Malassezia Dermatitis

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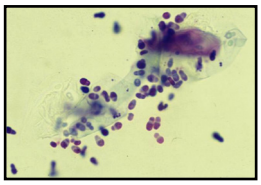
Etiology

- *Malassezia pachydermatis*
  - Lipophilic non-mycelial saprophytic monopolar budding yeast, 2-7 µm
  - Normal inhabitant of skin and mucosa of dogs and cats (perianal ring, ear canal, lips, interdigital skin)
  - Also isolated from wild carnivores, herbivores and monkeys; rare in man



Etiology

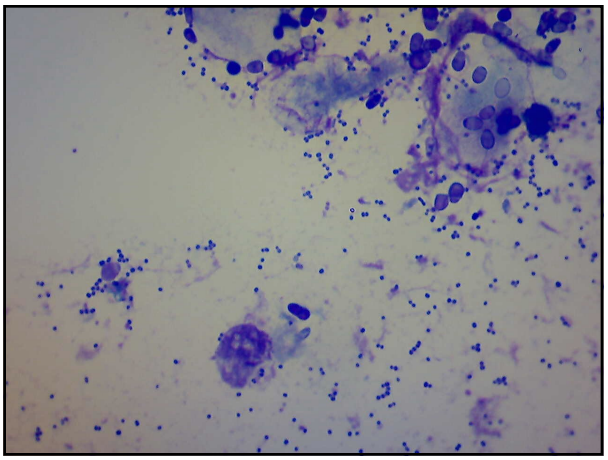
- *M. sympodialis* – cat, man
- *M. globosa* – cat, dog, man, bovine
- *M. furfur* – man, indian elephant, monkey
- *M. slooffiae* – pig, man, goat, sheep
- *M. obtusa* and *M. restricta* – man
- Identification is difficult due to the requirement for different lipid sources
- All are lipid-dependent, in contrast to *M. pachydermatis* which is non-lipid-dependent

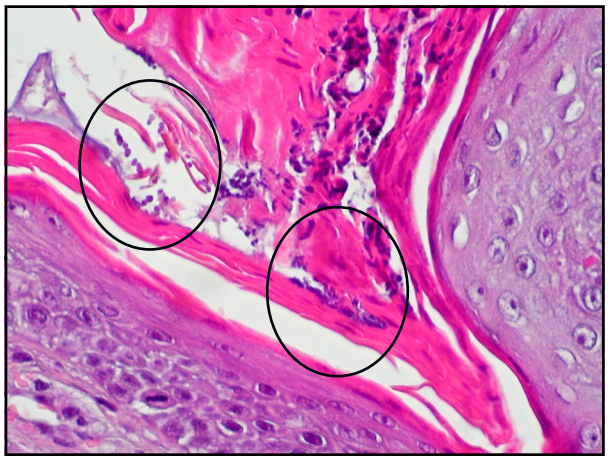
Epidemiology

- *M. pachydermatis* is a commensal in healthy animals
  - Lives on the surface of corneocytes
  - Excessive colonization and infection is prevented by:
    - Cutaneous desquamation
    - Fungistatic effect of epidermal lipids
    - Specific immunologic defense mechanisms (IgA)

Epidemiology

- Breed predisposition for:
  - Basset hound, WHWT, dachshund, poodle, cocker spaniel, shar pei, shih tzu, Labrador, German shepherd
- May have a symbiotic relationship with commensal *Staphylococci*
  - Dogs with *Malassezia* dermatitis often have bacterial overgrowth or pyoderma





Pathogenesis of Disease

- Opportunistic pathogen:
  - Increased colonization and infection when host defense mechanisms (physical, chemical, immunologic) are compromised:
    - Atopic dermatitis and other allergies
    - Endocrinopathies
    - Cornification disorders
    - Immune suppression (FIV/FeLV), glucocorticoids
    - Conformational effects (skin folds) → increased humidity

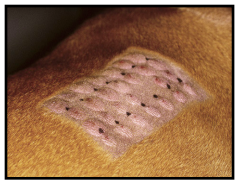
Malassezia	Noli et al. 2006	Bond et al. 1996	Plant et al. 1992
Atopic dermatitis	**36.9%**	**35.0%**	**26.3%**
Food allergy	**11.5%**	**15.0%**	**21.1%**
Flea allergy	**4.9%**		
Hypothyroidism	**1.6%**	**2.5%**	-
Testicular tumour	**1.6%**	-	-
Cushing's disease	**1.6%**	-	-
Seborrhea oleosa	**1.6%**	**5.0%**	**4.4%**

Pathogenesis

- Disruption of the stratum corneum
  - Mechanical due to pruritus
  - Biochemical
    - Humidity, immunologic abnormalities, alteration of skin surface lipids due to keratinization defects and endocrinopathies
    - Enzymes of yeasts and bacteria (esterase, lipase, lipoxigenase, protease, urease)
- Immune system is exposed to *Malassezia* antigens and products → *Malassezia* dermatitis/hypersensitivity

Pathogenesis: Hypersensitivity Reaction

- High levels of yeast-specific IgE have been found in some atopic dogs
- Atopic dogs with *Malassezia* infection have stronger intradermal test reactions to *Malassezia* antigens than atopic dogs without *Malassezia* dermatitis

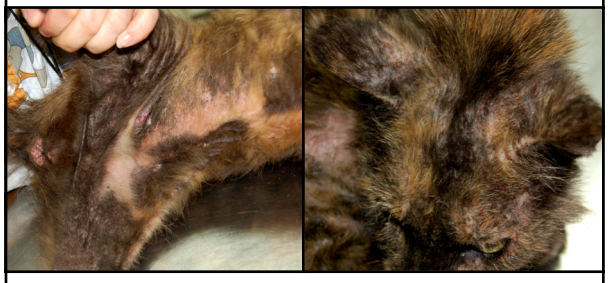


Clinical Signs

- Erythema, yellow scale and crust, greasy, malodorous skin, hyperpigmentation, lichenification
- Face, feet, ventral neck, abdomen, interdigital spaces, nail beds, skin folds
- Pruritus (often intense)

Malassezia Dermatitis in Cats

- Much less common vs. dogs
- Little is known about predisposing factors; higher numbers in FIV/FeLV+ cats
- Generalized infection in cats with neoplasia or severe diseases, including allergy
- Paronychia and chin acne
- Devon rex and sphynx predisposed



Diagnosis

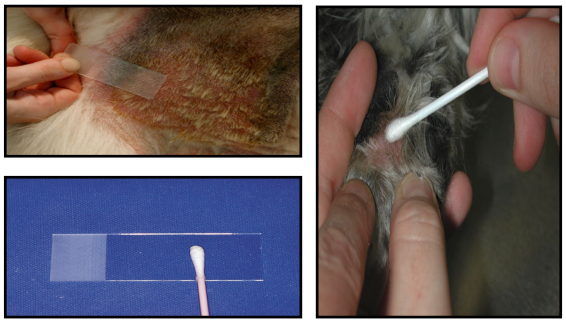
- Evaluate for *Malassezia* with any pruritic dermatitis
- May closely resemble and coexist with allergic disease, pyoderma and disorders of keratinization
- Diagnosis: elevated *Malassezia* population on lesional skin with good clinical and mycological response to antifungal therapy

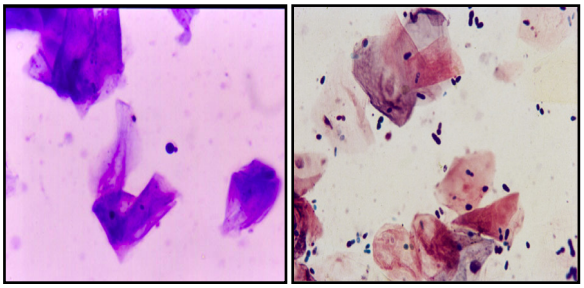
Diagnosis - Cytology

Acetate Tape Prep Technique

Diagnosis - Cytology

Impression Smear and Swab



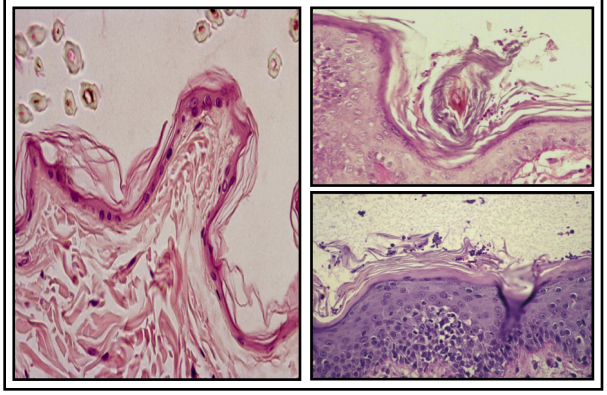


Normal Malassezia Infection

Histopathology

- Epidermis
  - Yeast in the stratum corneum
  - Hyperplasia and spongiosis
  - Lymphocytic exocytosis
- Dermis
  - Perivascular mononuclear infiltrate
  - Subepidermal linear alignment of mast cells
- Failure to visualize the yeast does not exclude infection

Normal Malassezia Infection



Diagnosis - Trial Therapy

- Difficult to assess because some antifungal agents have other actions:
  - Ketoconazole has immunomodulatory effect
  - Miconazole/chlorhexidine shampoo has antibacterial activity

Therapy - Topical

- Shampoo
  - Chlorhexidine 4%
  - Ketoconazole, miconazole, climbazole, econazole, selenium sulfide
  - Chlorhexidine 2%/miconazole 2%
  - 10 minute contact time
  - Twice weekly for 1 month then once weekly

Therapy - Topical

- Many preparations
  - Rinse, spray, cream, lotion, mousse
  - Best for localized infection
    - Enilconazole 0.2%
    - Miconazole cream/lotion
    - Clotrimazole cream/lotion
    - Climbazole pads/mousse
    - White vinegar:water (1:3 to 1:1)

Therapy - Oral

- Ketoconazole
  - 5-10 mg/kg q 24 hours with food for 21-30 days
- Fluconazole
  - 5-10 mg/kg q 24 hours with food for 21-30 days
- Itraconazole
  - 5 mg/kg q 24 hours
  - Pulse therapy – 2 consecutive days/week
- Terbinafine
  - 20-30 mg/kg q 24 hours
  - Pulse therapy – 2 consecutive days/week

Therapy – Follow up

- Re-exam in 3-4 weeks
- Very important to identify and manage underlying disease
- Maintenance therapy beneficial for recurrent infections
  - Once-twice weekly medicated shampoo combined with other topicals
  - Pulse therapy