame to achieve this is approximately 6-7 months with appropriate treatment. A fairly recent (and free!) article in Veterinary Dermatology reviewed all of the recommend treatments for demodicosis in dogs and should be referred to for practice guidelines (see references). This article however came out prior to the release of some of our newer veterinary products which have excellent activity and success against Demodex mites in dogs (e.g. the isoxazoline compounds – afoxalaner, flurolaner, sarolaner).
There are several reasons for why treatment for demodicosis fails, however one of the most common reasons is failure to address the secondary bacterial pyoderma. As Demodex mites live deep within the hair follicles, it is not uncommon to diagnose superficial and/or deep bacterial pyoderma secondary to demodicosis. This needs to be addressed and effectively treated in order for demodicosis to be resolved as well.

**Dermatophytosis**

Dermatophytosis is one of the most common infectious diseases that veterinarians will encounter in practice. It is a contagious disease with zoonotic implications. Feline dermatophytosis in particular is one of the most common dermatologic diseases in the cat; dermatophytosis should be considered a differential for most feline dermatologic conditions until proven otherwise. A complete history and physical exam are essential for establishing a diagnosis of feline dermatophytosis. Age of the animal as well as any concurrent diseases is helpful information; dermatophytosis is most commonly found in “at risk” populations including young kittens or cats with other immunosuppressive conditions (e.g. FeLV, FIV, and neoplasia). History should also include whether other people or animals in the house are affected by dermatologic abnormalities, whether the cat lives indoor/outdoor, and what type of environment the cat lives in; this final bit of information will be helpful for client education with regards to environmental decontamination. Complete physical examination of the haired skin including the inner and outer aspects of the pinnae, face, and distal extremities including all claw folds is necessary to assess the extent and severity of disease.

Dermatophytosis in cats has a highly variable appearance. While the classical clinical appearance is localized or multifocal, patchy, circular areas of alopecia with scaling along the margins, the disease can take on many pleomorphic forms. Most commonly however, cats will present with one or more circular or irregular areas of alopecia; commonly affected body areas include the face, pinnae, and distal extremities. Broken hairs, erythema, crusts, scales, and hyperpigmentation are frequent clinical signs. Inflammation and pruritus however is often variable. Cats, particularly long-haired breeds such as Persians, can frequently be asymptomatic carriers of dermatophyte spores; this is different from the disease in dogs in which a carrier state is considered rare. Less common clinical signs of dermatophytosis in cats include miliary dermatitis, chin folliculitis/furunculosis, widespread exfoliative erythroderma, and nodules and ulceration (kerion reactions or dermatophytic pseudomycetomas).

Most dermatophyte infections are caused by *Microsporum canis*, however other dermatophyte species can also be commonly isolated including *Microsporum gypseum* and *Trichophyton mentagrophytes*. Of these species, *M. canis* is the only one that typically fluoresces with Wood’s lamp evaluation. Although the information has been extrapolated from people, it is suspected that only about 50% of those *M. canis* strains exhibit fluorescent ability. Moral of the story: a negative Wood’s lamp evaluation does not rule out dermatophytosis. With a positive Wood’s lamp evaluation however, the hair shaft glows a bright apple-green to yellowish color. Since the fungus invades the hair shaft itself, it causes weakening and breakage of the hairs; it is not uncommon to see only these broken tips exhibit the fluorescence. Again however, it is the hair itself which glows green. Other things which will falsely fluoresce under a Wood’s lamp include scales and crusts (yellowish), synthetic fibers and dander (bluish), and some topical products or medications (e.g. soap, antibiotics). It is important not to misinterpret this type of glowing as true fluorescence. DTM fungal culture is one of the most effective ways to diagnose dermatophytosis. If a positive Wood’s lamp evaluation is obtained, preference is given to glowing hairs for sampling. These may be collected from edges of lesions using sterile forceps. This technique however can lead to false negative results if positive hairs are missed. For more diffuse or subclinical dermatophyte infections, the MacKenzie toothbrush technique is highly beneficial. The entire body is brushed for several minutes using a new
toothbrush; special attention should be paid to “high-risk” areas such as the face, pinnae, and toes. Lesional areas should be brushed last so as not to spread infection more diffusely. Most cats find this procedure unobtrusive and non-irritating. The toothbrushing procedure is also recommended for follow-up cultures to assess resolution of disease.

Feline dermatophytosis is a self-limiting disease in most cases, however due to the zoonotic potential, treatment is typically recommended to speed resolution. This includes not only medical treatment for the cat for also environmental decontamination so as to prevent continued reinfection. Cats are notorious little “sporulators” when it comes to dermatophytosis. They will frequently shed infective spores over their entire body and into the environment (including the air!) even with localized disease. A study evaluated the detection of Microsporum canis spores in the environments of both infected cats and dogs; in all households with cats, spores were readily detected in the environment. Infective spores were only found in less than half of the households with infected dogs. In cats, dermatophytosis should NOT be considered a localized disease due to their ability to product large amounts of infective spores and turn themselves into carriers of the organism. Where dogs can often be treated with local, topical medications, it is recommended that cats be treated both with a general topical agent (e.g. lime sulfur, miconazole/chlorhexidine) as well as a systemic antifungal agent (e.g. intraconazole, terbinifine). It is important to remember that the cat will look better and appear clinically “cured” prior to a mycological cure. Owners discontinuing treatment prior to complete cure of the animal is one of the most common reasons for treatment “failure” with feline dermatophytosis. Treatment length is variable; some patients may cure as early as about 4 weeks while others may take several months to cure. On average, it takes about 6-8 weeks to reach a mycological cure for dermatophytosis. It is recommended that treatment be continued until two or three consecutive fungal cultures are obtained at 1-2 week intervals (keeping in mind that it takes 3 weeks to finalize a fungal culture!). The toothbrushing technique can be demonstrated to the owner for ease in obtaining repeated cultures.

**Pemphigus foliaceus**

Pemphigus foliaceus is one of the most common autoimmune skin diseases in dogs and cats. The disease is characterized by production of autoantibodies directed against a component of adhesion molecules on keratinocytes (recently reported to be desmocollin 1) and deposition of antibody in intercellular spaces causing cells to detach from each other within upper epidermal layers. This process is caused acantholysis; these prematurely detached keratinocytes are round, stain deeply basophilic on cytology, have retained nuclei, and are referred to as “acantholytic keratinocytes.” Although these cells can be present in any skin disease which causes premature exfoliation of the epidermis (they can be seen in small numbers with superficial pyoderma and dermatophytosis in dogs), when present in large numbers (small clusters often referred to as “rafts”) this can be consistent with pemphigus foliaceus.

Although any age, sex or breed can be affected, there appears to be predisposition for disease development in Akitas, Chow Chows, bearded Collies, Newfoundlands, Schipperkes, and Doberman Pinschers. Pemphigus foliaceus usually develops spontaneously and without a known trigger; it is noted most commonly in adult dogs. This can however be a manifestation of cutaneous adverse drug reactions (documented thoroughly with Promeris® administration for example); a detailed medication history is necessary when working the patient up for pemphigus foliaceus. Although treatment is the same whether the disease developed spontaneously or due to a drug reaction, this can help provide future recommendations for treatments and more importantly those which should be avoided.
With pemphigus foliaceus, the primary lesion is a pustule. These pustules can be very large, often spanning multiple hair follicles, and frequently rather flaccid. They tend to be quite fragile and rupture easily. It is often common for the owner to miss this stage of disease and rather bring the pet in when more "chronic" lesions are present. These would include secondary lesions of superficial erosions, crusts, scales, epidermal collarettes, and alopecia. The disease tends to wax and wane. If you are lucky, you may notice a wave of pustules (lesions all in the same stage); this would be uncommon for pyoderma which typically has lesions of all stages of folliculitis (e.g. papules, pustules, collarettes). Lesions of pemphigus foliaceus often develop on the nasal planum, ear pinnae, or footpads; these locations are unique and characteristic of autoimmune skin disease. That said however, haired skin can also be affected. When lesions are on non-haired skin though, the clinician should be prompted to consider autoimmune (or metabolic or neoplastic) diseases higher on the list of differentials; these locations do not have hair follicles and so this would not be a manifestation of folliculitis (e.g. pyoderma, demodicosis, dermatophytosis). Pemphigus foliaceus often begins on the bridge of nose, the periorcular skin, or ear pinnae, however it will often progress to more generalized disease. Footpad lesions are common and may be the only body region affected in some dogs and cats; hyperkeratosis is also typically noted along with “peeling” of the pad along the margins. In cats, lesions may also be noted around nail beds and nipples; these are also unique and a common feature of pemphigus in the feline patient. In some patients, you may see evidence of systemic illness with generalized skin disease; this can be manifested as lymphadenomegaly, limb edema, fever, anorexia, or depression.

Diagnosis for pemphigus foliaceus includes a good clinical examination of the patient and detailed history particularly as it pertains to previous medication administration. Cytology will typically show neutrophils and acantholytic cells, and variable eosinophils; a suspicious absence of bacterial organisms should prompt the clinician to consider pemphigus foliaceus. The condition however is confirmed by biopsy; ideally an INTACT pustule should be submitted to a dermatopathologist. Biopsy will show subcorneal pustules containing neutrophils and acantholytic cells, often in “rafts”, and variable eosinophils. Although secondary bacterial pyoderma is possible, culture will generally be negative for organisms. There are several options available for treatment, however immunosuppression of the patient is necessary. Prognosis is considered to be fair to good for the disease; most patients however require life-long therapy to remain in remission, however generally at doses much lower than required for initiation. Typically however, it is the side effects of long-term medication administration which become problematic. Careful monitoring with bloodwork and recheck examination along with periodic dose adjustment (be careful not to taper medication too quickly; this can make the disease more difficult to get back in remission) is necessary to control pemphigus foliaceus long-term.