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**Malassezia dermatitis: diagnosis & management**

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Introduction

*Malassezia pachydermatis* (Pityrosporum pachydermatis, *P. canis*) is a lipophilic, non-mycelial yeast with characteristic thick-walled elongated oval shape and unipolar budding. It is a commensal organism in the dog, residing on or in the skin, ear canals, anal sacs, vagina and rectum. It is considered as a 'secondary' pathogen in canine otitis externa. *Malassezia pachydermatis* was recently reported as an opportunistic nosocomial pathogen in a human intensive care nursery. *Pityrosporum ovale* (a related organism that colonizes humans) is considered to be both a commensal and an opportunistic pathogen.

Pathogenesis

*Malassezia pachydermatis* was controversially implicated by Mason as a cause of localized or generalized pruritic and inflammatory skin disease in dogs (1987). Mason hypothesized:

1. Alterations in host defense mechanisms and skin surface microclimate allow the organism to become a pathogen - a facultative pathogen?
2. Excessive sebum production, moisture accumulation and disruption of normal barrier function may lead to yeast proliferation
3. Lipases produced by the yeast may further alter the sebum film and zymosan in the yeast cell wall
4. Yeast proliferation and disruption of normal barrier function may lead to inflammation and pruritus. (A similar pathogenesis has been proposed in humans).

Role of hypersensitivity

Atopic dogs with *Malassezia* dermatitis developed an IgE-mediated, type 1 hypersensitivity to intracellular protein extracts of *M. pachydermatis*. Immediate hypersensitivity reactions occurred in 30.4% of 46 dogs and delayed hypersensitivity in 6.9% of 29 dogs with seborrheic dermatitis by intradermal skin testing. Seborrheic dermatitis in humans may be partially due to a Type 1 hypersensitivity reaction to *Pityrosporum ovale*.

Number of organisms required to cause clinical disease

Still not clearly defined. Apparently, surface yeast colonization without tissue invasion can lead to clinical disease. Concurrent colonization or infection with *Staphylococcus intermedius* may enhance the inflammation seen with *Malassezia* dermatitis.

Predisposing causes

1. Allergic disease - especially atopic dermatitis
2. Diseases of cornification
3. Chronic or recurrent inflammatory skin disease
4. Previous treatment with antibiotics or corticosteroids?
5. Marked breed predilections - bassett hounds, springer spaniel, German shepherd dog, West Highland white terrier, silky, Maltese, chihuahua, poodle, Shetland sheepdog, dachshund, Australian terrier, Newfoundlands?

Clinical 'syndromes' and features

Three distinct syndromes are described by Mason:

1. Secondary *Malassezia* dermatitis - associated with chronic, inflammatory skin disease, characterized by a strong 'seborrheic' odor and severe pruritus (common)
2. Primary *Malassezia* dermatitis - generalized, inflammatory skin disease with a strong 'seborrheic' odor, rapid onset, rapid therapeutic response and lack of reoccurrence, (rare)
3. Severe pruritus and self-trauma - localized to the muzzle or perianal area (pruritus out of proportion to the visual severity of the skin lesions), owners perceive as seizures!, rapid response, (very rare!)

Lesions are focal, multifocal or generalized with erythema, hyperpigmentation, alopecia, lichenification, scaliness and/or greasiness. Exudative dermatitis and a 'musty' seborrheic odor occur. Pruritus is a common unifying clinical feature. Lesions may be sharply demarcated, closely adjacent skin may be un-inflamed and normally haired. Alopecic, greasy, lichenified lesions gradually expand peripherally to involve previously normal adjacent skin. Site predilections are ventral neck, ventral abdomen, axilla, face, ears (pinna), feet, forelegs, any skin folds.

Diagnostic procedures

1. Yeast culture - not recommended as a diagnostic procedure due to difficulty in interpreting non-quantitative results
2. Skin biopsy - not a very helpful diagnostic procedure, histopathology of lesions with large numbers of organisms identified by cytology may not exhibit organisms. This seeming discrepancy could be explained by a non-linear distribution of organisms or removal of organisms during automatic processing.

However, other characteristics of *Malassezia* dermatitis besides the presence of organisms may be present and
increase the index of suspicion for this disease.  
3. Skin cytology - diagnostic method of choice - controversy still exists as to the best method of obtaining a reproducible number of organisms. Skin scraping, cotton swabs, direct impression smears, tape stripping and 'sticky' glass slides have been recommended.
   a) dry skin scraping - material obtained is smeared onto a glass slide
   b) dry cotton swabs - harvest debris and transfer it to glass slides
   c) direct impression smears - in regions where a glass slide can be firmly pressed against the affected area
   d) clear cellophane tape stripping - to acquire debris with the tape being used as a cover slip applied to ribbon of stain on a glass slide
   e) sticky glass slides - (Durotak® - Delasco Dermatologic Supply)
4. Microscopic evaluation - heat fixed on the glass slide, stained with Dif Quik® or new methylene blue and examined under oil
5. Malassezia - oval to peanut-shaped budding organisms that stain blue varying from faint to dark. The presence of at least three or four organisms per oil emersion field is considered significant by most dermatologists

Diagnosis
1. Identification of the organism in sufficient number to be considered a pathogen
2. Identification of predisposing underlying causes - if Malassezia dermatitis is treated simply as the pathogenic proliferation of yeast, than reoccurrence after therapy is the rule rather than the exception since in most cases, Malassezia dermatitis is a secondary disease.

Therapy
Ongoing management of documented underlying disease(s) or predisposing causes that may be the predisposing cause for the opportunistic pathogen:
Systemic therapy
Antifungals - most likely to both induce speedy resolution of skin lesions and markedly diminish the number of organisms that can be harvested by cytology. The availability of generic ketoconazole has greatly diminished the cost of therapy (200 mg tablets - $0.75 to $1.50 each, wholesale in the United States.) However, systemic therapy is still expensive, especially in larger dogs.
1. Ketoconazole (Nizoral® - Janssen, or generic) - orally, 5 mg/kg once daily - current drug of choice, given for a minimum of thirty days. (Some of the dramatic improvement seen with this therapy may be due to anti-inflammatory effects, plus the effect on the cornification process seen with ketoconazole). Some dermatologists advocate monitoring liver enzymes during therapy due to the controversy of possible hepatotoxicity in humans. Monitoring of healthy dogs probably is not necessary.
2. Itraconazole (SporonoX®-Janssen) - orally, 5 mg/kg given once daily for at least thirty days, itraconazole lacks anti-inflammatory effect seen with ketoconazole.
3. Fluconazole (Diflucan® - Roerig, or generic) - orally, 5 mg/kg once daily

Topical therapy - shampoos
Adjunct to systemic therapy, can be used as sole therapy if financial constraints, minimum of twice weekly for at least six weeks, (ideal product does not exist! - degreasen, anti-fungal, residual)
1. Miconazole/chlorhexidine gluconate - Malaseb® (DVM)
2. Ketoconazole/chlorhexidine gluconate - KetoChlor™ (Virbac)
3. Acetic Acid/boric Acid - MalaAcetic® (DermaPet)

Topical therapy - rinses
1. Miconazole - ResiZole® (Virbac)
2. Chlorhexidine - ResiChlor® (Virbac)
3. Acetic acid and water 1:1 (white vinegar & water)
4. Enilconazole (Imaverol® - Janssen), side effects?

Overview
Control or cure underlying causes, kill the yeast, combine systemic and topical therapy, hope no recurrence!

Recurrent Malassezia dermatitis
Similar to recurrent pyoderma, persistent underlying skin disease is the most commonly diagnosed cause of recurrent Malassezia dermatitis, frequently occurs together! Recurrent secondary Malassezia dermatitis is a frustrating problem both in general practice and in specialty dermatology referral practice. Recurrent secondary Malassezia dermatitis is more likely to occur when multiple predisposing causes are present in a predisposed breed. Consider the likelihood of a West Highland white terrier with multiple allergies and a defect in cornification developing secondary Malassezia dermatitis!

Prevention of recurrent Malassezia dermatitis
1. Goals of long term management - prevent or diminish frequency of recurrence
2. Long-term management of underlying skin diseases or other predisposing causes. (especially canine atopic dermatitis)
3. Topical therapy - shampoos and rinses
4. Systemic therapy - antifungals, extended regimens
   a) Ketoconazole (generic) - 5 mg/kg three days per week

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b) Fluconazole (generic) - orally, 5 mg/kg 3 days per week

References