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## Disseminated CNS Histoplasmosis

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Although an estimated 40 million Americans have been infected with *Histoplasma capsulatum* [1], CNS involvement is rare except in the disseminated form. Lesions of the CNS have been reported to occur in nearly one fourth of the documented cases of disseminated histoplasmosis [2], but only in a few cases has the infection solely involved the CNS [3, 4]. This article details the cranial CT and spinal MR evaluation of a patient with multiple biopsy proved histoplasmosis lesions in the brain and spinal cord.

### Case Report

A 57-year-old man presented with a 3-month history of progressive lower extremity paresthesias, weakness, and bowel and bladder dysfunction. At the time of admission, he was so weak as to be unable to walk. His medical history was unremarkable except for pulmonary tuberculosis treated 2 years previously. At that time, an unenhanced brain CT scan was normal.

A physical examination on admission revealed mild cachexia with normal vital signs. The patient was alert and oriented, his cranial nerves were intact, and motor and sensory functions in the upper extremities were normal. In the lower extremities, the patient had only trace voluntary movement in the adductor muscles bilaterally and in the quadriceps muscles on the right. There was a sensory level at approximately L2 with some sacral sparing. Deep tendon reflexes were 3+ at the knees and 1+ at the ankles. Rectal sphincter tone was decreased.

White blood cell count and differential were normal. Lumbar puncture revealed a glucose of 56 mg/dl (normal range = 40–70 mg/dl) and protein of 107 mg/dl (normal = 20–45 mg/dl). The CSF cell differential showed five white cells (98% lymphocytes) and 24 red cells. The chest radiograph revealed elevation of the left and right hila with a calcified granuloma in the right upper lobe.

A spin-echo MR examination of the lower thoracic spine with T1-weighted (550/20/4) (TR/TE/excitations) and T2-weighted (2000/30–80/2) sequences revealed an intramedullary lesion at T11 with diffuse hyperintensity on T2-weighted images in the lower thoracic cord and conus (Fig. 1B). Following administration of contrast medium, an enhancing mass was identified at T11, and two small enhancing lesions were seen caudally in the conus (Fig. 1C). An iodinated contrast-enhanced CT scan of the head demonstrated enhancing lesions in the right frontal and parietal lobes as well as in the left parietal lobe with surrounding edema (Fig. 2).

The results of a stereotaxic biopsy of the right basifrontal cerebral lesion demonstrated granulomatous inflammation. A histologic smear showed yeast forms, and culture was positive for *Histoplasma capsulatum*. Subsequently, an open biopsy of the T11 lesion was done, the results of which demonstrated a well-encapsulated intramedullary mass. This also was positive for *Histoplasma capsulatum* (Fig. 3).

### Discussion

Histoplasmosis is a systemic fungal infection caused by *Histoplasma capsulatum*. This disease is most common in the Ohio, Mississippi, and St. Lawrence river valleys. The primary portals of infection are believed to be the respiratory tract, gastrointestinal tract, and skin.

Histoplasmosis usually presents clinically as an acute infiltrative or chronic cavitary pulmonary inflammatory process, or as a progressive disseminated somatic infection. The great majority of infections fall into the first category and present as respiratory infections of varying severity. The chronic cavitary form is clinically and radiologically similar to tuberculosis. The disseminated form is seen most frequently in infants, elderly males, and immunocompromised adults.

Cooper and Goldstein [2] reported involvement of the CNS in 24% of cases of disseminated histoplasmosis. Presumably, CNS infection occurs during the preceding hematogenous dissemination. Three types of CNS involvement were described by Shapiro et al. [5]. Their classification, based on pathologic observations, included (1) miliary granulomas in brain, meninges, and choroid plexus; (2) meningitis and associated vasculitis usually involving the basilar portion of brain; and (3) scattered focal regions of cerebritis. Goodwin et al. [6] added a fourth category of spinal cord involvement. Dion et al. [3] described a thalamic histoplasma as a ring-enhancing lesion on postcontrast CT with slight mass effect. T1-weighted images demonstrated an 18-mm mass surrounded by a hypointense rim, which became hyperintense on T2-weighted images. Voelker et al. [7] described a cervical cord histoplasma with subtly decreased intensity on T1-weighted images and increased intensity on T2-weighted images. In neither report [3, 7] were contrast-enhanced images available.

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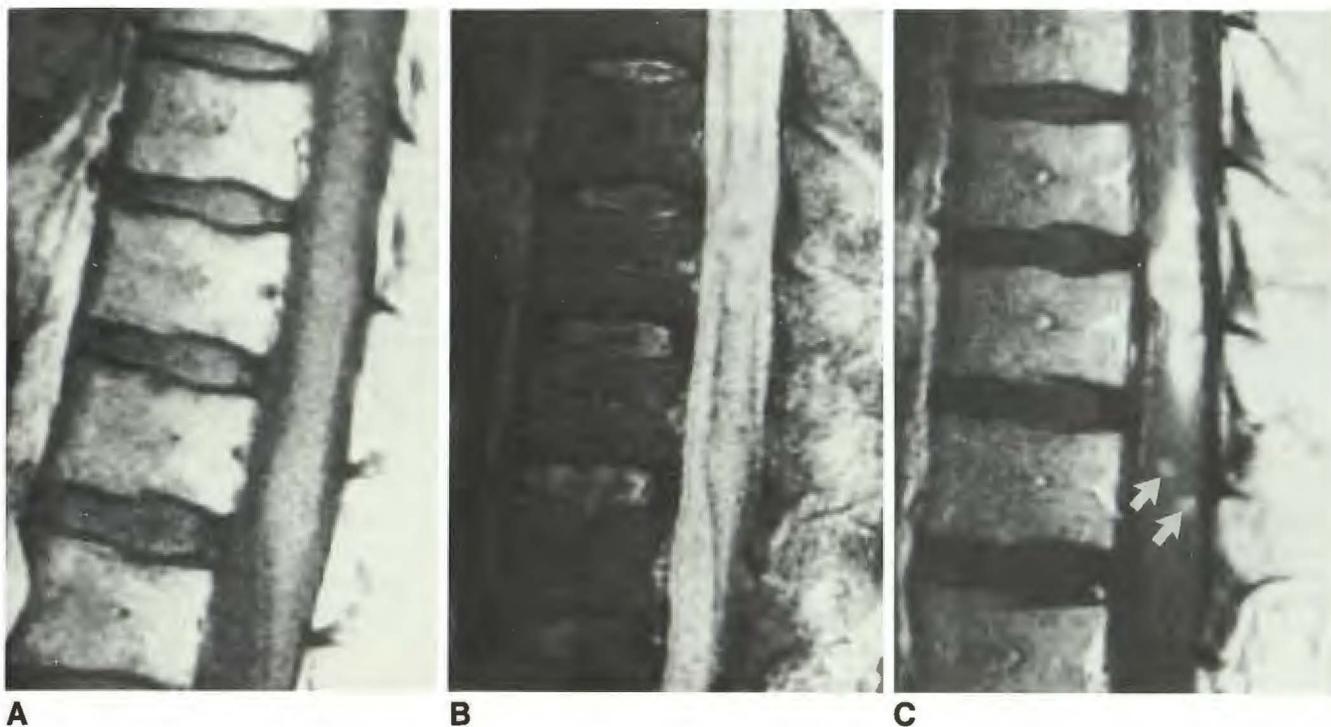


Fig. 1.—A, Sagittal T1-weighted MR image (550/20/4) shows minimal cord irregularity.  
 B, Sagittal T2-weighted MR image (2000/80/2) shows diffuse increased signal within lower thoracic cord and conus.  
 C, Sagittal postcontrast T1-weighted MR image (550/20/4) shows large enhancing lesion at T11 level with two satellite enhancing lesions in conus (arrows).

Fig. 2.—Iodinated contrast-enhanced axial CT scan of brain shows a right basifrontal ring-enhancing lesion surrounded by edema.

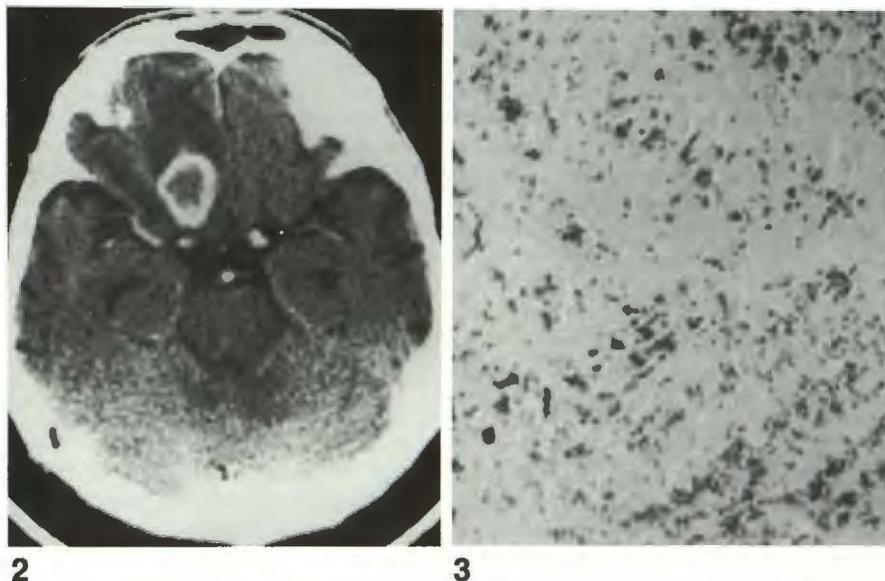


Fig. 3.—Photomicrograph from biopsy of T11 spinal cord lesion shows yeast forms of *Histoplasma capsulatum*. (Gomori methenamine-silver stain)

In our case the diffuse hyperintensity of the conus and lower thoracic cord on T2-weighted images was likely caused by edema and/or inflammation, although no enlargement of the cord was present. Except for three focal lesions, the spinal cord presumably was minimally affected since the

spinal cord–blood barrier was generally intact as evidenced by lack of diffuse contrast enhancement.

The appearance of the cerebral and spinal cord enhancing lesions was not specific. The multiplicity of lesions and the patient's age led to a preliminary diagnosis of metastatic

neoplasm. The history of prior tuberculosis raised the possibility of multiple tuberculomas, although this diagnosis was thought to be less likely, as the patient had received an adequate course of antituberculous therapy and had no evidence of active tuberculosis. The discovery of yeast forms in the biopsy specimens and the growth of *Histoplasma capsulatum* in culture were completely unexpected, since the patient had no history or evidence of disseminated histoplasmosis. Multiple enhancing lesions although often neoplastic in origin can also result from hematogenous spread of infection, ischemia, and even trauma. This case again demonstrates the nonspecific nature of enhancing lesions discovered on CT and MR examinations.

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