



roentgenogram of the month

Cavitary Lung Lesions in an Immunosuppressed Child*

Helen T. Winer-Muram, MD, FCCP; Sergio Vargas, MD;
and Karen Slobod, MD

(*Chest* 1994; 106:937-38)

An 11-year-old boy, diagnosed 3 years earlier with acute lymphoblastic leukemia, presented in May 1993 with a hematologic relapse. One week after starting high dose multiagent chemotherapy, he developed diffuse pulmonary opacification with respiratory failure requiring mechanical ventilation. Lung biopsy performed after two nondiagnostic bronchoscopies revealed diffuse alveolar hemorrhage and necrotizing bronchiolitis. The patient recovered spontaneously; mechanical ventilation was discontinued, and the chest radiograph returned to normal.

In June, he developed a temperature up to 40°C and was neutropenic (absolute neutrophil count <500) as a result of chemotherapy. On physical examination, he was a chronically ill, emaciated child. The lungs were clear to auscultation. The following day, multiple small nodular erythematous skin lesions appeared, which were thought to be embolic. These lesions were biopsied. Echocardiogram examining the cardiac valves and the tip of the central line was normal. Blood cultures from the central line grew *Staphylococcus epidermidis* and the line was removed. The patient was receiving appropriate broad spectrum antibiotics. Amphotericin B and fluconazole were empirically added. The chest radiograph showed new ill-defined nodular opacities at the right upper and lower lobes (Fig 1).

In July, as the neutropenia resolved (ANC >500), the chest radiograph revealed enlargement of the opacities and development of air crescents (Fig 2). Computed tomography (CT) of the abdomen showed a 3-mm solitary hypodense renal lesion (not shown). As pulmonary cavitation manifested, the patient had two episodes of mild hemoptysis which resolved with platelet transfusions and cough suppressants.

*From the Departments of Diagnostic Imaging (Dr. Winer-Muram), and Infectious Diseases (Drs. Vargas and Slobod), St. Jude Children's Research Hospital, Memphis Tennessee, and the Department of Radiology (Dr Winer-Muram), University of Tennessee, Memphis.

Supported in part by National Cancer Institute Cancer Center Support (Core) grant P30CA21765, CA 23099, and by the American Lebanese Syrian Associated Charities (ALSAC).

Reprint requests: Dr. Winer-Muram, St. Jude Children's Research Hospital, Diagnostic Imaging, 332 N. Lauderdale, Memphis, TN 38101

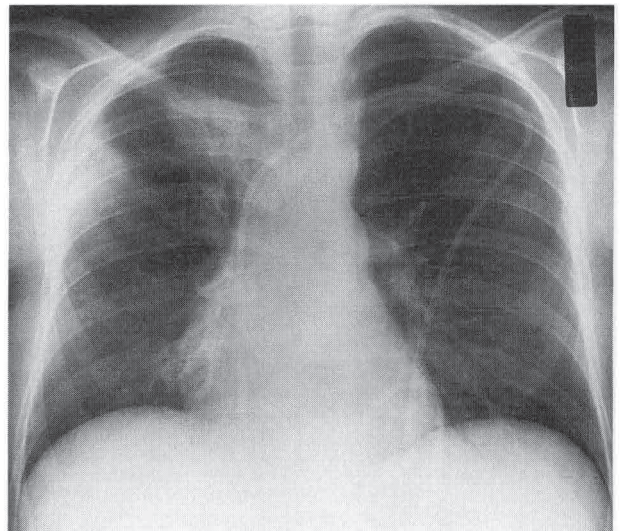


FIGURE 1

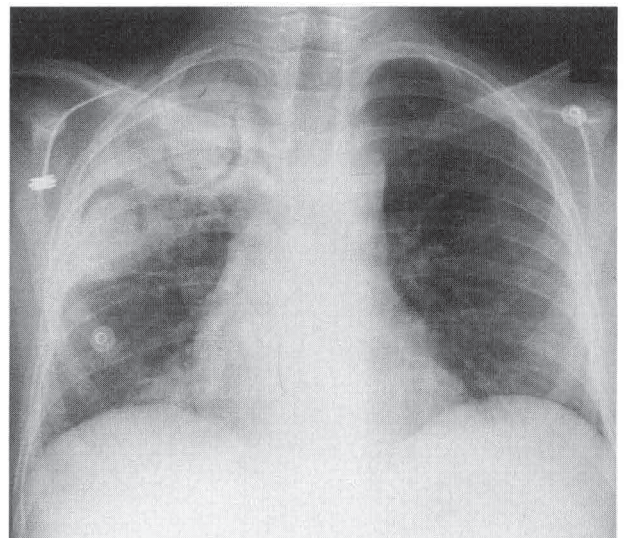


FIGURE 2

Diagnosis: Disseminated *Pseudallescheria boydii* involving lung, kidney, and skin. The organism was isolated from the skin biopsy.

The chest radiograph and CT scan obtained during the period of neutropenia demonstrate multiple bilateral ill-defined nodular opacities (Figs 1 and 3). The large right opacity has a peripheral rim of ground glass opacification, known as the "halo sign" (Fig 3). This halo is believed to represent the peripheral rim of hemorrhagic infarction, described in the angioinvasive fungal diseases, aspergillosis, mucormycosis, and *Pseudallescheria boydii* infection.¹

As the patient's absolute neutrophil count increased (ANC >500), the nodules developed air crescents and cavitation (Fig 2). This is believed to occur as a result of retraction of the central infarcted region from the periphery of the lesion. White blood cells are recruited to resorb necrotic tissue at the periphery of the lesion and the space between the dead tissue and periphery fills in with air, resulting in the air crescent. Since resorption of the periphery of the sequestrum requires neutrophils, it is not surprising that air crescent formation is a late radiographic finding that coincides with recovery of the white blood cells. Eventually, the air crescent may transform into a cavitory space, filled with necrotic debris and fungus.¹

Air crescents also may be seen rarely in cavitory bacterial infection, such as moraxella pneumonia. Occasionally, neoplasms such as squamous cell carcinoma may cavitate in an asymmetric fashion causing an air crescent. Air crescents may be seen in cavities partially filled with mycetoma, necrotic tumor, or blood clot. Such air crescents will change with repositioning of the patient since the cavity contents are mobile. Mycetomas are usually due to saprophytic overgrowth of *Aspergillus* species in a preexisting cavity from remote tuberculosis or sarcoidosis. Rarely mycetomas due to *Nocardia*, *Candida*, *Streptomyces*, *Phycomycetes*, *Coccidioides*, *Pseudallescheria* or *hydatid* have been described.^{2,3}

Pseudallescheria boydii, formerly known as *Petrellidium boydii*, is a ubiquitous fungus found in soil, gaining entry to the body either by inhalation of ascospores or by traumatic inoculation into the skin. Invasive pneumonia usually occurs in immunocompromised hosts and dissemination is common to lungs, brain, kidneys, heart, skin, and eyes. Survival is rare in disseminated disease.⁴

Radiographically, invasive pulmonary disease in

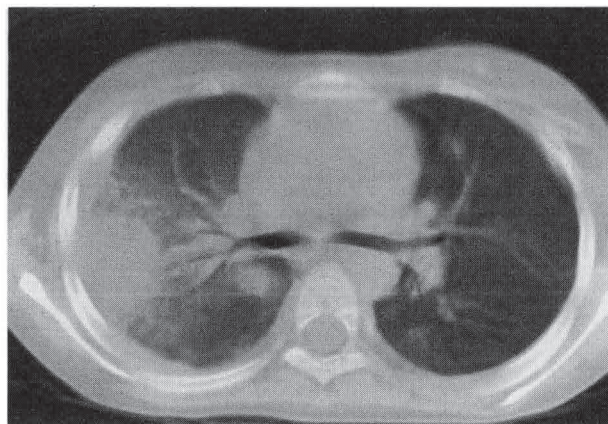


FIGURE 3

immunosuppressed patients manifests as consolidation, nodules, necrotizing pneumonia, pulmonary abscesses, mycetomas, and pleural effusions. In immunocompetent patients, pulmonary involvement manifests as mycetomas in preexisting cavities, asymptomatic coin lesions, or as allergic bronchopulmonary disease.^{4,5}

Histopathologic examination may not distinguish *P boydii* from *Aspergillus* species since both organisms have thin, septate hyphae with dichotomous branching. *P boydii* can be identified by its unusual ability to form conidia in infected tissue or in *in-vitro* cultures. The differentiation is important because characteristically *P boydii* is resistant to amphotericin B, while administration of imidazoles, such as miconazole, improves therapeutic results.⁶ Surgical resection of infected tissues when possible, is the preferred treatment.⁴

REFERENCES

- 1 Kuhlman JE, Fishman EK, Siegelman SS. Invasive pulmonary aspergillosis in acute leukemia: characteristic findings on CT, the CT halo sign, and the role of CT in early diagnosis. *Radiology* 1985; 157:611-14
- 2 Geftter WB. The spectrum of pulmonary aspergillosis. *J Thorac Imag* 1992; 7(4):56-74
- 3 Chaudhary BA, McAlexander D, El Gammal T, Speir WA. Multiple mycetomas due to *Pseudallescheria boydii*. *S Med J* 1987; 80(5):653-54
- 4 Thompson GP, Roberts GD. *Pseudallescheriasis*. In: GA Sarosi, SK Davies, eds. *Fungal diseases of the lung*, second edition. New York: Raven Press, 1993; 205-215
- 5 Lake FR, Tribe AE, McAleer R, Froudust J, Thompson PJ. Mixed allergic bronchopulmonary fungal disease due to *Pseudallescheria boydii* and *Aspergillus*. *Thorax* 1990; 45(6):489-91
- 6 Enganno IL, Hughes WT, Kalwinsky DK, Pearson TA, Parham DM, Stass SA. *Pseudallescheria boydii* in a patient with acute lymphoblastic leukemia. *Arch Pathol Lab Med* 1984; 108:619-22