Cerebral Pseudallescheriasis Due to Pseudallescheria boydii as the First Manifestation of AIDS

Pseudallescheria boydii is a saprophytic fungus that is present in moist soil, natural fertilizers, and decomposing vegetation [1]. Clinical entities associated with P. boydii range from subcutaneous cumuli or suppurative chronic infections (mycetomas) [2] to a wide variety of conditions known as pseudallescheriasis. P. boydii colonizes paranasal sinuses, and invasive infections that extend to neighboring structures such as the eye and meninges have been reported [3–6]. Severe pseudallescheriasis has been described in immunocompromised patients [7, 8] and is a very rare complication of AIDS. We report an HIV-positive patient with multiple cerebral lesions due to P. boydii as the first manifestation of AIDS.

A 33-year-old, HIV-positive man was admitted to the hospital for evaluation of generalized tonic-clonic seizures. He had a 20-day history of intense headaches and cognitive decline during the last 72 hours. He was confused and agitated. His speech was slurred, and there was spontaneous movement of his upper and lower extremities. Babinski’s sign was noted bilaterally. There was no history of opportunistic infections. Laboratory studies revealed the following values: hematocrit, 37%; WBCs, 4,000 × 10^6/L; CD4⁺ cell count, 37 × 10⁶/L. Evaluation of CSF revealed WBCs, 5 × 10³/µL; protein level, 1.20 g/dL; and a highly reactive Pandy test. CSF was negative for bacteria and fungi on direct examination. A latex test for antibodies to Cryptococcus species was negative, as were molecular studies for Epstein-Barr virus, Toxoplasma gondii, and cytomegalovirus. A head CT scan showed multiple, non-contrast-enhancing hypodense lesions in the basal ganglia, pons, and subependymal area, with marked edema of the right frontoparietal area causing collapse of the basal cisterns and ventricular system.

Empirical treatment with antitoxoplasma agents and corticosteroids was instituted. The patient’s condition deteriorated, and 48 hours later he required mechanical ventilation. On the fifth day he went into a profound coma with bradycardia and hypotension. He died 24 hours later. Cultures of CSF, performed on the first and third day yielded P. boydii. In addition, the organism was isolated in cultures of brain tissue obtained by postmortem cranial trephination.

Severe cases of pseudallescheriasis have been reported in immunocompromised patients without AIDS undergoing immunosuppressive treatment and those with leukemia, lymphoma, lupus, or Crohn’s disease [3]. In these patients, progressive local invasion from colonized sinuses may precede CSF involvement. Reports of P. boydii as the causative agent of systemic infections in patients with AIDS are rare [9], but the spectrum of fungal sinusitis agents reported during HIV infection includes P. boydii [10]. In our patient, the paranasal sinuses were the most likely source of cerebral P. boydii involvement, mainly because blood cultures were negative for fungi.

P. boydii caused the first opportunistic infection in our patient. This underscores a growing problem: fungi are emerging as new opportunistic infectious agents in significant numbers, and most of these agents are completely unfamiliar to clinicians. P. boydii must be included in the expanding spectrum of life-threatening opportunistic pathogens affecting the brain, and clinicians should be aware that it can be an initial manifestation of AIDS. Because P. boydii grows in most culture media including Sabouraud dextrose agar, the diagnosis of cerebral pseudallescheriasis is relatively easy.

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Isolated Pleural Effusion with Pleural Fibrosis in a Patient with Subacute Progressive Disseminated Histoplasmosis

Progressive disseminated histoplasmosis (PDH) is an infrequent and serious complication of infection due to *Histoplasma capsulatum*. Known to occur in both acute and subacute-to-chronic forms, PDH manifests as a systemic illness with organ dysfunction resulting from unregulated fungal growth. Pulmonary involvement is variable in PDH. Pleural effusions are uncommon and usually occur in conjunction with pulmonary infiltrates or nodules, adenopathy, and/or subpleural infection [1–2], and in acute PDH. Isolated pleural disease is rare, particularly in patients with chronic disease. We describe a patient with subacute PDH who presented with isolated pleural effusion and fibrosis linked ultimately to T cell lymphoma.

A 55-year-old Puerto Rican man who had never smoked presented to an outside institution for evaluation of right-sided chest pain and dyspnea of 1 year’s duration. Prior evaluation consisted of a chest radiograph and a chest CT scan that demonstrated minimal right-sided pleural thickening and effusion without lymphadenopathy or parenchymal abnormalities. Evaluation of a specimen obtained on thoracentesis revealed a bloody exudate consisting predominantly of lymphocytes. Cytologic studies were negative, as were smears for acid-fast bacilli (AFB) and cultures. No fungal studies were performed.

The patient’s medical history included retinal hemorrhage, which was not believed to be related to PDH. A review of systems was remarkable for bilateral ankle edema and decreased visual acuity. His travel history was notable for an 18-month stay in Puerto Rico 3 years before presentation. He had worked for 17 years on an automobile assembly line in Ohio. There was no history of exposure to birds.

On examination the patient appeared chronically ill, but his vital signs were normal. Fundoscopic examination revealed multiple branch artery occlusions. Breath sounds were diminished at the right lung base, and there was bilateral lower-extremity pitting edema.

A repeated chest radiograph and chest CT scan (figure 1) were unchanged from previous films. Findings on an abdominal CT scan were normal. Routine laboratory studies revealed a hemoglobin level of 6.9 g/dL and normal platelet and WBC counts. An autoimmune profile and serology for antibodies to HIV were negative. Thoracoscopic pleural biopsy pathology was nondiagnostic, but tissue culture yielded *H. capsulatum*. Examination of a bone-marrow biopsy specimen revealed granuloma formation; however, special stains and cultures for the fungus were not completed.

Treatment with itraconazole was associated with abatement of malaise and with weight gain over the next month and stability in the patient’s condition for 3 months. By 5 months, however, there were new symptoms of fatigue, weight loss, chills, and night sweats. On examination, there was new anterior cervical and inguinal lymphadenopathy, diffuse abdominal pain, and significant lumbosacral spinal tenderness. A repeated CT scan of the abdomen demonstrated multiple new masses in the liver and spleen, as well as ascites, retroperitoneal lymphadenopathy, and lytic lesions of the lumbar vertebrae. Histopathologic evaluation of liver and bone-marrow biopsy specimens revealed infiltration by small noncleaved lymphocytes, ultimately diagnostic for T cell lymphoma. The patient died shortly thereafter of multiorgan failure despite chemotherapy and itraconazole treatment.

As a native of Puerto Rico and resident of Ohio, this patient lived and worked within some of the areas where histoplasmosis is most endemic. The vast majority of such exposed individuals are able to regulate infection, presumably because of normal T cell–mediated immunity [3]. In this case, however, occult lymphoma likely predisposed the patient to PDH. Although lymphoma may have been responsible for many of the patient’s symptoms, the clearcut clinical improvement associated with itraconazole treatment suggests that PDH contributed as well.

Pleural effusion and thickening occur frequently in cases of histoplasmosis, particularly as isolated findings. When effusions do...