DIAGNOSIS AND MANAGEMENT OF MALASSEZIA DERMATITIS

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OVERVIEW

Malassezia is a genus of lipophilic yeast found as a commensal of the skin and mucosal surfaces that may cause skin disease in a variety of mammalian species. In normal dogs these organisms are present in very small numbers on the skin (fold areas-lip, vulvar, axillae, interdigital), oral and anal mucosal surfaces, in the ear canals and in the anal sacs. In contrast to Candida, MD is not associated w/recent antibiotic administration, in fact, there appears to be a symbiotic relationship between the surface staphylococcal organisms and the yeast. It is theorized that the organisms produce growth factors and micro-environmental changes (eg inflammation) that are beneficial to each so it is not uncommon to see concurrent infections w/Malassezia and staphylococcus. Why do animals develop Malassezia dermatitis (MD)? There have been numerous studies comparing the strain of Malassezia organisms found on skin of affected dogs vs. the skin of unaffected dogs. To date there has not been an identifiable difference in virulence and/or adhesion in Malassezia organisms found on skin of affected dogs vs. the skin of unaffected dogs. Since the organism virulence doesn’t explain MD, the explanation seems to be the host response to Malassezia organism. Both type I and type IV hypersensitivity reactions to Malassezia have been identified in dogs w/MD. With Malassezia overgrowth, there are a variety of events that occur that can contribute to cutaneous inflammation. The metabolism of the surface free fatty acids by lipases produced by Malassezia leads to the production of inflammatory eicosanoids, changes in the cutaneous pH and decreases in the normal cutaneous barrier function. Due to the percutaneous absorption of Malassezia antigen, complement activation may occur. Disorders that affect the barrier function of the skin (eg pruritic skin disease) or the cutaneous lipid content (eg hypothyroidism) are risk factors for developing MD

SIGNALMENT

There is no age or sex predilection

HISTORY

MD is always secondary to another skin disease. A clue that MD may be present is that the clinical features and/or the previously effective therapy of the underlying disease becomes ineffective. For example, pruritus that was seasonal becomes nonseasonal; the distribution of the pruritus changes, responsiveness to previously effective antibiotic and/or glucocorticoid therapy is decreased. Any allergic animal whose pruritus (intensity or distribution) or the therapeutic responsiveness of the pruritus changes suddenly should be evaluated for MD, pyoderma and ectoparasites.

CLINICAL FINDINGS

On physical examination lichenification, erythema, greasy exudate, dry scale, papules, plaques, alopecia or hyperpigmentation may be present. A moist dermatitis with a musty odor is not an uncommon clinical finding. Pruritus may vary from mild to intense and erythema may be present with minimal pruritus especially interdigitally. The lesions may be focal or generalized and the distribution of the lesions overlaps with other pruritic diseases. Affected areas include interdigitally, intertriginous areas, face, nail folds, perioral (lateral muzzle), pinna and flexor surface of the elbow

DIAGNOSIS

MD may cause a folliculitis that is clinically identical to staph pyoderma. Therefore if there are follicular papules, epidermal collarettes or lichenification you can’t assume that there is a bacterial component to the skin disease without performing skin cytologies. Remember to include skin scrapings for ectoparasites as part of your minimum data base.

Identifying Malassezia organisms budding yeast from the affected area is necessary to establish a diagnosis of MD. Tape impression or direct impression smear are the most common method used for sampling affected areas. Because of the ease of collection, the author prefers the former. If the skin is dry a scalpel blade or the edge of a glass slide is used to collect the sample. If using slides for an impression smear there is no need to heat fix the sample nor to use all 3 stains that are part of the Diff-Quik® procedure. Using just the 3rd stain is adequate and will make processing the slide faster. Even faster is to take a tape impression, place a drop of the 3rd Diff-Quik® stain on the slide and then lay the tape on top of the stain. Use the tape as a coverslip and examine the sample after placing oil immersion on top of the tape.

The question is “how many is too many organisms?” A previous report found that normal dogs had 1 yeast per 2700 oil field. MD is confirmed when, on cytology, you find ANY field that has more 1 organism OR if there is 1 organism every 1-3 fields (1000X).

The ACVD task force on atopic dermatitis discussed MD as a complication of atopic dermatitis. The task force states that “Surface cytology of the skin and ear is useful to determine whether or not Malassezia or Staphylococci are present at lesional sites. Making antimicrobial treatment decisions based solely on microbe numbers is incorrect and inappropriate.” The article goes on to discuss that the host response to these normal
organisms determines the severity of clinical signs. Their recommendation was “the result of cytology might better be limited to the sole report of ‘presence’ or ‘absence’ of detectable bacteria or yeast”.

An interesting question is “Why can’t I find yeast when I’m sure the dog has Malassezia dermatitis?” Obviously site selection has a significant impact. Because Malassezia is a surface organism whose numbers may be decreased by the animal’s licking, multiple sites need to be sampled. In addition, because even small numbers of organisms can be significant, multiple fields need to be examined. Lastly, interpretation of the number of organisms from skin cytology is very different than interpreting ear cytologies in which 1 or 2 organisms may be interpreted as “negative” or “rare” and is of no clinical significance. This is not true when interpreting skin cytologies.

TREATMENT

In order to prevent recurrence of MD the underlying cause must be identified and treated. As previously mentioned any disease that disrupts the barrier function, the lipid content of the skin surface, the cutaneous microclimate or host defense mechanisms may predispose the animal to MD. These include hypersensitivities (atopy, cutaneous adverse food reactions), ectoparasites (demodex, sarcoptes, and fleas), endocrinopathies (hypothyroidism, hyperadrenocorticism), metabolic diseases (metabolic epidermal necrosis), neoplasia (cutaneous T-cell lymphoma) and excessive skin folds. Genetic factors, as seen in Bassett hounds, predispose a dog to maintaining higher number of Malassezia organisms on their skin, putting them at greater risk for developing MD.

Unless the MD is very focal, the author prefers both topical and systemic therapy. This combination will be the most successful treatment of MD. Eliminating MD as the cause of pruritus is important so that when the dog is rechecked any remaining pruritus is a result of the underlying hypersensitivity reaction, not the MD.

There are a variety of effective topical agents including selenium sulfide, miconazole, ketoconazole, clotrimazole and chlorhexidine. A review article revealed strong evidence for the shampoo combination miconazole/chlorhexidine (Malaseb®), although further studies are needed to further evaluate the true of some of the other topical products. In the authors experience any shampoo that contains at least 3% chlorhexidine or contains 2% chlorhexidine combined w/an azole is effective. Shampooing should be followed by a leave on conditioner containing an antifungal ingredient such as 2% miconazole. Depending on the severity and extensiveness of the lesions the frequency of application varies from daily to 3x/week.

Ketoconazole (200 mg tabs) 5-10 mg/kg sid is the systemic drug of choice. If the dog doesn’t tolerate ketoconazole (eg GI disturbances), itraconazole 5 mg/kg given 2 consecutive days/week is also effective, albeit more expensive. Since fluconazole is not metabolized or excreted by the liver, if the dog has liver disease, fluconazole at 5-10 mg/kg daily would be a good choice. Also because it comes in a 50 mg tablet, it is more amenable for dosing small dogs. Treatment should be continued for 14 days beyond clinical resolution BASED ON YOUR examination (not a phone call) with a minimum treatment time of 21 days. Please note that griseofulvin is ineffective against Malassezia.

Be sure to evaluate the dog for concurrent superficial bacterial pyoderma since MD and pyoderma occur simultaneously in dogs. In cases of concurrent superficial bacterial pyoderma, antibiotic therapy should be used simultaneously.

References