Spinal Epidural Aspergillosis in a Patient With HIV Resulting From Long-Standing (3 Years) Lung Infection

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Case Report

A 39-year-old man was diagnosed in 1997 with HIV. He was started on stavudine (Zerit) and lopinavir and ritonavir (Kaletra) with good control of his disease. In May 2003, his white blood cell (WBC) count was 5800 cells/μL with 58% neutrophils (reference range >34%). CD4 count was 507 (reference range >400 cells/μL) with a quantitative serum HIV count of less than 20. In September 2003, he presented with cough and chest pain. Plain films and chest CT revealed a right upper lobe mass without definite chest wall or mediastinal invasion. He had a WBC count of 9200 with only 5.8% neutrophils and an absolute neutrophil count (ANC) of 0.5 (normal >1.6). Results of 2 CT-guided percutaneous biopsies revealed only chronic inflammation and fibrosis. Results of an open biopsy in January 2004 revealed fibrosis and “ribbonlike” fungal elements with occasional septations, for which he was given intravenous amphotericin B and, later, oral voriconazole for presumed aspergillosis.

Discussion

Invasive aspergillosis has not traditionally been considered a complication of well-controlled HIV infection. Host defense against the Aspergillus species is considered to be a function of neutrophils. As such, invasive aspergillosis is relatively uncommon in human immunodeficiency virus (HIV), a disorder of lymphocyte function. We describe a case in which a man with well-controlled HIV presented with invasive pulmonary aspergillosis with cord compression. We will provide several reasons why invasive aspergillosis is thought to occur in patients with HIV, as well as describe the MR imaging features of this case, which can serve as important diagnostic clues.

Summary: We present an unusual case of a man with human immunodeficiency virus (HIV) with pulmonary aspergillosis and spinal invasion and compression of the spinal cord occurring during a long period (3 years), as documented by MR imaging and surgical intervention. Invasive pulmonary aspergillosis with cord compression has been reported in the past, but, to the best of our knowledge, none of these have been in a patient with HIV.

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that period, was asymptomatic. At the time of initial presentation, however, he was found to have significant neutropenia with only 5.8% neutrophils and an ANC of 0.5 (normal 1.6). This finding correlates with the hypothesis that host defense against the Aspergillus species is at least, in part, because of neutrophil function. It is not known why the patient became neutropenic, and it is not fully understood why the patient had progression of disease despite subsequent maintenance of normal neutrophil counts, treatment with appropriate antifungal therapy, and maintenance of CD4 counts well above 200.

Finally, the presence of isointensity to low intensity on T2-weighted images in the lesion is of interest. A study by Herold et al. described the “targetlike” appearance of pulmonary nodular infiltrates, with low signal intensity centrally and increased intensity peripherally on both T1- and T2-weighted images. The central low signal intensity was attributed to both central cavitation and coagulative fungal necrosis, whereas the rim of increased signal intensity was attributed to subacute hemorrhage or hemorrhagic infarction. A study by Kawashima et al. reported MR imaging findings in 2 patients with chronic granulomatous disease, pulmonary aspergillosis, and invasion into the chest wall. In both cases, the lesions were increased in T2 signal intensity. Finally, a case report by Fujimoto et al. described low T2 signal intensity of an aspergilloma located within a cavitary lung neoplasm that showed high T2 signal intensity. In our patient, the areas of pulmonary consolidation and invasion into the chest wall were uniformly low or isointense on T2-weighted images. On pathologic examination, the resected epidual mass was found to have large amounts of attenuated connective tissue with areas of both acute and chronic inflammation. We postulate that good control of HIV in this patient allowed for production of an effective immune response, thereby producing chronic inflammatory changes and fibrosis. The fibrosis, in turn, may account for the decreased T2 signal intensity. Furthermore, the presence of paramagnetic and ferromagnetic elements intrinsic to the fungi may contribute to the T2 hypointensity.

In summary, invasive aspergillosis is felt to have a generally poor outcome. As such, early diagnosis and treatment are imperative to survival. The radiologist should be aware of the imaging characteristics and clinical presentation of invasive aspergillosis in patients with HIV. We conclude that epidural aspergillosis should be considered even in the setting of well-controlled HIV and that the isointense to hypointense signals on T2-weighted images are a helpful diagnostic clue.

References
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