DERMATOLOGY

Deep Fungal Dermatoses in Cats: Diagnosis and Treatment

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Abstract

Deep fungal dermatoses in cats are uncommon but potentially serious infections. They may be confined to the skin or may originate from, or disseminate to, other locations, such as the respiratory tract or central nervous system. Key diseases include sporotrichosis, cryptococcosis, histoplasmosis, blastomycosis, coccidioidomycosis, phaeohyphomycosis, and pythiosis. Clinical signs vary widely and frequently resemble bacterial infection, immune-mediated disease, or neoplasia. Consideration of geographic location, environmental exposure, and travel history is essential when formulating differential diagnoses. Definitive diagnosis typically requires integration of cytology, histopathology with special stains, serologic testing, diagnostic imaging, and fungal culture with molecular confirmation. Prompt diagnosis and appropriate antifungal therapy are critical to optimize clinical outcomes and inform prognosis.

Take-Home Points

- Apply a structured, stepwise approach to the evaluation of suspected deep fungal dermatoses in cats by integrating signalment, exposure history, cytology, advanced diagnostics, and appropriate therapeutic planning.
- Recognize key morphologic features of major feline deep fungal pathogens to guide provisional diagnosis and appropriate confirmatory testing.
- Select appropriate first-line and adjunctive antifungal therapies based on pathogen identification, disease distribution, and organ involvement.

Deep fungal dermatoses in cats are uncommon but clinically significant and may result in substantial morbidity or even death. Cats typically present with firm to fluctuant dermal or subcutaneous nodules or ill-defined swellings that may ulcerate and form draining tracts. Lesions may be solitary or multifocal and can exude serosanguineous to purulent material. Importantly, nodular or ulcerative skin disease does not necessarily indicate infection.

The most critical step is distinguishing infectious from noninfectious causes, as treatment and prognosis differ markedly. Misdiagnosing a fungal infection as an immune-mediated condition can be dangerous; immunosuppressive therapy, particularly glucocorticoids, may worsen local invasion and facilitate systemic spread. Therefore, an accurate diagnosis before initiating treatment is essential.

In North America, important pathogens include species of *Sporothrix*, *Cryptococcus*, *Histoplasma*, *Blastomyces*, *Coccidioides*, and melanized (dematiaceous) fungi. *Pythium*, although not a true fungus, can produce clinically similar invasive disease and is increasingly recognized. Infection typically follows inhalation of environmental spores or traumatic inoculation. Thus, a systematic diagnostic strategy, including careful history review, cytology, histopathology, fungal culture, molecular techniques, and antigen/antibody testing, facilitates accurate pathogen identification and supports timely clinical decision-making. **BOX 1** summarizes the red flags for suspicion of deep fungal dermatoses in cats, and **FIGURE 1** illustrates the step-by-step clinical diagnostic algorithm for deep fungal dermatoses in cats.

History and Physical Examination

Signalment

Deep fungal infections can affect cats regardless of age, breed, or sex; however, specific predispositions have been documented. For example, cryptococcosis is often seen in young adult cats, and sporotrichosis is most frequently identified in young, sexually intact, free-roaming male cats, likely due to increased environmental exposure and cutaneous inoculation from fighting and trauma.[1] While many affected cats are immunocompetent, severe or disseminated disease may be more likely in immunocompromised individuals.

Lifestyle, Environment, and Travel History

A thorough residence and travel history is essential, as many pathogens are associated with defined endemic regions, such as semiarid desert areas (e.g., Arizona) for coccidioidomycosis.[2] Geographic location can substantially influence the likelihood of specific mycoses and should always be interpreted in the context of travel history.

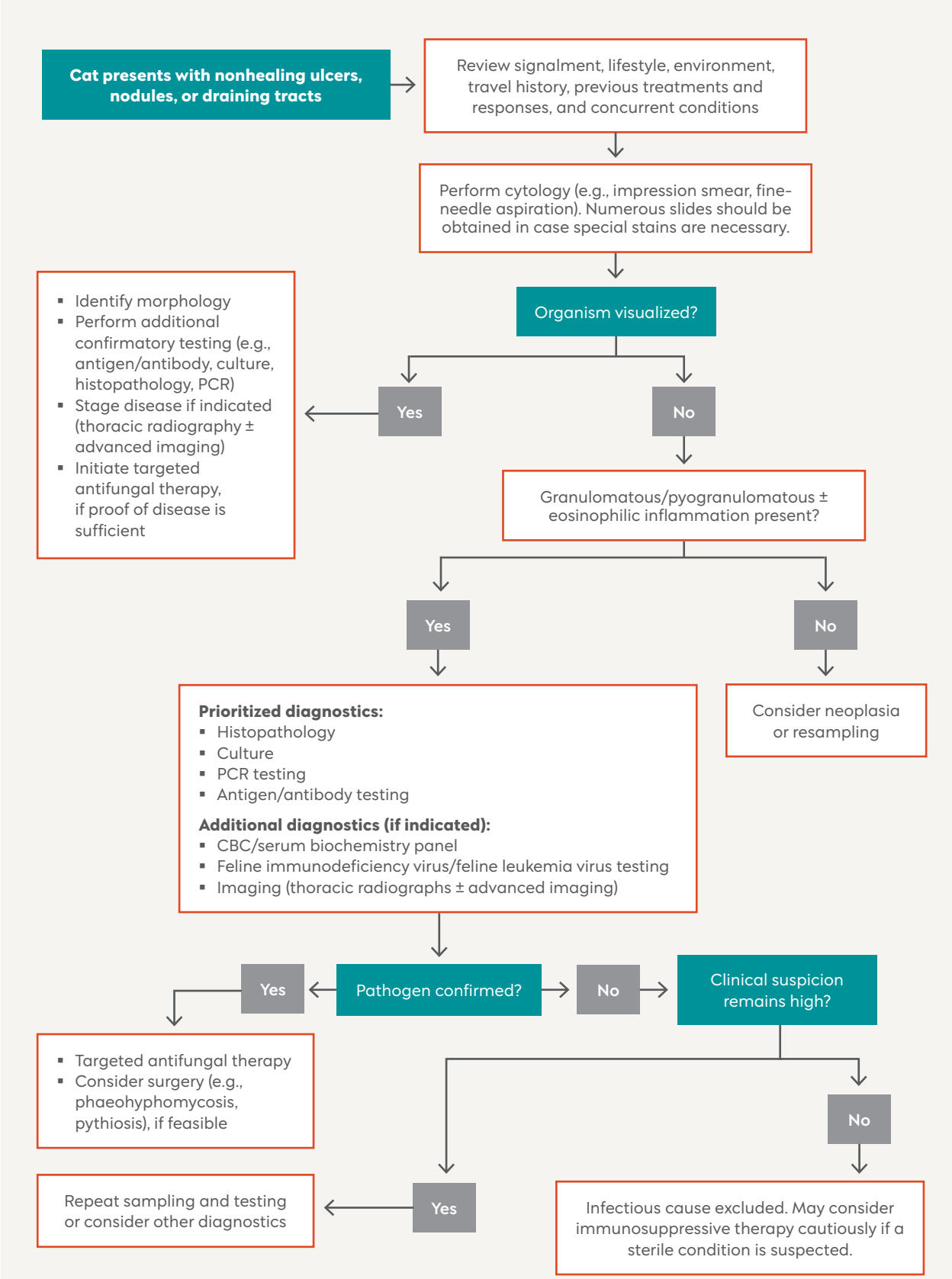

FIGURE 1. Diagnostic algorithm for deep fungal dermatoses in cats.

Environmental exposure plays a central role in pathogenesis. Inhalation of environmental spores is the most common route of infection for most systemic fungi, whereas some fungal infections are typically acquired through traumatic inoculation of the skin. Outdoor access; hunting behavior; and exposure to soil, decaying vegetation, bird guano, or standing water should be carefully documented. Importantly, indoor-only cats are not exempt from risk. Many fungal spores are airborne or may be introduced into the home through contaminated organic materials, including potting soil. Therefore, absence of outdoor access does not eliminate the possibility of deep mycosis.

Previous Treatments and Responses

A typical history includes chronic, nonhealing nodules or draining tracts that showed limited to partial response to multiple courses of empirical antibiotics. Temporary improvement may reflect control of secondary bacterial infection rather than cure. A particular red flag is rapid progression or systemic spread after initiation of immunosuppressive medications (e.g., glucocorticoids, cyclosporine) prescribed for a presumed immune-mediated condition. Such deterioration should immediately prompt reconsideration of an infectious etiology.

BOX 1
Red Flags for Suspicion of Deep Fungal Dermatoses in Cats

1. **Exposure risk (endemic region, travel history, outdoor access)**
   - Soil, vegetation, bird/bat guano, water, trauma
2. **Chronic, nonhealing ulcers, nodules, or draining tracts**
   - Unresponsive or partially responsive to antibacterial treatment
3. **Rapid worsening after immunosuppressive therapy**
   - Reconsider infectious etiology immediately
4. **Granulomatous/pyogranulomatous inflammation on cytology**
   - Other differentials include bacterial infections, immune-mediated disorders, and foreign body reactions

Concurrent Conditions

Although some cats with deep fungal infections are systemically immunocompetent, concurrent diseases can affect susceptibility, treatment response, and prognosis. Retroviral testing for feline immunodeficiency virus (FIV) and feline leukemia virus (FeLV) is recommended, as infection may impair immune function and increase relapse risk. Neoplasia or prior immunosuppressive therapy should also be documented, as immunosuppression predisposes to dissemination and complicates management.

Determining the Cause

Assess Cytology

Cytology is a critical first-line diagnostic tool because it is rapid, is minimally invasive, and often yields clinically actionable information. Fine-needle aspiration of intact nodules or lymph nodes, along with impression smears from ulcerated areas or draining tracts, should be routinely performed and evaluated using Romanowsky-type stains (e.g., Diff-Quik). Identification of pyogranulomatous or granulomatous inflammation should raise suspicion for fungal infection.

Organisms may appear as yeasts (*Sporothrix*, *Cryptococcus*, *Histoplasma*, *Blastomyces*), pigmented hyphae (dematiaceous fungi), or negatively stained hyphae (*Pythium*). However, failure to visualize organisms does not exclude infection. If cytology reveals a pathogen consistent with the clinical presentation, subsequent diagnostics should focus on confirmation and speciation.

Choose Further Diagnostics

If cytology is inconclusive or suggests infection without definitive organism identification, additional diagnostics are necessary to confirm the pathogen, assess systemic involvement, and guide treatment planning.

CBC and Biochemistry

Systemic fungal infections frequently produce nonspecific laboratory abnormalities. Common findings include an inflammatory leukogram, mild to marked hyperglobulinemia, and nonregenerative anemia. Hypercalcemia may occur secondary to

macrophage activation within granulomatous lesions. Serum biochemistry also establishes baseline hepatic and renal function prior to prolonged antifungal therapy. Concurrent testing for FIV/FeLV is recommended, as retroviral status may influence treatment duration, clinical response, and overall prognosis.

Fungal-Specific Tests

Antigen and antibody assays can provide valuable, minimally invasive diagnostic information and are particularly useful early in the workup. For example, the cryptococcal capsular polysaccharide antigen test (latex agglutination) demonstrates high sensitivity (95%) when performed on serum or cerebrospinal fluid.[3] Urine antigen enzyme-linked immunosorbent assay (ELISA) for *Histoplasma* is similarly sensitive (90% to 94.4%),[4,5] although cross-reactivity with *Blastomyces* may occur. For suspected coccidioidomycosis, antibody detection via agar gel immunodiffusion (AGID) remains the preferred diagnostic modality, with reported sensitivity of 97.7% to 100%.[2,6]

Diagnostic Imaging

Thoracic radiography is often recommended, as inhalation is the primary route of some infections. Advanced imaging (computed tomography or magnetic resonance imaging) may be used when evaluating sinonasal, orbital, or central nervous system disease and for determining the full extent of regional invasion.

Histopathology

Histopathology is commonly employed to characterize the pattern of cutaneous lesions, classify disease, exclude neoplasia, assess depth of invasion, and ideally identify and provisionally classify fungal organisms. Multiple deep biopsies are recommended, as fungal elements are often concentrated within necrotic centers of lesions. Routine hematoxylin and eosin staining may not always reliably highlight fungal structures; therefore, special stains such as periodic acid–Schiff and Grocott methenamine silver, among others, are sometimes necessary to enhance visualization.

Fungal Culture

Culture of fresh tissue biopsy specimens, rather than swabs, significantly improves the likelihood of isolating the causative fungal organism. Laboratories should be notified in advance if highly infectious and zoonotic pathogens such as *Blastomyces*, *Histoplasma*, or *Coccidioides* are suspected. Culture of these organisms should be performed only when clinically necessary.

TABLE 1 Key Clinical Features and Treatments of Selected Deep Fungal Dermatoses in Cats

	SPOROTRICHOSIS	CRYPTOCOCCOSIS	HISTOPLASMOSIS
Primary mode of transmission	▪ Traumatic inoculation ▪ Cat bites/scratches	▪ Inhalation of basidiospores or desiccated yeast	▪ Inhalation of microconidia
Major clinical signs	▪ Ulcerated nodules ▪ Draining tracts on nasal bridge, limbs, and tail base	▪ Respiratory signs ▪ Nasal swelling, nodules ▪ CNS, ocular signs	▪ Respiratory signs ▪ Nodules, ulcers
Preferred diagnostic tests	▪ Cytology ▪ Tissue culture ▪ Histopathology	▪ Cytology ▪ Serum/CSF antigen test ▪ Histopathology	▪ Cytology ▪ Urine antigen ELISA ▪ Histopathology
Key morphologic features	▪ 1–3 µm × 2–10 µm cigar-shaped yeasts	▪ 3.5–20 µm encapsulated yeasts (“soap bubble”)	▪ 2–4 µm intracellular yeasts in macrophages
First-line treatment	▪ Itraconazole ± terbinafine	▪ Fluconazole ▪ Itraconazole	▪ Itraconazole

AGID = agar gel immunodiffusion; CNS = central nervous system; CSF = cerebrospinal fluid; ELISA = enzyme-linked immunosorbent assay

Molecular Diagnostics

Panfungal PCR testing targeting the internal transcribed spacer regions can support species identification. It may be performed on cultured isolates and on fresh, frozen, or formalin-fixed paraffin-embedded (FFPE) tissues when culture is unavailable or negative. In one study, amplifiable fungal DNA was obtained from FFPE samples in 69.5% of cases, with 61.7% agreement between molecular and histologic results.[7] PCR testing can also be applied to stained cytology slides, showing up to 86% sensitivity when fungal organisms are abundant.[8] Since this technique may detect environmental contamination, results must be interpreted in conjunction with clinical presentation, cytology, and histopathology.

Determining the Treatment

If no pathogen is identified despite a thorough diagnostic investigation, noninfectious conditions such as sterile nodular panniculitis should be considered. These disorders often require immunosuppressive therapy, which must be administered cautiously and monitored closely. Rapid clinical deterioration following immunosuppression should prompt reconsideration of an infectious cause and repeat diagnostic testing.

> Laboratories should be notified in advance if highly infectious and zoonotic pathogens such as *Blastomyces*, *Histoplasma*, or *Coccidioides* are suspected.

If a specific pathogen is identified and consistent with the clinical presentation, antifungal therapy should be initiated and appropriate additional tests performed to assess the extent of disease. Itraconazole is commonly used as first-line medical therapy for sporotrichosis, histoplasmosis, and blastomycosis, whereas fluconazole is often preferred for cryptococcosis and coccidioidomycosis due to its superior penetration into the central nervous system when neurologic involvement is present. Posaconazole is a broader-spectrum triazole that may offer improved efficacy in some refractory cases, and pharmacokinetic data are available to support use of the oral suspension formulation.[9] Amphotericin B may be indicated for severe, disseminated, or life-threatening infections; the

BLASTOMYCOSIS	COCCIDIOIDOMYCOSIS	PHAEOHYPHOMYCOSIS	PYTHIOSIS
▪ Inhalation of conidia	▪ Inhalation of arthroconidia	▪ Traumatic inoculation	▪ Zoospores from stagnant water penetrate damaged skin
▪ Respiratory signs ▪ Nodules, draining tracts	▪ Respiratory signs ▪ Nodules, nonhealing ulcers ▪ Bone, ocular, CNS signs	▪ Pigmented nodules, ulcers, draining tracts	▪ Subcutaneous masses, draining tracts
▪ Cytology ▪ Urine antigen ELISA ▪ Histopathology	▪ Cytology ▪ Antibody detection via AGID ▪ Histopathology	▪ Cytology ▪ Tissue fungal culture ▪ PCR testing ▪ Histopathology	▪ Cytology ▪ Tissue fungal culture ▪ PCR testing ▪ Histopathology
▪ 5–20 µm broad-based budding yeasts	▪ 10–100 µm spherules containing 2–5 µm endospores	▪ 2–10 µm dark-walled hyphae	▪ 2–7 µm broad, irregularly branching, pauciseptate, negatively stained hyphae
▪ Itraconazole	▪ Fluconazole ▪ Itraconazole	▪ Surgical excision (if resectable) with itraconazole/posaconazole/terbinafine	▪ Surgical excision (if resectable) ▪ Limited evidence on anti-inflammatory doses of glucocorticoids, terbinafine, minocycline, mefenoxam, and hyperbaric oxygen therapy

Sporothrix brasiliensis has primarily been reported in South America, specifically Brazil and Chile, whereas *S schenckii* has a worldwide distribution.

deoxycholate formulation is effective but associated with greater nephrotoxicity, whereas lipid complex formulations are better tolerated but more costly.

In selected cases of phaeohyphomycosis and pythiosis, complete surgical excision may be the treatment of choice when feasible. Medical treatment is typically prolonged for several months and should continue beyond clinical resolution. Close monitoring for adverse effects and relapse is essential.

Selected Deep Fungal Dermatoses

Key features and treatments of the following deep fungal dermatoses are summarized in **TABLE 1**.

Sporotrichosis

Sporotrichosis is caused by the dimorphic fungi *Sporothrix schenckii* species complex. *Sporothrix brasiliensis* has primarily been reported in South America, specifically Brazil and Chile, whereas *S schenckii* has a worldwide distribution. Infection follows traumatic inoculation with contaminated plant material; however, horizontal transmission through cat bites and scratches is also common, especially among free-roaming intact males. Cats often harbor large numbers of organisms and represent an important zoonotic source. Owners should wear gloves and protective clothing, and, in severe cases, respiratory and eye protection when handling affected cats.

Dermatologic lesions include multifocal ulcerated nodules and draining tracts, frequently involving the nasal bridge, distal limbs, or tail base. Cytology is often highly diagnostic in cats, revealing pleomorphic oval- to cigar-shaped yeasts measuring approximately 1 to 3 µm wide and 2 to 10 µm long, typically observed within macrophages,[10] whereas deep tissue culture remains the most reliable diagnostic test (sensitivity exceeds 98%).[1]

Itraconazole is the preferred first-line therapy for *S brasiliensis*, whereas combination therapy with terbinafine and itraconazole may provide synergistic benefit in cases caused by *S schenckii*.[11]

Cryptococcosis

Cryptococcosis is most commonly caused by the dimorphic fungi *Cryptococcus gattii* or *Cryptococcus neoformans*, acquired primarily through inhalation of environmental basidiospores or desiccated yeast from

FIGURE 2. An adult female spayed domestic short-haired cat diagnosed with cryptococcosis showing **(A)** swelling of the bridge of the nose and an ulcerated nodule on the periocular region, **(B)** ulceration on the dorsal aspect of the distal paw of the left thoracic limb, and **(C)** severe extensive ulceration with crusts on the dorsal lumbar region.

Figures 2 and 3: courtesy Ben Tham, DVM, DACVD

soil, plant debris, or pigeon guano. *C neoformans* is distributed worldwide, whereas *C gattii* occurs in regions such as Western Europe, Australia, southern Africa, the Pacific Northwest, and coastal California.

The nasal cavity is the usual site of initial infection, with potential extension to the skin, eyes, or central nervous system. Affected cats often present with nasal swelling ("Roman nose"), firm cutaneous nodules, ulceration, or neurologic signs (**FIGURE 2**). Cytology frequently reveals numerous 3.5- to 20-µm oval yeasts with a thick, clear mucopolysaccharide capsule ("soap bubble") and narrow-based budding.[12] Detection of cryptococcal capsular antigen in serum or cerebrospinal fluid is highly sensitive and specific and is the preferred diagnostic test.[3]

Fluconazole is commonly administered, especially in cats with neurologic involvement, although itraconazole may be considered in refractory cases.[13]

Histoplasmosis

Histoplasmosis is caused by the dimorphic fungus *Histoplasma capsulatum*. *H capsulatum* favors tropical and subtropical climates. Areas of intense endemicity include the Ohio and Mississippi River valleys, South America, and regions of southeast Asia and India. Infection most commonly results from inhalation of airborne microconidia in soil contaminated with bird or bat guano, and even indoor-only cats may be at risk.

Disease is often systemic, with clinical signs such as lethargy, respiratory distress, or lameness, and cutaneous nodules or ulcers are reported in 16.8% of cases.[14] Urine antigen ELISA serves as a highly sensitive (90% to 94.4%) but less specific (64.6%) noninvasive screening test.[4,5] However, a positive result is not definitively diagnostic as cross-reactivity with other fungi, including *Blastomyces*, can occur. Definitive diagnosis is based on identification of small (2 to 4 µm) yeasts with a clear halo within macrophages or neutrophils on cytology or histopathology.[10] Fungal culture is generally avoided due to biohazard risk.

Itraconazole is the preferred first-line therapy.

FIGURE 3. An 8-year-old male castrated domestic short-haired cat with blastomycosis showing **(A)** extensive, deep ulceration at the medial canthus of the left eye (ulcerated nodules were also present ventral to the nasal planum) and **(B)** an ulcerated nodule with crusts on the paw pad.

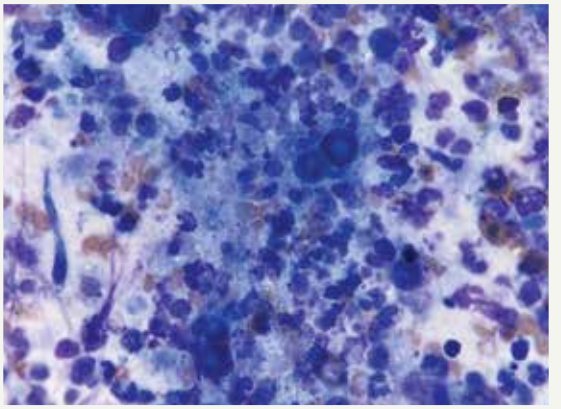

FIGURE 4. Impression smear cytology stained with Diff-Quik demonstrating *Blastomyces* yeasts and numerous degenerate neutrophils. The yeasts exhibit thick, double-contoured walls and broad-based budding.

Blastomycosis

Blastomycosis results primarily from the dimorphic fungi *Blastomyces dermatitidis* or *Blastomyces gilchristii* and, rarely, *Blastomyces helicus*. Infection occurs following inhalation of airborne conidia from moist, acidic soil, particularly in regions surrounding waterways. In North America, cases are most frequently reported in the Mississippi, Missouri, and Ohio River valleys and parts of the mid-Atlantic.

Affected cats commonly develop respiratory signs, including dyspnea and coughing, in addition to cutaneous nodules or draining tracts (**FIGURE 3**). Cytology and histopathology typically reveal large (5 to 20 µm),[10] thick-walled yeasts with broad-based budding within pyogranulomatous inflammation (**FIGURE 4**). Urine antigen ELISA is highly sensitive (93.5% to 100%) but cross-reacts with *Histoplasma*, limiting specificity.[15,16]

Itraconazole is the preferred first-line therapy.

Coccidioidomycosis

Coccidioidomycosis is caused by the dimorphic fungi *Coccidioides immitis* and *Coccidioides posadasii*. Infection follows inhalation of airborne arthroconidia from semiarid, alkaline soils in endemic regions (Arizona and south-central valley of California); notably, many affected cats are strictly indoor.[2]

Clinical signs most commonly include respiratory manifestations such as cough, tachypnea, and dyspnea. With dissemination to the skin, cats may develop nodules, nonhealing ulcers, or plaque-like crusting, especially on the trunk and distal extremities. Fever, lameness due to bone involvement, and ocular or neurologic abnormalities may also occur.

Morphologically, large (10 to 100 µm), thick, double-walled spherules containing endospores (2 to 5 µm) are observed.[10,12] However, since organisms are often sparse in samples, antibody detection using AGID is the primary diagnostic test (sensitivity 97.7% to 100%).[2,6]

Fluconazole is generally the preferred first-line therapy, although itraconazole may be favored in cases with bone involvement.

Phaeohyphomycosis

Phaeohyphomycosis is caused by dematiaceous, or melanized, fungi such as *Alternaria*, *Exophiala*, *Curvularia*, *Fonsecaea*, and neurotropic *Cladophialophora* (formerly *Xylohypha* or *Cladosporium*), among others. These organisms have a worldwide distribution. Infection typically follows traumatic cutaneous inoculation from contaminated soil or plant material.

Affected cats commonly develop solitary or multifocal nodules, ulcers, or draining tracts, often involving the nose, ears, or digits. Because of dark coloration and nodular appearance, these lesions can be misidentified as melanoma. Cytology and histopathology may identify dark-walled hyphae measuring 2 to 10 µm.[12] Fungal culture and panfungal PCR testing aid in species identification.

Management generally consists of aggressive surgical excision, when feasible, combined with prolonged systemic antifungal therapy such as itraconazole, terbinafine, posaconazole, or combination regimens.

Pythiosis

Pythiosis is caused by the aquatic oomycete *Pythium insidiosum*. *P insidiosum* is widely distributed in tropical and subtropical regions, including the Gulf Coast and southeastern United States (particularly Florida), and has also been reported across the Americas, Europe, Asia, and Australia, reflecting global presence. Infection presumptively occurs when motile zoospores in warm, stagnant water penetrate damaged skin or gastrointestinal mucosa.

In cats, disease is primarily cutaneous and locally aggressive, presenting as subcutaneous masses and draining tracts, commonly affecting the inguinal and perianal regions, tail base, limbs, or neck. Cytology and histopathology typically reveal marked pyogranulomatous inflammation with eosinophilic sleeves around broad (2 to 7 µm), irregularly branching, pauciseptate, negatively stained hyphae.[12] Definitive diagnosis relies on culture and/or PCR testing.

Treatment is challenging as oomycetes lack ergosterol, a target of many antifungals. Aggressive surgical excision with wide margins is recommended, often combined with antifungal therapies, although

supporting evidence remains limited. A combination oral regimen consisting of anti-inflammatory doses of glucocorticoids, terbinafine, minocycline, and the agricultural antioomycotic compound mefenoxam, along with hyperbaric oxygen therapy, has been reported in dogs and may be associated with improved survival.[17]

Summary

Deep fungal dermatoses in cats should be considered in cases of chronic nodular, ulcerative, or draining skin lesions, particularly when response to antibiotics is incomplete or disease worsens with immunosuppression. Geographic location, travel history, and environmental exposure are key components of risk assessment. Cytology is an essential primary diagnostic step, followed by serology, imaging, culture, molecular identification, and histopathology as needed. Accurate identification of the causative pathogen enables appropriate antifungal selection and improves prognosis through timely, sustained therapy and careful follow-up. **TVP**

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CE Quiz Questions

1. **Which diagnostic test should be performed first when evaluating nodular or draining cutaneous lesions?**
   a. Thoracic computed tomography
   b. Panfungal PCR testing
   c. Fungal culture
   d. Skin cytology

2. **Large, thick-walled yeasts (5 to 20 µm) with broad-based budding identified within pyogranulomatous inflammation are most consistent with:**
   a. *Histoplasma capsulatum*
   b. *Cryptococcus* species
   c. *Blastomyces* species
   d. *Sporothrix* species

3. **Urine antigen enzyme-linked immunosorbent assay is preferred for suspected coccidioidomycosis.**
   a. True
   b. False

4. **Which statement about treatment is most accurate?**
   a. Therapy can be discontinued once lesions resolve.
   b. Surgical excision is contraindicated in phaeohyphomycosis and pythiosis.
   c. Antifungal therapy is rarely required for > 4 weeks.
   d. Amphotericin B lipid formulations are less nephrotoxic than the deoxycholate formulation.

5. **Fluconazole is often selected over itraconazole for cryptococcosis.**
   a. True
   b. False