



BLASTOMYCOSIS - A PRIMER

by *Bill Wojciechowski, MS, RRT*

Fungi have the capacity to cause many diseases. From a respiratory standpoint some of these diseases occur when fungal spores are inhaled and proliferate in the lungs of humans. An interesting fact about fungi is they cause disease as a parasite, but the parasitic phase is not essential to their life cycle. They are first and foremost saprophytes, (organisms that thrive on decaying organic matter). They become parasites only if their spores encounter an environment suitable for that life form.

Medically, four types of fungal infections, called mycoses, occur. They are (1) cutaneous mycoses, (2) dermatomycoses, (3) subcutaneous mycoses, and (4) systemic, or deep, mycoses. Systemic mycoses infect internal organs and disseminate throughout the body, often proving fatal.

Blastomycosis is a rare infection caused by inhaling fungal spores, or blastospheres, produced by the microorganism *Blastomyces dermatitidis*, which is found in decomposing wood and soil laden with decaying organic material. Persons who are most prone to contract this endemic disease are those with exposures to wooded sites and areas featuring thick vegetation. People most susceptible tend to include farmers, forestry workers, hunters, and campers.

Before antifungal drugs were available, mortality rates associated with fungal infections were close to 60%

means that this fungus can thrive in two different environments at two different temperatures, i.e., saprophyte and parasite. First, as a saprophyte, *B. dermatitidis* lives in rotting wood and in soil enriched with decaying organic matter. If the soil contains rich organic debris such as animal feces, plant fragments, insect remains, and dust, and if the substrate is moist, lacks exposure to direct sunlight, and has a pH below 6.0, isolation of *B. dermatitidis* is likely. Room temperature (~25°C) supports the growth of this fungus in the saprophytic phase

As *B. dermatitidis* exists as a saprophyte, it develops extensive long, branched, tubular filaments called hyphae. This structure can be likened to a large collection of "monkey bars," as seen in a park, but on a microscopic level. These hyphae form spores, or blastospheres, which can become airborne when the soil is disturbed. Once these fungal spores are airborne, people and other mammals are subject to inhaling them. When these spores are exposed to an environment with a temperature around 37°C, they

Microbiology

Blastomyces dermatitidis is a thermally dimorphic fungus. Thermal dimorphism

develop into the parasitic phase in the form of yeast buds. Thick cell walls of the yeasts provide resistance to phagocytosis by alveolar macrophages and polymorphonuclear leukocytes. Then, the yeast forms multiply and disseminate through the blood and lymphatics to other organs. The evoked inflammatory response causes an initial influx of neutrophils, followed by macrophage and granuloma formation.

Interestingly, hunting dogs can contract canine blastomycosis. These animals have their noses close to the ground and can inhale fungal spores while tracking or retrieving game.

Epidemiology

Dimorphic systemic mycoses generally have regional environmental preferences. Most clinical cases of blastomycosis are reported in states surrounding the Mississippi and Ohio rivers and the Great Lakes region. Microfoci are also found in Central and South America and parts of Africa. Blastomycosis has also been reported in parts of Canada, India, Africa, and Central and South America. Because of the erroneous belief that the disease is limited only to the United States, blastomycosis is often referred to by the term North American blastomycosis, which is an obsolete term.

Men acquire blastomycosis more than women only because men tend to hunt, farm, and camp more than women. Nonetheless, studies have revealed no predilection for this disease based on sex, age, or race. The same tendency is true when adults and adolescents are compared.

Diagnosis

The diagnosis of blastomycosis can be confirmed by the isolation of *B. dermatitidis* in a culture of the sputum, saliva, skin, bronchial washes, lung biopsy, or biopsy of infected tissue. Blood specimens may also be used to determine if a person had a preceding *B. dermatitidis* infection. However, blood tests will not identify all cases and on occasion may be falsely positive. Similarly, skin testing is not accurate in diagnosing blastomycosis, nor is serology.

Sign & Symptoms

About 50% of infections are asymptomatic. Many infections resolve without treatment. In fact, most of these people are completely unaware they have been exposed to and infected with *B. dermatitidis* spores. Immunocompetent hosts have a natural resistance to infection with *B. dermatitidis* because alveolar macrophages inhibit the transformation of blastospores into yeast. Such natural resistance has been demonstrated in clinical studies of outbreaks of blastomycosis in which asymptomatic infection

occurs in at least 50% of persons in whom *B. dermatitidis* has colonized. A smaller percentage develops an acute lung infection that begins with fever and dry cough which progresses to a persistent productive cough, weight loss, and chest pain. These signs or symptoms and the infection may disappear spontaneously without treatment.

An even smaller percentage of those infected with *B. dermatitidis* demonstrate these flu-like signs and symptoms, as well as fever, chills, night sweats, myalgia, arthralgia, and general malaise. Some of these patients fail to recover and exhibit chronic pulmonary infection which can last anywhere from 2 to 6 months, while others develop widespread disseminated infection. The disseminated form affects the skin, bones, genitourinary tract, and the central nervous system. After initial exposure to the spores, symptomatic persons generally exhibit clinical manifestations between 3 and 15 weeks.

Treatment

Amphotericin B is the mainstay drug for treating fungal infections. Amphotericin B has negligible gastrointestinal absorption; therefore, is only available in the intravenous route of administration. Discovered in 1956, it is derived from the bacterial genus *Streptomyces*. The mechanism of action is to disrupt the fungal cell membrane by interacting with ergosterol a fungal membrane component. Mammalian cell membranes are devoid of ergosterol, but possess cholesterol. Amphotericin B therefore will interfere with mammalian cell membranes producing serious side effects, which can include renal toxicity, increase in bilirubin, erythema, and infusion-related symptoms, such as fever, chills, and severe vomiting. Despite these severe side effects, they seem to be dose-dependant, and lessen or disappear with decreased doses of amphotericin B. Known by the trade names Abelcet, Ambisome, and Amphotec are lipid formulations of amphotericin B. Their purpose is to deliver to the infected person a formulation that has a lower toxicity level, but does not compromise the efficacy of amphotericin B. The liposomes seem to sequester amphotericin B in a manner which makes it unavailable for mammalian cells, but maintains its access to fungal cells. Patients treated for systemic mycoses such as blastomycosis often take amphotericin B for months.

The other class of antifungal medications is the azoles, e.g., itraconazole and keto-



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conazole, which also targets ergosterol, but it does so by inhibiting an ergosterol precursor, i.e., lanosterol. The azoles, taken orally, are equally effective for treating the acute and chronic forms of blastomycosis. However, they are not beneficial for the treatment of this infection when it involves the central nervous system. Amphotericin B is then the drug of choice. Essentially, the azoles are not used for life-threatening mycoses. Therefore anytime their efficacy appears compromised, amphotericin B is substituted. Patients treated for deep mycoses may be required to take itraconazole for as long as two years.

Prognosis

Antifungal medications have drastically reduced the mortality rate associated with blastomycosis to around 5%. Before antifungal drugs were available, mortality rates were close to 60%. In contrast, one third of immunocompromised patients do not respond to the therapy, and 30% to 40% die of this mycosis. Most of these deaths occur within the first few weeks of therapy. Hematogenous spread produces painless cutaneous lesions in more than 70% of patients. These lesions frequently affect the face, upper limbs, neck and scalp.

Avoidance

People who live in geographical areas endemic to certain fungal spores may not be able to completely avoid exposure to the fungus. However, those who are immunocompromised may consider avoiding (1) wooded areas to evade *Blastomyces dermatitidis*, (2) semi-arid and arid regions to elude *Coccidioides immitis*, and (3) Ohio, Mississippi, and St. Lawrence River valleys to circumvent *Histoplasma capsulatum*. Avoiding travel to an area where infection is known to prevail may help prevent exposure to a particular fungus, but may not always be possible.

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