Blastomycosis

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Amanda J. Tschetter

Overview

- Caused by the dimorphic fungus, *Blastomyces dermatitidis* which inhabits the soil
- Infections are endemic to U.S. Southern and Southeastern states bordering the Mississippi River and Ohio River valleys, the Midwestern states and Canadian provinces bordering the Great Lakes, and areas of South Africa and Zimbabwe
- Male gender, low socioeconomic status, outdoor occupations/recreations, and diabetes are risk factors for pulmonary and disseminated disease
- Immunocompromised patients (AIDS, sarcoidosis, organ transplantation, long-term corticosteroid therapy, etc) are at risk for severe, progressive disease with early systemic dissemination, CNS involvement, relapses, and an increased mortality despite therapy
- Skin is the most common organ affected by extrapulmonary blastomycosis and lesions occur via dissemination or through primary inoculation

Clinical Presentation

- Primary pulmonary blastomycosis is the most frequent form, as most infections are acquired via inhalation, with presentations ranging from asymptomatic infection to frank pneumonia, high fevers, weight loss, chest pain, fevers and acute respiratory distress syndrome (ARDS)
- Self-resolving papules, pustules and regional lymphadenopathy can be seen in primary cutaneous blastomycosis following direct inoculation (penetrating injury with contaminated soil, laboratory accident, dog bite); multiple nodules may develop overlying infected lymphatic channels (Fig. 42.1)
- Skin lesions more frequently occur from systemic dissemination than from primary cutaneous inoculation, are present in up to 80% of cases, and may be the presenting symptom
  - Cutaneous manifestations vary greatly and include scaly papules and pustules (Fig. 42.1), ulcerated verrucous and vegetative plaques, and fungating tumors
  - Oral and nasal mucosa involvement presents with ulcers or friable lesions
  - Lesions often expand peripherally and heal with central atrophy or cribiform scarring

A. J. Tschetter, MD
University of Minnesota Health, Minneapolis, MN, USA
e-mail: tschette@umn.edu

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- Bone involvement (long bones, vertebrae, ribs, cranium, sacrum) is variable and may present as osteomyelitis and, rarely, septic arthritis; subcutaneous abscesses may develop from direct extension from infected bone
- Central nervous system dissemination (meningitis, abscess, granuloma) occurs in 5–10% of cases
- Genitourinary disease (10–30% of cases) most commonly involves the prostate, testicles and epididymis with organisms found in prostatic fluid; sexual transmission has been reported

Histopathology

- Large punch biopsy or incisional biopsy is often necessary
- *B. dermatitidis* yeasts are 8–15 μm round to oval organisms with thick, refractile double-walls that demonstrate broad-based budding (Fig. 42.2)
- Prominent pseudoepitheliomatous hyperplasia and intraepidermal pustules (Fig. 42.2) are characteristic
- Histopathologic findings depend on the time course and immune status of the patient
  - Early lesions/low immunity: neutrophils and many organisms
  - Late lesions/strong immunity: noncaseating granulomas, epithelioid histiocytes and giant cells with few organisms
- Stains: periodic acid-Schiff (PAS) and Grocott-Gomori methanamine silver (GMS) allow better visualization of organisms

Differential Diagnosis

In all cases, a travel and exposure history is essential in narrowing the diagnosis; pathologic findings are often diagnostic and biopsy should be considered if any of these entities are suspected.

- Dimorphic fungal infections (coccidioidomycosis, histoplasmosis, paracoccidioidomycosis): specific travel history is helpful, but with clinical overlap a biopsy is often necessary
- Tuberculosis: verrucous lesions of cutaneous TB can share some overlap, but biopsy will be diagnostic
- Mycobacterial infection: morphology may vary by type, but crusted nodules and plaques can clinically resemble some lesions of blastomycosis; biopsy can be helpful
- Chronic herpes simplex infection: often crusted or eroded vegetative plaques which still maintain some scalloped edging; biopsy again is often diagnostic
- Pyoderma gangrenosum: classically more of an actively inflamed, violaceous, edematous border; vegetative PG can clinically resemble blasto, but pathologic features can differentiate the two

Work-Up

- Chest radiography must be obtained in all forms to evaluate for pulmonary involvement
- Further approach and specimen collection depends on the affected organ system
  - Pulmonary: sputum or bronchial washings, biopsy
  - Bone: joint fluid, synovial tissue biopsy (imaging not specific)
  - CNS: cerebrospinal fluid (ventricular puncture more sensitive than lumbar puncture)
  - Genitourinary: urine collection (FNA for prostate)
- If there is suspicion for blastomyocosis infection the mycobacteria laboratory should be informed as there are specific culture requirements
- Visualization of characteristic yeasts forms via direct smear or histopathologic exam
- *In situ* hybridization is accurate, but not widely available

Treatment

- Primary cutaneous disease tends to resolve spontaneously without treatment
- Itraconazole, posaconazole, or voriconazole, continued for months, are appropriate treatments options
- Immunocompromised patients and those with chronic pulmonary or disseminated extrapulmonary blastomycosis: amphotericin B for 1–2 weeks followed by itraconazole for 12 months
- CNS involvement requires lipid amphotericin B for 4–8 weeks followed by an oral azole antifungal for 1+ year

Suggested Readings

1. López-Martínez R, Méndéz-Tovar LJ. Blastomycosis. Clin Dermatol. 2012;30(6):565–72.
2. Motswaledi HM, Monyemangene FM, Maloba BR, Nemutavhanani DL. Blastomycosis: a case report and review of the literature. Int J Dermatol. 2012;51(9):1090–3.
3. Saccente M, Woods GL. Clinical and laboratory update on blastomycosis. Clin Microbiol Rev. 2010;23(2):367–81.

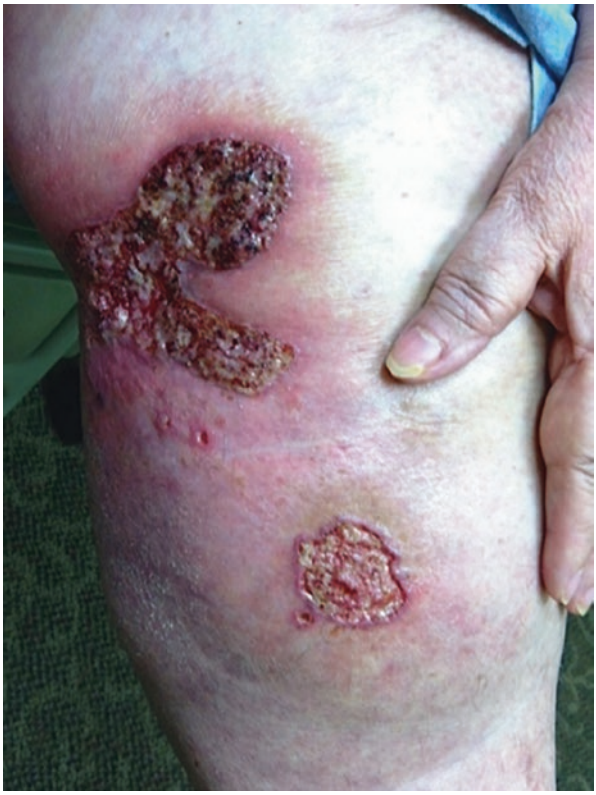

Fig. 42.1 Blastomycosis: ulcerative and verrucous plaques of blastomycosis with sporotrichoid spread along cutaneous lymphatics. Courtesy of Diane Thaler MD and Robert Rudolph MD.

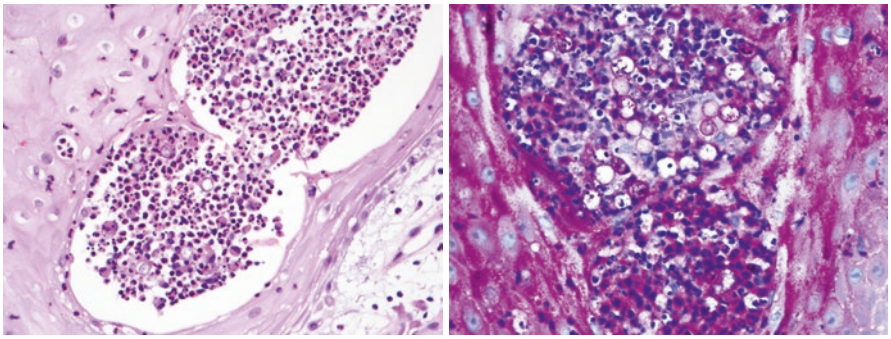

Fig. 42.2 Acute and chronic inflammation with neutrophils and histiocytes can be seen on low power. Overlying pseudoepitheliomatous hyperplasia often can be seen. *Blastomycosis dermatides* have a doubly refractile wall, broad based budding, and are approximately 10–15 μm. PAS and GMS stains highlight these organisms. Photos courtesy of Brian Swick, MD.