Malassezia dermatitis

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Malassezia pachydermatis is a saprophytic yeast commonly found on normal and abnormal skin of dogs. Malassezia pachydermatis and staphylococci play a significant role in seborrheic dermatitis. Malassezia dermatitis (MD) is common and should be considered in any case exhibiting erythematous, oily, and pruritic dermatitis (1). Specialty dermatology practices may see 2 or more cases of MD a week.

Malassezia dermatitis is often the reason for therapeutic or diagnostic failures. When there is a lack of response to glucocorticoids, antibiotics, antiseborrheic shampoos, insecticides, or miticides, one should consider a diagnosis of MD (2). Some atopic patients that have failed to respond to desensitization have subsequently been diagnosed as having MD and, then, have responded well to desensitization after the MD was successfully treated. The diagnosis of food allergy relies upon the owner’s observation of decreased pruritus while the patient is on an elimination food trial. If the patient has MD concurrently with the food allergy, the owner will not observe any decrease in pruritus until the MD has been successfully treated.

Malassezia organisms can be cultured easily from normal dog skin, but cytological demonstration of the organism from normal skin is much more difficult. A decrease in the host’s defenses or changes in the surface microclimate, such as, excessive sebum, accumulation of moisture, and disruption of the epidermal barrier, may lead to proliferation of Malassezia organisms. Allergic, hormonal, and bacterial skin diseases may be predisposing factors, as are longterm glucocorticoid administration and antibiotic administration (2). One study demonstrated that atopic dogs had higher numbers of yeast than did normal dogs, but not as many as dogs with MD. Factors associated with increased prevalence of higher counts of Malassezia pachydermatis were seborrheic dermatitis, recent antibiotic treatment, and certain breeds (3).

Malassezia pachydermatis produces lipases that can liberate fatty acids and zymogen in the yeast cell wall, which activates complement, both actions may contribute to cutaneous inflammation (1). Two studies have suggested that the pathogenesis of MD may be related to the yeast antigens acting as an allergen and thus causing an immediate-type hypersensitivity reaction. The 1st study showed that when seborrheic dogs were skin tested with M. pachydermatis antigen, 30% of them demonstrated immediate skin reactions (4). The other study, which also injected M. pachydermatis intradermally, showed that the mean immediate skin test reactions were greater in atopic dogs with MD than in atopic dogs without MD, which, in turn, were greater than in normal dogs (5). When dogs with MD are biopsied, the histology is usually consistent with a hypersensitivity pattern (1). Some of this hypersensitivity reaction may be due to the yeast, while some can be attributed to an underlying food allergy or atopy.

There are many clinical manifestations of MD. Pruritus of a moderate to severe nature is usually a constant feature. Erythema, hyperpigmentation, erythroderma, grey to white scales, and waxy or oily seborrhea are all variable in degree, as is the associated offensive odor.
Regional MD is far more common than the generalized form. The areas of the body that tend to be more moist and oily are more prone to MD. The most common presentation is pododermatitis involving the interdigital spaces or the area between the pads. It is helpful to ask the owner if the dog actually turns the paw over to chew the undersurface of the paws, rather than just licking the top. Although many allergy-prone breeds may be affected, Malassezia pododermatitis is commonly seen in cocker spaniels and golden retrievers. Other common regional areas commonly affected include the neck, perianal (under the tail), face (lips, chin, facial folds), and leg folds (Figure 1). Paw licking and face rubbing are often associated with allergies, but MD may also be an important contributor. Continual face rubbing with frenzied fits of nose, chin, or lip scratching may be observed in conjunction with mild erythema, scale, and minor amounts of alopecia. However, this latter dermatitis may be overlooked, as long hair in the area may obscure these clinical signs. It is imperative that the hair be clipped and the skin closely examined (6).

Pruritus in the perianal area is often associated with anal sacculitis or allergies. However, dogs that rub their back end should be examined for MD under the tail. Dachshunds with “accordion-like” folds of skin on their legs are also predisposed to developing MD (Figure 2). Paw sucking may be associated with a dark brown discharge from swollen nail beds and may indicate paronychia due to Malassezia pachydermatis (7).

Generalized MD is much less common than regional MD. Most of the generalized MD cases occur in medium to small breed dogs, although 1 author reported 2 cases in German shepherds, and I have treated a case in a Samoyed that was secondary to hypothyroidism (Figure 3). In addition to the predisposed breeds listed in Table 1, I would add golden retrievers with MD of the paws secondary to atopy to the list of predisposed breeds in Table 1.

A diagnosis of MD is usually suggested by the history, clinical signs, and failure of other therapeutic modalities. The most useful and readily available procedure for
the diagnosis of MD is cytological examination. A sample for this can be collected by rubbing the skin with a cotton swab, taking a skin scraping, or making a slide impression. The slide is heat-fixed and stained with Diff-Quick (Dade Diagnostics, Puerto Rica) or new methylene blue. Some clinicians have found clear cellophane tape to be superior for collecting surface skin debris and scales (8). Packing tape is preferred, as it does not curl up as Scotch tape does after immersion in staining solutions. The tape is dipped directly into the stain with no heat fixing, and then viewed at 1000×. I have found a certain commercial pad of clear cellophane tape (Pat-it, 3M Masking and Packaging System Division, St. Paul, Minnesota, USA) the easiest to use, and it gives excellent results. The tape comes in dimensions of 3½ in by 6 in and there are 25 sheets to a pad. Each sheet can be cut longitudinally into three equal widths using a box cutter. The tape fits between toes or pads to facilitate the collection of samples (Figures 4–6). Ironically, 1 of the main purposes of this product is the removal of pet hair from clothes. The diagnosis of MD is achieved by demonstrating 1 or 2 yeast per field at 1000×. Generally, the more yeast organisms that are found, the more confident one is of the diagnosis of MD (8). When there are low numbers of yeast bodies present, the diagnosis of MD may be tentative, in which case a trial therapy may be the only way to confirm the diagnosis. Systemic treatment or intense topical therapy should result in a positive response (6).

The most effective treatment for MD is systemic therapy with ketoconazole (Nizoral, Janssen Pharmaceutica, Mississauga, Ontario), 10 mg/kg body weight (BW), PO, q12h, for 20 to 30 d (10). Topical therapy is usually applied concurrently. Topical treatments, when used alone, appear to be less reliable than systemic ketoconazole (11). One reason for this may be that top-

Table 1. Dog breeds predisposed to Malassezia dermatitis (2)

<table>
<thead>
<tr>
<th>Breed</th>
<th>Strain</th>
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<tbody>
<tr>
<td>Silk terrier</td>
<td>Shetland sheepdog</td>
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<tr>
<td>Australian terrier</td>
<td>Collie</td>
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<tr>
<td>Maltese terrier</td>
<td>German shepherd</td>
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<tr>
<td>Jack Russell terrier</td>
<td>Dachshund</td>
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<tr>
<td>West Highland white terrier</td>
<td>Bassett hound</td>
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<tr>
<td>Chihuahua</td>
<td>Cocker spaniel</td>
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<tr>
<td>Poodle</td>
<td>Springer spaniel</td>
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Topical therapy requires more rigorous frequent applications, either daily or every other day bathing, to be effective. Some cases may fail to respond to topical therapy because the yeast is too deep in the hair follicle or the patient is hypersensitive to the organism, thus requiring a more complete clearing of the organism than the topical therapy can achieve alone (4,11).

Topical therapy can be divided into 3 categories: Creams, shampoos, and rinses. Creams that have been used successfully on paws include miconazole (Conofite Cream 2%, Janssen Pharmaceutica), ketoconazole (Nizoral Cream, Janssen Pharmaceutica), and clotrimazone (Canesten, Miles Canada, Etobicoke, Ontario). Shampoos that have been successful include ketoconazole (Nizoral Shampoo, Janssen Pharmaceutica) and miconazole (Dermazole, Allerderm-Virbac, Fort Worth, Texas, USA); (Sebolyse, Dermcare-Vet, Queensland, Australia). Helpful but less effective shampoos include selenium sulphide (Selsun Blue, Abbot Laboratories, Montreal, Quebec; Sellen Suspension, P.V.U., Sanofi Santé Animale Canada, Victoriaville, Quebec), chlorhexidine 4% (Hibitane, Ayerst Laboratories, St. Laurent, Quebec), or chlorhexidine 1% (ChlorhexiDerm, DVM, Miami, Florida, USA). The best rinse is probably enilconazole (Imaverol, Janssen Pharmaceutica). Other rinses include lime sulfur (LymDyp, DVM; vinegar 50/50 dilution), and chlorhexidine disinfectant (Hibitane, Ayerst Laboratories) (1,9,11–13).

Generalized cases are usually treated with ketoconazole administered orally for up to 30 d. Occasional nausea or vomiting can be controlled by giving the tablets with food (6). Topical therapy is used concurrently, utilizing a shampoo with or without a rinse.

The high cost of ketoconazole has prompted some to use it only q24h, or to decrease the dosage to 5 mg/kg, BW, PO, q12h, while intensifying the topical therapy. While this approach may work for some cases, the overall success rate may decrease (9–11).

Regional or localized cases of MD may be treated fairly successfully with a combination of topical therapies without the concurrent use of systemic therapy. However, many of these cases may still benefit from concurrent use of systemic therapy (11).

References

This book was compiled by one of the founding medical scientists with a special interest in comparative hemostasis. This reviewer has had the privilege of knowing Dr. Lewis and learning from her work for nearly 3 decades. Her interest and research in the platelets and hemostatic mechanisms of unusual vertebrates and domesticated animals, like the goat, have clearly advanced our knowledge of these species.

The book is presented in 3 parts. Part I offers introductory materials and an overview of hemostasis in humans. Part II reviews studies of representative groups of vertebrates, mostly performed over the years by the author and her colleagues and collaborators. Part III summarizes comparative studies from the author’s laboratory and by others who have investigated hemostasis, hematology, and serology of a variety of mammals in comparison with those of humans.

The strength of this book comes from the data collected by Dr. Lewis and her associates on many unusual animal species over a period spanning several decades. It also provides an in-depth literature review of most of the relevant publications in this field prior to the 1980s. For this reason alone, the book is a treasure trove of knowledge gleaned from a wide literature base, most of which is not readily available to anyone interested in or studying this subject. Parts II and III are especially strong sources of the original literature upon which today’s clinical and research efforts in comparative hematology are largely based.

As with any text of this type, it suffers somewhat from being incomplete, especially with respect to publications from the last decade. Many of these more recent studies address the clinical and research aspects of animal hemostatic components at the molecular level, and have begun to investigate the genetic manipulation of spontaneous and induced animal models of the hemorrhage diseases. Research literature on hemostasis in rabbits, guinea pigs, rats, and hamsters is more extensive than described, although much of the control animal data for these species is found within published experimental studies and may be harder to locate. For example, several earlier publications of in-depth studies on these species from this reviewer’s group have not been mentioned. Similarly, early versions of review textbooks are cited, but the more recent ones are not (Kaneko’s “Clinical Biochemistry of Domestic Animals,” 4th ed, Academic Press, 1989; with a 5th edition currently in press; Schalm’s “Veterinary Hematology,” 4th ed, Lea & Febiger, 1986; and a new title by Jain, “Essentials of Veterinary Hematology,” Lea & Febiger, 1993). Other sections on the dog, cat, and horse (Part II, Chapters 19 and 23) are relatively weak in terms of their scope and the references, which are outdated or missing. However, some of the missing material is presented later in Part III, Chapter 27. Table 27.12 (p.349) has several notable omissions — namely, that von Willebrand’s disease is very common in the dog (should be ++++) and has also been reported in the horse; factor IX deficiency has been reported in several cat breeds; factor XII deficiency occurs in the dog; and hemophilia A has been reported in sheep and cattle. In relation to these literature sources, it is unclear why a bibliography is given on pp. 409–417, when it does not include all the references cited throughout the text. Readers wishing more information on literature from the last decade, especially on studies of acquired bleeding and thrombotic disorders in animals, can refer to the chapter and bibliography contained in the forthcoming 5th edition of the Kaneko text cited above.

On balance, this book is a very interesting and useful review of the subject of comparative hemostasis. It is an excellent literature resource for earlier works in this field.

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