Gd-DTPA-Enhanced MR of Multiple Cryptococcal Brain Abscesses

MR imaging has been shown to offer greater sensitivity over CT for CNS infections in AIDS patients [1]. This report describes MR findings in a non-AIDS patient with biopsy-proved cryptococcal CNS infection and suggests improved sensitivity of Gd-DTPA-enhanced MR imaging.

Case Report

A 39-year-old man with insulin-dependent diabetes presented to the emergency room with headaches, nausea, vomiting, and slight changes in mental status. CSF studies on admission showed elevated protein, normal glucose, and WBCs with a predominance of lymphocytes, consistent with chronic meningitis. Chest radiograph was again showed findings consistent with chronic meningitis.

An open excisional biopsy of the right anterior pons [5] reported a single linear focus of hyperintensity at the right temporooccipital fissure showed enhancement. The noncontrast T2-weighted images were unchanged from the prior study. There was slight meningeal enhancement and thickening adjacent to the right anterior pons. The previously seen linear focus of hyperintensity at the right temporocerebellar fissure showed enhancement.

The patient was discharged approximately 1 month after admission with discontinuation of treatment. Noncontrast MR was obtained at the time of discharge (Fig. 1B). Three weeks later, a Gd-DTPA-enhanced MR study was obtained (Figs. 1C and 1D).

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An open excisional biopsy of the right anterior temporal gyrus was performed. The exposed surface of the brain was remarkable for multiple 2- to 4-mm whitish granuloma-appearing lesions, particularly patterned around blood vessels. The histologic sections confirmed the presence of multiple microabscesses, appearing as numerous discrete small necrotic lesions. The microabscesses contained amorphous debris and cryptococcal organisms encased by an outer fibrous capsule. The surrounding brain tissue showed numerous reactive astrocytes and perivascular lymphoid cuffing. The pathologic specimen did not include sufficient meningeal tissue for examination.

Discussion

Cryptococcus neoformans infection of the CNS occurs in both immunocompetent and immunocompromised hosts. In immunocompetent individuals, it usually is a manifestation of disseminated pulmonary cryptococcosis. In AIDS patients, CNS cryptococcus is the most common fungal infection and may present as the initial opportunistic infection or as a later complication [2].

CNS cryptococcosis affects persons of all ages and presents as a basilar meningitis (or less frequently as a cryptococcoma). Meningeal symptoms include headache, fever, nausea, vomiting, change in mental status, neck stiffness, and visual disturbances, although some patients may be asymptomatic. The space-occupying cryptococcomas can produce focal signs [3].

The CSF findings are not specific, and in chronic meningitis they may be due to other causes (elevated pressure, protein and cells, mostly lymphocytes). The CSF protein and glucose levels and cell count may be normal in cryptococcal meningitis in patients with AIDS [2].

In Cryptococcus neoformans infections, the inflammatory cellular response is mild but sometimes granulomatous. Invasion of blood vessels and resulting vasculitic changes may occur. The formation of cysts, filled with organisms, may occur in the Virchow-Robin spaces, the depths of sulci, the superficial layers of the cortex, and (occasionally) the deep white matter and basal ganglia [4].

Cryptococcosis is difficult to detect on head CT scans [1, 5-8]. Usually, CT scans are normal, and CT findings are limited to mild or moderate ventricular and basal cisternal enlargement and minimal cortical atrophy without meningeal enhancement on contrast-enhanced CT. Cryptococcomas have appeared as ring-enhancing lesions [5]. Post et al. [8] reported negative CT scans in the two patients of their study who had cryptococcal perivascular infiltration of the brain proved at autopsy.

MR descriptions of CNS cryptococcosis are limited. Jarvik et al. [9] reported a single AIDS patient in whom MR revealed CNS cryptococcosis. The multiple, enhancing, punctate lesions seen in our case most likely represent focal areas of blood/brain barrier breakdown around microabscesses, granulomas, or perivascular inflammation. True vasculitic changes were not seen on the pathologic sections from the rather small specimen.

Thomas J. Riccio
John R. Hesselink
University of California, San Diego
Medical Center
San Diego, CA 92103

Fig. 1.—A, Noncontrast CT scan shows multiple punctate calcifications and mild ventricular dilatation. Calcifications were not present 15 months earlier. B, T2-weighted noncontrast MR image (3000/80) shows a single linear focus of hyperintensity (arrow) at right temporocerebellar fissure. C and D, Gd-DTPA-enhanced MR image (600/20) reveals numerous punctate areas of enhancement, which are in some areas related to the vessels. Most are within sulci.
REFERENCES