The Pyoderma Prescription

The surface of the skin is not sterile, and it plays host to a variety of resident, transient, opportunistic and pathogenic bacteria. It is important to understand how this occurs, since pyoderma (bacterial infection of the skin) is commonplace, and is often a frustrating condition in veterinary practice.

Resident bacteria live and multiply on normal skin. They are located on the skin surface, and in the most superficial aspects of the hair follicles, and discourage opportunistic infections through effective competition.

Staphylococcus pseudintermedius is the most important organism in pyoderma of dogs, and healthy dogs are a frequent carrier of the organism. Dogs also carry the organism in the following locations: anus; genital tract, buccal mucosae, and; conjunctivae. Most dogs develop their own genetically unique strains of *Staphylococcus*, present in carriage sites and in pyoderma lesions, suggesting that dogs are not routinely colonized by the staphylococci of other dogs. These staphylococci can also be passed from dam to pup through the milk. If this organism is present on many normal dogs and lives as a commensal organism in most cases, then the logical question should be asked as to why it is responsible for perhaps 90% of canine pyodermas. The answer is that since the organism is not overly pathogenic, in must be host factors that allow the organism to take hold and do its damage.

To cause pyoderma, bacteria must be able to adhere to keratinocytes, take advantage of nutrients on the skin, compete effectively with resident organisms and overcome host defense mechanisms. Bacteria can enter from several different routes, but most penetrate through the hair follicles. There are then a number of host factors to overcome, in order for the bacteria to cause problems.

Any process that disturbs the skin's barrier function will predispose to pyoderma. This includes any abrasion that removes surface barrier functions, any inflammatory process that causes epidermal disorganization, any process that affects local temperature and relative humidity on the skin, or any proliferative epidermal disease that results in keratinocyte disarray. Finally, any underlying disease that impairs immune function can predispose to pyoderma. Therefore, it is most important to investigate underlying diseases that predispose to pyoderma, rather than just responding with antibiotics.

Pyodermas are typically characterized by their depth (superficial, intermediate, deep), or by their particular characteristics. Superficial (surface) pyodermas involve the epidermis and the outermost aspects of the hair follicles. Examples include pyotraumatic dermatitis ("hot spots"), fold pyodermas, and juvenile pustular dermatitis (often incorrectly referred to as impetigo, but much different than impetigo in children). These conditions are so

superficial that systemic antibiotics are often not needed. However, it is critical that the predisposing causes be addressed, and that the bacteria are managed, at least topically.

Intermediate pyodermas involve deeper penetration of bacteria, and most often result in folliculitis and perifolliculitis. These most commonly result from secondary reactions to allergies, parasites, keratinization disorders, and metabolic disorders.

Deep pyodermas involve yet deeper structures in the dermis and panniculus, including furunculosis (rupture of hair follicles with their contents discharged into the surrounding tissues), cellulitis, abscesses, and fistulating diseases (including German shepherd dog pyoderma, mycobacteriosis, etc.). These diseases either reflect a more exaggerated reaction to the causes of intermediate pyodermas, a major breach in immune function, or infiltration of an organism more pathogenic than *Staphylococcus pseudintermedius*.

Regional pyodermas are described based on the part of the body affected, and while they still obey the clinical doctrines of superficial, intermediate and deep pyodermas, they do have their own therapeutic approaches.

Staphylococcus schleiferi (*Staphylococcus schleiferi schleiferi* and *Staphylococcus schleiferi coagulans*) is also a significant pathogen in dogs, and many isolates are methicillin resistant and some are fluoroquinolone resistant. Opportunistic infections are not only capable of causing disease in dogs, but also transferring multi-drug resistance. It is therefore important to properly assess and manage cases, so as not to encourage multi-drug resistance to important antibiotics.

Superficial (Surface) Pyoderma

Cytologic evaluation of the pustular contents reveals neutrophils and phagocytosed cocci. Further confirmation is rarely needed, but histopathologic assessment often demonstrates a subcorneal pustular dermatitis. Treatment can almost always be accomplished with topical antiseptic agents, and systemic antibiotics are only required if the condition fails to respond to topics alone. Rarely, a short 7-10 day course of antibiotics will resolve the condition.

Intermediate Pyoderma (Folliculitis, Perifolliculitis, and Furunculosis)

These intermediate pyodermas are common in the dog and relatively rare in the cat. The problem starts as a maculopapular to papulocrustous eruption and the distribution depends somewhat on the underlying cause of the problem. There are a variety of underlying causes for the problem, including allergies, endocrinopathies, keratinization disorders and virtually any other process that disturbs the body’s ability to control surface infections.

Clinically, the process starts with macules and papules that evolve into pustules, although the pustular stage may not always be evident at the time of examination. In time, there is an outward peeling rim of scale (epidermal collarette), with central hyperpigmentation, sometimes collectively referred to as a target lesion. When hair follicles rupture and discharge there contents into the underlying tissues, marked inflammation ensues, and the process is known as furunculosis.

At this stage, culture may not initially be required, and empirical selection of antibiotics can be based on the presumption of infection with *Staphylococcus pseudintermedius*. Beta-lactam antibiotics (such as cefovecin, cefpodoxime, amoxicillin-clavulanate, cefadroxil, and cephalexin) should be considered first-line therapies for empirical therapy; fluoroquinolones should be reserved for use on the basis of culture and sensitivity. Lesions should be cultured in recurrent, deep, or non-responsive pyodermas. Antibiotics alone, however, are not sufficient for long-term management, as the underlying problem must be identified and corrected or the problem is likely to recur once antibiotic therapy has been discontinued. Twice weekly bathing with an antiseptic shampoo and conditioner is often very helpful in keeping surface bacterial numbers under control. Eventually the bathing interval can be weaned to once every 1-2 weeks, if the underlying problems have been resolved. If the underlying problem cannot be identified, pulse therapy with antibiotics and even staphylococcal bacterin immunotherapy can be considered. In this instance, bacterial cultures are indicated, and should be performed on minced tissue collected by biopsy.

Deep Pyoderma

Deep pyoderma occurs when the infection involves the deep dermis, subcutis, and other underlying tissues. It may occur as an extension of furunculosis, secondary to puncture wounds and other methods of microbial inoculation, and as independent entities.

Cellulitis refers to a deep bacterial infection that spreads between tissue planes and fails to localize as an abscess. Abscesses normally result from traumatic inoculation of microbes into the dermis or panniculus. They may appear as firm to fluctuant subcutaneous nodules that may exude pus and may be painful to the touch. With deep pyodermas, it is not unusual to see surface ulceration, and even fistulous tracts.

With deep pyodermas, it is imperative to determine underlying cause(s). In some cases, such as when the pyoderma has resulted from bite wounds or other trauma, the cause is apparent. In other instances, the condition could result secondary to immune compromise, or metabolic diseases. A search for the underlying cause is necessary or the condition is unlikely to ever be completely resolved. Culture is indicated in most cases of deep pyoderma, and the best sample is taken by biopsy, with minced tissue prepared for microbe identification and susceptibility testing.

Antibacterial therapy is best attempted following culture, since the treatment period is often prolonged, perhaps months. Antibacterial resistance is common. In the case of

abscesses, surgical drainage, hydrotherapy, and the use of hot compresses should ideally precede antibacterial therapy.

Biofilms

Biofilms are complex communities of bacteria embedded within a slime matrix that adheres to tissue surfaces. This matrix allows bacteria to shield themselves from the host immune response and antimicrobial therapy.

Biofilms are more often associated with deep and persistent infections and Staphylococcus *pseudintermedius* is a potential biofilm producer. *Pseudomonas aeruginosa* can be a biofilm producer in otitis cases.

Biofilm-associated bacteria may not be readily accessible for samples obtained using standard culture techniques and growing them on standard culture media can be challenging.

Treating biofilms can be complicated by the fact that antimicrobial sensitivity testing may only test planktonic (non-biofilm-forming) bacteria, and antibiotic doses for biofilm can be thousands of times higher than that needed to kill planktonic bacteria of the same strain. Thus, antimicrobial therapy may resolve clinical signs of infection temporarily, only to have the problem recur after cessation of therapy. The best treatment for biofilm-associated infections is debridement and tissue irrigation preceding antimicrobial therapy.

Non-Pyodermas

It is also important to remember that not everything that looks like a pyoderma is a primary bacterial disease. For example, pemphigus foliaceus may present with pustules and with crusting, and is often initially confused with a pyoderma. The same can be said for zinc-responsive dermatosis, dermatophytosis, demodicosis, sterile eosinophilic pustulosis, juvenile cellulitis, and sometimes even cutaneous T-cell lymphoma.

Recommended Reading:

Ackerman, L: Atlas of Small Animal Dermatology. Inter-Medica Publishing, 2008.

Banovic, F; Olivry, T; Baumer, W; et al: Diluted sodium hypochlorite (bleach) in dogs: antiseptic efficacy, local tolerability and in vitro effect on skin barrier function and inflammation. Vet Dermatol, 2018; 29: 6-13.

Larsen, RF; Boysen, L; Jessen, LR; et al: Diversity of *Staphylococcus pseudintermedius* in carriage sites and skin lesions of dogs with superficial bacterial folliculitis: potential implications for diagnostic testing and therapy. Vet Dermatol, 2018; 29: 291-295.

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