Case Report

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Disseminated Blastomycosis in a Local Farmer from Himachal Pradesh, North India: A Diagnostic Dilemma

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Blastomyces dermatitidis has been isolated only infrequently from environmental sources and thus the ecology of *B. dermatitidis* remains incompletely understood. Other methods of detection of prevalence are also insensitive. Our knowledge is based on the collected reports of sporadic cases in humans and dogs as well as the studies of 11 epidemics or clusters of disease. It can mimic many diseases and may remain undiagnosed till late. In disseminated infections, without treatment mortality remains high (78%). Most cases reported from India are imported from USA. We are reporting a case of disseminated blastomycosis acquired in North India in a native farmer. Its diagnosis remained a challenge in our laboratory setting.

Keywords: Disseminated blastomycosis, Disseminated gonococcal infection, India

INTRODUCTION

Blastomycosis is an uncommon, geographically restricted, systemic mycoses, caused by the thermally dimorphic fungus, *Blastomyces dermatitidis*. Its endemic areas are North America and Africa along the river basins. In India, there are only few well-documented cases though it is strongly suspected to be endemic.[1,2] Its areas of environmental distribution and prevalence remain undetermined. Here, we are presenting a case of disseminated blastomycosis in a native farmer from Himachal Pradesh, North India and challenges encountered in a routine microbiology lab in reaching the diagnosis.

CASE REPORT

A 65-year-old farmer, a widower for 15 years, living in the surroundings of Satluj River in the Mandi District (Himachal Pradesh) presented in the Dermatology Outpatient Department with:

1. Painful joints with low-grade fever, off and on - 3 months.
2. Pustules over face and extremities - 5 days.
3. Painful swelling of scrotum with pus discharge - 5 days.
4. Whitish discharge after micturition - 5 days.

Clinically disseminated gonococcal infection (DGI) was suspected, though there was no history of any sexual contact in past 15 years. Patient had never travelled abroad. On examination, there were papulopustular lesions on the exposed parts of body (Figure 1) and the scrotum had typical "water can perineum" appearance with multiple sinuses. Crepts were present over right lower part of chest. Abdomen and chorionic villus sampling had normal findings.

Swabs from pustules, scrotal pus discharge and urine sample were submitted for microscopy and culture for gonococci. Gram stain and its modification with neutral red as counter stain were used to stain smears from all the samples. All samples were inoculated on blood agar, chocolate agar, MacConkey agar, and Mueller-Hinton agar. They were incubated in a candle jar at 37°C.

Smears were negative for gonococci. However, all samples showed broad based budding yeast cells with double walls. These structures were initially overlooked by reporting residents as juniors kept looking only for gonococci and seniors presumed it to be a stain contamination with yeast.

After 48 h of incubation, cultures showed white, creamy, smooth colonies on blood, and chocolate agars. There was

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no corresponding growth on MacConkey agar. Mueller-Hinton agar was used to isolate gonococci, but an off white, filamentous, spiky, confluent fungal growth was obtained after 4 days. It was sent to mycology lab for identification where it was accidentally discarded presumed to be fungal contamination.

From scrotal pus discharge a mixed growth of *Proteus vulgaris* and *Enterobacter* spp. was isolated and sensitivity was reported.

On smears, broad based budding yeast cells (Figure 2) were picked up by a consultant and simultaneously culture isolates also showed presence of yeast cells. Results were correlated and further isolates were processed on sabouraud dextrose agar (SDA) and blood brain heart infusion agar to demonstrate thermal dimorphism (Figure 3).

At room temperature growth was dry, wrinkled and spiky again (left culture bottle in Figure 3) and showed transitional forms, i.e., both filamentous and yeast forms (Figure 4). Growth was pure but spores and pure filamentous growth was not obtained despite several attempts.

Growth was preserved in several vials of SDA. It was sent to Department of Microbiology, PGIMER, Chandigarh, India, (reference center) where it could not get recovered after 2 months of preservation and molecular identification could not be done.

Patient eventually developed features of the central nervous system and eye involvement. Yeast cells were detected from cerebrospinal fluid (CSF), soft palate lesion, nasal scraping, and sputum. X-ray chest also showed infiltrations. HIV test was negative.

Patient remained admitted for 3 weeks. He was given Amphotericin B intravenously. He developed side effects of high fever with shaking chills. His serum creatinine levels also rose significantly. Finally, he developed disseminated intravascular coagulopathy and became LAMA.

DISCUSSION

B. dermatitidis grows in geographically restricted microfoci in proximity to water bodies associated with soil rich in organic matter in shaded areas. Foggy weather helps in release of microconidia which are infective forms and

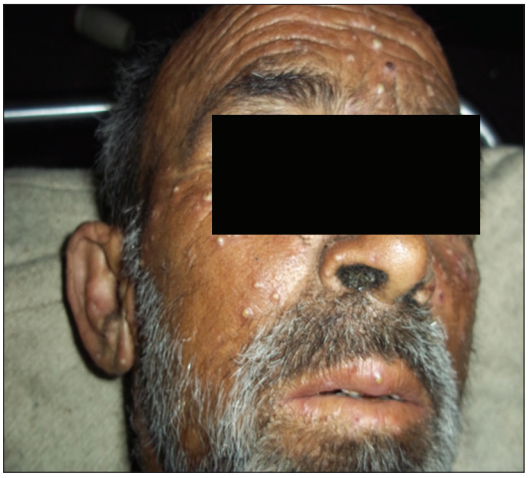

Figure 1: Disseminated papulopustular lesions over face

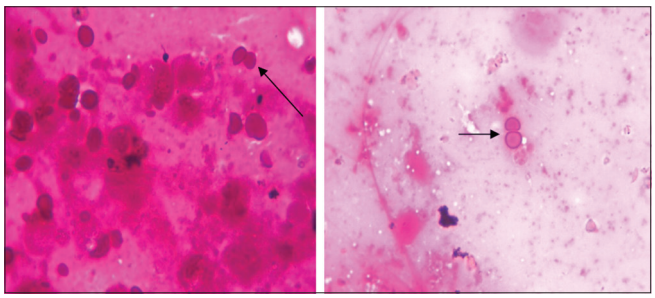

Figure 2: Thick walled broad based budding yeast cells in modified Gram stain

Figure 3: Showing thermal dimorphism of growth

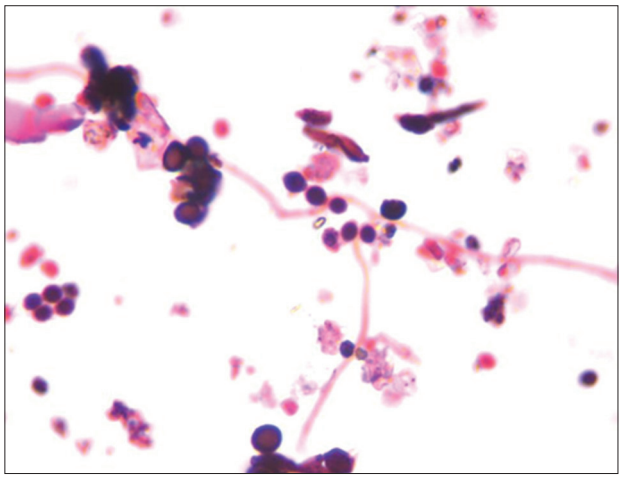

Figure 4: Transitional forms showing both hyphae and yeast cells on culture at 25°C

inhaled by the host.[3] Our patient also belonged to similar climate and lived near river Satluj in Himachal Pradesh.

Systemic infection occurs by inhalation of mycelial fragments and conidia and localized cutaneous infection by direct inoculation of skin.[4] The incubation period is 4-8 weeks and 1-5 weeks, respectively.[3]

Epidemiological tools like use of skin test surveys, selective media for isolation from soil and serological studies available for other systemic mycoses are not effective against blastomycosis.[5] Recent application of polymerase chain reaction (PCR) for rapid detection of *B. dermatitidis* in clinical and soil sample is highly promising.[1]

It is a systemic pyogranulomatous infection, primarily involving lungs. Disseminated involvement is common for skin, bones and genitourinary system, but almost any organ can be infected. Skin disease is the most common extra pulmonary manifestation and a marker for multiorgan infection.[5] Skin and mucosa involvement is often misdiagnosed as squamous cell carcinoma. Pulmonary disease may be acute or chronic and mimics infection with pyogenic bacteria, tuberculosis, other fungal infections, sarcoidosis, and malignancy.[5,6] Canine blastomycosis can be a harbinger of disease in humans. Majority of cases are sporadic or endemic, epidemics associated with exposure to common outdoor source are documented.[4] Genitourinary involvement is also seen in cryptococcosis and pneumocystis.[6] DGI occur mostly in women and are seen as hemorrhagic papules and pustules with purpuric centers in a centrifugal distribution. There is lower incidence of DGI at present in comparison to 1970s attributable to a decline in the particular strains that are likely to disseminate (PorB.1A serotype, highly susceptible to penicillin, and AHU auxotype).[7]

Microscopically, the single bud with a wide base and a thick wall is pathognomonic of blastomycosis. The yeast cells when found without buds, may be confused by less experienced laboratorians with the immature spherules of *Coccidioides immitis* lying next to each other, the yeast forms of *Histoplasma capsulatum* var. *duboisii* with microforms of *B. dermatitidis*, *Cryptococcus neoformans* with absent capsule or an unbudded yeast cell of *Paracoccidioides brasiliensis*.[3] Several staining methods and searching more definitive forms can help but morphology is presumptive method of identification and the specific nucleic acid probe is confirmatory.[3,4]

Culture isolation and thermal dimorphism are important for diagnosis. Many strains do not convert completely and show transitional forms.[3] They are slow growing (10-30 days) except in heavy infection (1 week).[6] Some molds such as *Pseudallescheria boydii* and *Chrysosporium* sp. have similar appearance but they do not grow on media containing cycloheximide and later does not grow at 37°C. Several means of verifying the identity of *B. dermatitidis* have been developed: Exoantigen test, deoxyribonucleic acid probe, PCR, sequencing, and deterministic finite automata.[4] Antigen detection in urine/CSF/serum/bronchoalveolar lavage is promising.[6]

CONCLUSION

Our experience makes us conclude that clinical opinion helps a microbiologist in investigating the sample properly but it should not narrow the vision and approach to the sample processing and its outcome. White molds should be processed carefully in a biosafety cabinet. They should not be ignored as contaminants. Preservation of cultures should be done appropriately to investigate them in future. A high index of clinical suspicion coupled with pro-active mycological investigation is likely to reveal the occurrence of many more autochthonous cases of Blastomycosis in India.

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