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MR of Isolated Leptomeningeal Glioma

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Summary: We report the MR study of a case of high-grade glioma that mimics leptomeningeal carcinomatosis. Superficial gliomas should thus be included in the differential diagnosis of isolated meningeal enhancement.

Index terms: Glioma; Meninges, magnetic resonance; Meninges, neoplasms

Leptomeningeal extension is a common feature of superficial gliomas. High-grade gliomas localized in the leptomeningeal space without any intraparenchymal components are unusual.

Case Report

A 26-year-old woman with no previous disease was admitted for recent seizures. The patient underwent a computed tomographic study that did not show any abnormality, so magnetic resonance (MR) was performed. The MR (Signa, General Electric, Milwaukee, Wis) included sagittal T1-weighted images, axial T2-weighted images, and axial, sagittal, and coronal T1-weighted images after injection of gadopentetate dimeglumine (0.1 mL/kg) (Fig 1). No abnormality was seen on preenhancement T1- or on T2-weighted images. Injection of gadopentetate dimeglumine revealed clear pathologic leptomeningeal enhancement of the postcentral and interparietal sulci. Neither the MR study of the spine nor of the orbit revealed any abnormality. The cerebrospinal fluid analysis demonstrated an elevated protein value (150 mg/L) but negative cytology. No surgical procedure was attempted at this time. The patient received steroid and anticonvulsive therapy. After 2 weeks a second MR study disclosed less pial enhancement, so lymphoma or sarcoidosis remained the main diagnosis. Biopsy by open procedure was elected because of the superficial location of the lesion. Pathologic analysis revealed clusters of glial tumoral cells interspersed by collagen septa largely invading the meningeal space; high-grade glioma was diagnosed.

Discussion

Diffuse involvement of leptomeninges by neoplasic cells is a common feature of the neurectodermal tumors. Meningeal gliomatosis is usually caused by seeding by way of the cerebrospinal fluid from tumors abutting on the meningeal or more frequently on the ventricular surface. Reactive fibrosis is often observed in the subarachnoid space; this limits meningeal extension when the tumors reach the surface of pia mater (1). Meningeal gliomatosis occurs in the terminal course of spinal cord, brain stem, or huge supratentorial parenchymal gliomas and seems to be more prevalent in patients with long postoperative survival (2). Meningeal gliomatosis has been extensivly described in pathology publications (2–6).

Primitive meningeal gliomas are tumors that lie entirely in the subarachnoid space and remain separated from the underlying cerebral parenchyma by pia mater. No evidence of parenchymal lesion can be demonstrated. Sometimes lesions are linked to the parenchyma by glial bridges extending via the perivascular spaces into the superficial layers of the cortex (6). It remains unclear whether such lesions should be considered genuine primitive tumors or as seeding of unknown intraparenchymal gliomas (4). Theoretically, diagnosis can be made only after postmortem examination, which excludes the spinal cord and other sites of intraparenchymal tumor.

In our case, no intraparenchymal brain or spinal lesion could be seen on MR studies, and only minute cortical involvement could be seen on pathologic analysis of the biopsy. In such cases, primitive leptomeningeal gliomas are thought to result from neoplastic transformation of glial heterotopic nests found in about 1% of

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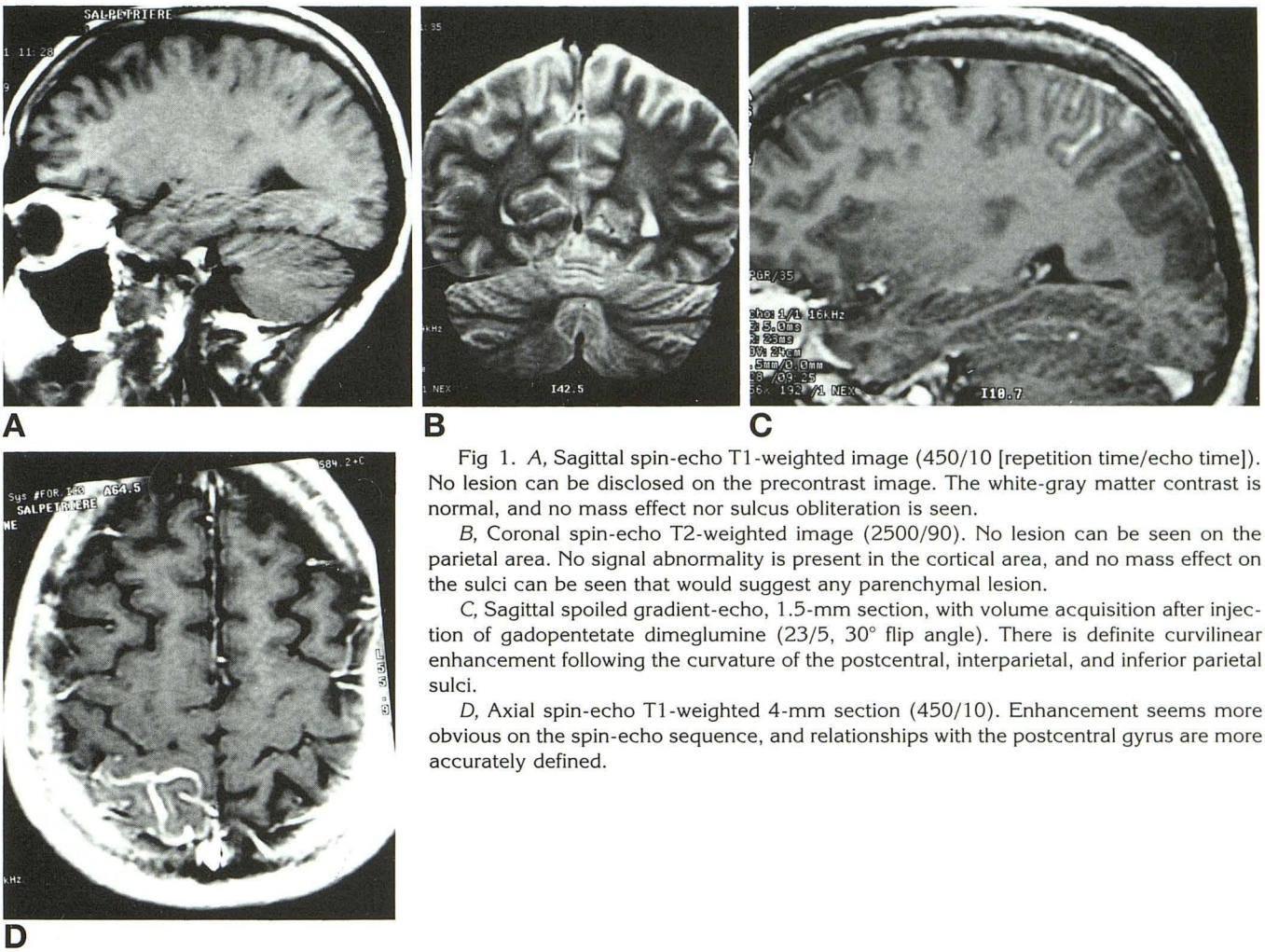

Fig 1. *A,* Sagittal spin-echo T1-weighted image (450/10 [repetition time/echo time]). No lesion can be disclosed on the precontrast image. The white-gray matter contrast is normal, and no mass effect nor sulcus obliteration is seen.

B, Coronal spin-echo T2-weighted image (2500/90). No lesion can be seen on the parietal area. No signal abnormality is present in the cortical area, and no mass effect on the sulci can be seen that would suggest any parenchymal lesion.

C, Sagittal spoiled gradient-echo, 1.5-mm section, with volume acquisition after injection of gadopentetate dimeglumine (23/5, 30° flip angle). There is definite curvilinear enhancement following the curvature of the postcentral, interparietal, and inferior parietal sulci.

D, Axial spin-echo T1-weighted 4-mm section (450/10). Enhancement seems more obvious on the spin-echo sequence, and relationships with the postcentral gyrus are more accurately defined.

the population (3). Unlike intraparenchymal gliomas, these tumors usually arise in young patients and have very poor prognoses. Symptoms and clinical signs are usually nonspecific and may be misleading, suggesting infectious meningoencephalitis. Cytologic analysis of cerebrospinal fluid is often disappointing and is not diagnostic.

Cortical enhancement mimicking leptomeningeal carcinomatosis has been reported in cases of gliomatosis cerebri and has been ascribed to localized edema and blood-brain barrier disruption within the molecular layer of the cortex (7). In these cases, the cortical enhancement is an intrinsic feature of the lesion, because MR and tissue analysis disclose obvious intraparenchymatous involvement. In our case, enhancement seems to be related to the direct invasion of the pia-arachnoid meninges such as that observed in meningeal carcinomatosis or infectious meningitis. This is secondary to blood-meningeal barrier abnormalities or to increased leptomeningeal vascularity (8), because only minute cortical involvement could be noticed on all samples of pathologic examination.

Isolated leptomeningeal glioma is a very rare form of glioma. MR features can mimic other more frequent meningeal lesions such as meningeal carcinomatosis, infection, or inflammatory granulomatosis. Leptomeningeal gliomas, although much less frequent than these lesions, should be included in the differential diagnosis of meningeal enhancement. MR can disclose the exact location of the lesion, its relations with the adjacent parenchyma, and the ideal biopsy location.

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