Gliomatosis Cerebri: Comparison of MR and CT Features

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OBJECTIVE. Gliomatosis cerebri is a diffuse infiltrative glial neoplasm frequently involving both cerebral hemispheres. Diagnosis and evaluation of its extent with CT are known to be difficult. The purpose of this study was to compare the MR and CT findings in gliomatosis cerebri.

MATERIALS AND METHODS. MR images of nine patients were reviewed retrospectively and compared with CT scans. Pathology was determined by open or stereotaxic biopsy. The MR images included sagittal T1-weighted, axial proton density–weighted, and T2-weighted images. Contrast material was administered in seven patients. Unenhanced and enhanced CT scans were obtained in eight patients.

RESULTS. On proton density–weighted and T2-weighted MR images, the most common findings were poorly defined bilateral areas of diffuse high signal intensity in the cerebral hemisphere. On T1-weighted images, the lesions were isointense to hypointense compared with normal brain. Enhanced T1-weighted images showed focal parenchymal enhancement in three patients and meningeal enhancement in one. On CT scans, the lesions showed poorly defined areas of subtle low density or isodensity, and appeared much smaller than those on T2-weighted MR images. Enhancement was seen in only one case. The extent of disease was evaluated much better on T2-weighted MR images than on T1-weighted MR images and CT scans.

CONCLUSION. In gliomatosis cerebri, MR imaging is more sensitive than CT for detecting lesions and shows the extent of disease better than CT does. Accordingly, MR imaging should be used as a primary imaging study in the evaluation of gliomatosis cerebri.

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Gliomatosis cerebri is an uncommon primary brain tumor characterized by diffuse neoplastic proliferation of astrocytes, with good architectural preservation and relative sparing of the neurons [1–3]. Diagnosis is difficult because the symptoms and signs are variable and nonspecific, and neuroimaging usually does not show a localized mass in the brain. Even on CT scans, the lesion usually shows diffuse isodensity or slight hypodensity with no or minimal contrast enhancement [4–6]. Although MR imaging is superior to CT in the evaluation of most neurologic diseases, the MR findings of gliomatosis cerebri have been reported in only a few cases [6–9]. We describe the MR findings in gliomatosis cerebri and compare them with CT findings to determine the advantages of MR over CT in the evaluation of gliomatosis cerebri.

Materials and Methods

MR findings in nine patients, six men and three women 21–53 years old (mean, 36 years), with gliomatosis cerebri were retrospectively studied. The diagnosis of gliomatosis cerebri was confirmed by two pathologists on the basis of histopathologic specimens obtained through open craniotomy and tumorectomy in six patients and through stereotaxic biopsy in three.

Spin-echo MR images were obtained with a 2.0-T superconducting unit in five patients, a 1.0-T unit in two, and a 0.5-T unit in two. Sagittal T1-weighted (500–600/15–30 [TR/TE]), axial proton density–weighted (2000–2500/20–30), and axial T2-weighted (2000–2500/60–100) MR images were obtained in all patients. A slice thickness of 5–7 mm, an interslice gap

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of 1–3 mm, a matrix of 256 × 200–256, and a field of view of 22–25 cm were used. In seven patients, axial and occasionally coronal T1-weighted images were obtained after IV injection of gadopentetate dimeglumine (0.1 mmol/kg). Follow-up MR images were obtained 2 months after initial studies in two patients who had not undergone radiation or surgical therapy. CT scans were obtained in eight patients within 1 week prior to MR imaging. Unenhanced and contrast-enhanced scans were obtained in all eight patients.

The MR and CT images were retrospectively reviewed by five radiologists together in conference, who compared the two techniques with each other side by side. Discrepancies in interpretation were resolved by consensus. Attention was paid to the extent of the disease, signal intensity of abnormal brain parenchyma, and presence and pattern of contrast enhancement on MR and CT images.

Results

In all patients, proton density–weighted and T2-weighted MR images showed an area of diffuse, poorly defined high signal intensity in the cerebral hemispheres with variable degrees of swelling of the involved brain. The high signal intensity varied in degree from slight to severe. On T1-weighted images, the abnormality was slightly hypointense or isointense relative to normal brain. Contrast-enhanced T1-weighted images showed nodular 1–2 cm areas of enhancement in four patients, solitary focal enhancement in two, and multifocal enhancement in two. In one patient with parenchymal enhancement, short linear meningeal enhancement was observed in the inferior frontal lobe.

CT scans showed poorly defined low-attenuation lesions spreading along the corona radiata in eight patients. In two patients, the extent of distinct low attenuation on CT scans was comparable to the extent of high signal intensity on T2-weighted MR images. In the remaining six, areas of low attenuation on CT scans were smaller and more subtle than areas of high signal intensity on T2-weighted MR images. Mass effect was seen in eight patients on MR but in only six patients on CT. Involvement of the gray matter and corpus callosum was barely detectable on CT scans. Contrast enhancement similar to that seen on the MR images was seen on contrast-enhanced CT scans in only one of four patients in whom MR showed enhancing areas.

On the basis of T2-weighted MR images, the hemispheric lesions involved the frontal lobe in nine patients, the parietal lobe in eight, the temporal lobe in seven, and the insula in seven. The lesions crossed the midline through the corpus callosum in eight patients and through the thalami in one. The basal ganglia and thalami were involved in six patients each. The lesions extended into the external capsule in eight patients, the internal capsule in six, the septum pellucidum in five, the midbrain in three, and the pons in two. The cerebellum and occipital lobe did not appear to be involved. In four patients, both hemispheres were affected almost symmetrically (Figs. 1 and 2). In the remaining five patients, the extent of the lesion was more prominent in one hemisphere (Figs. 3 and 4). In the cerebral hemisphere, areas of gray

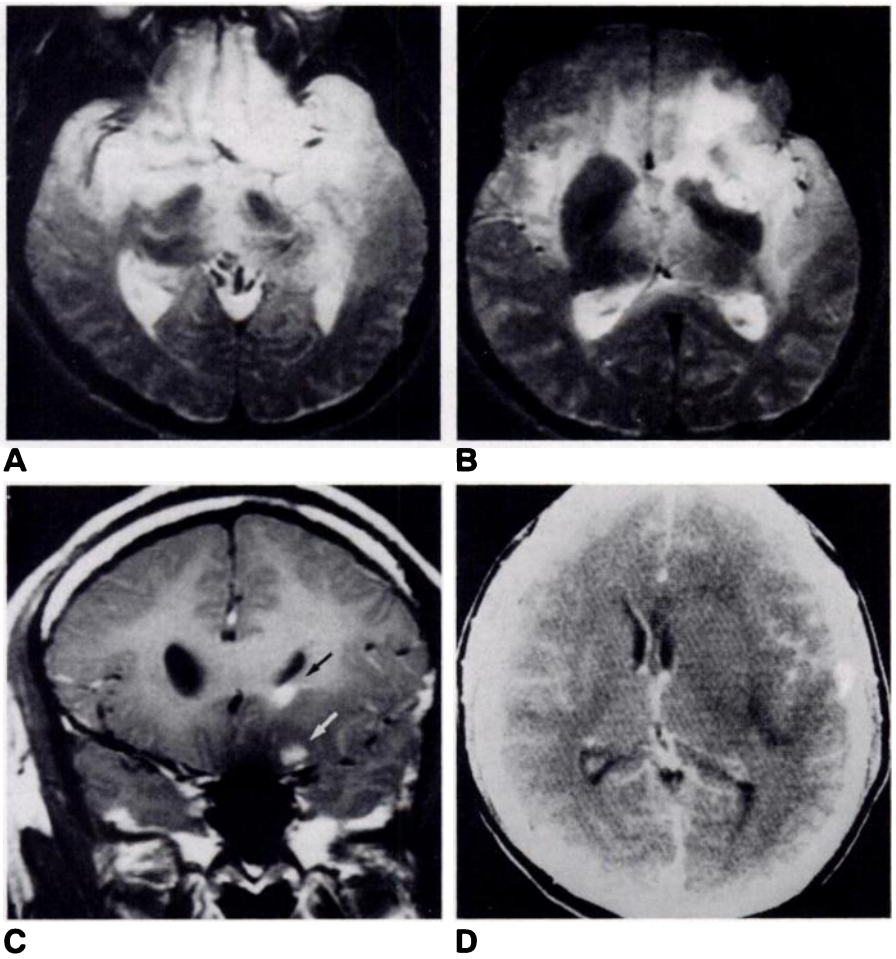

Fig. 1.—32-year-old man with seizures and visual disturbance.

***A* and *B*, Axial T2-weighted MR images show bilateral diffuse high-signal-intensity lesion involving frontotemporal area, insula, basal ganglia, internal capsule, periventricular white matter, and corpus callosum.**

***C*, Enhanced coronal T1-weighted MR image shows two areas of focal enhancement in left frontal gray and white matter (*arrows*).**

***D*, Contrast-enhanced CT scan shows poorly defined low attenuation in left frontal white matter and internal capsule and mass effect on left lateral ventricle. Compare contrast and extent of lesion with that seen in *A* and *B*.**

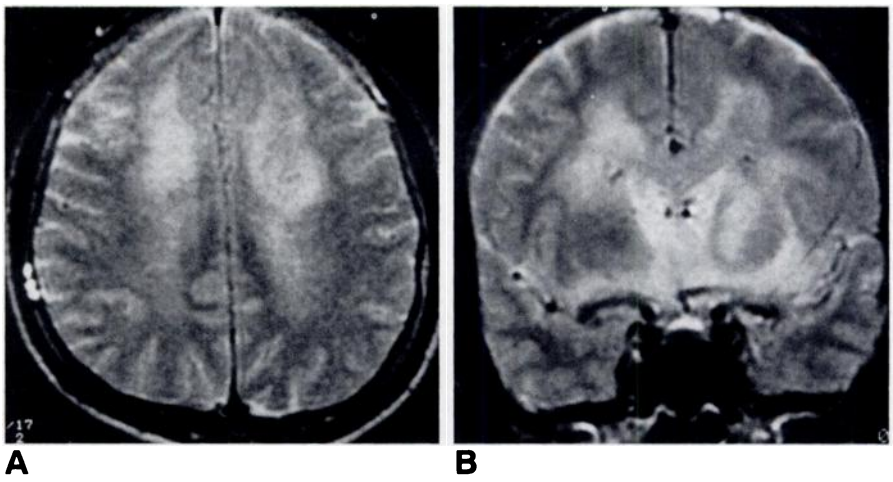

Fig. 2.—51-year-old man with aphasia, memory disturbance, and right-sided involuntary movement.

***A* and *B*, Axial (*A*) and coronal (*B*) T2-weighted MR images show symmetric bilateral areas of diffuse high signal intensity in cerebral hemispheres, most prominent in white matter, including periventricular white matter, internal and external capsules, centrum semiovale, and corpus callosum. Corpus callosum is thickened.**

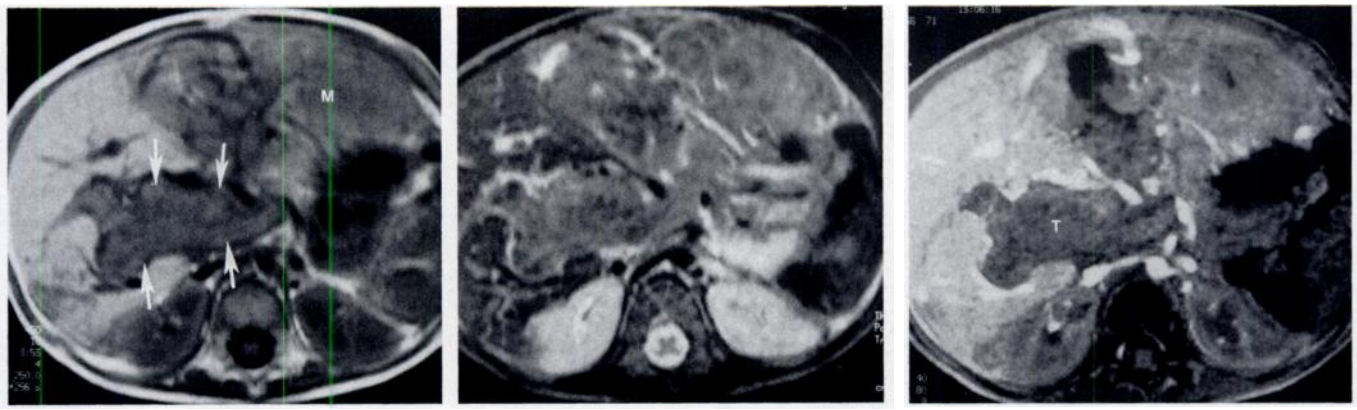

Fig. 3.—50-year-old woman with disorientation and olfactory hallucinations.

***A* and *B*, Axial T2-weighted MR images show asymmetric area of high signal intensity involving inferior frontotemporal area and thalami bilaterally. Lesion is more extensive on right side.**

***C*, Contrast-enhanced CT scan shows areas of subtle low density in right thalamus and deep frontal lobe, with mass effect on right lateral and third ventricles.**

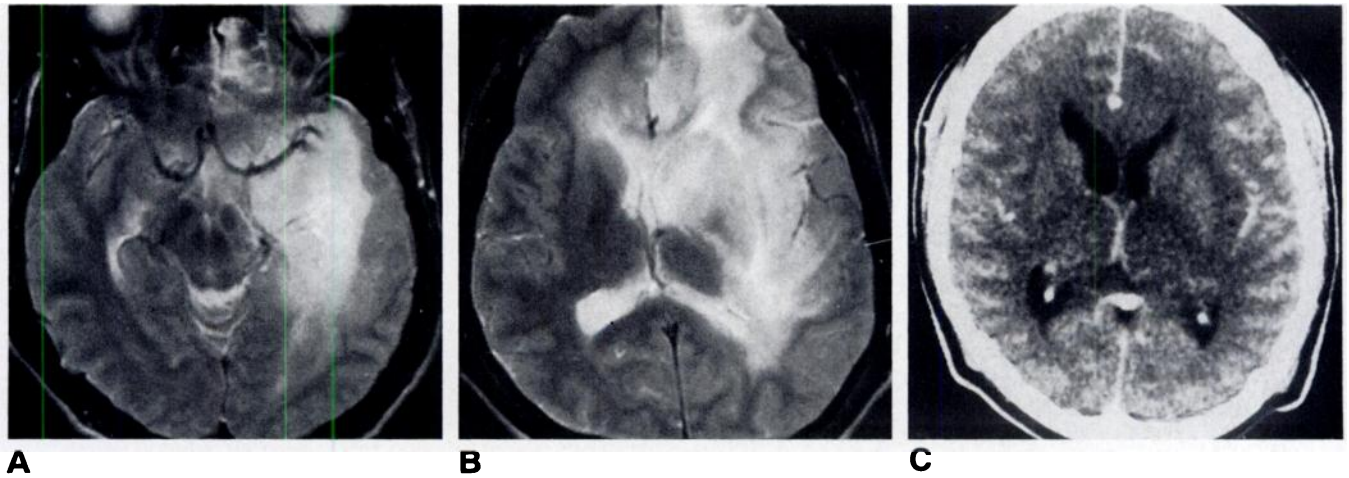

Fig. 4.—30-year-old man with right hemiparesis, alexia, acalculia, and memory disturbance.

***A* and *B*, Axial T2-weighted MR images show diffuse areas of high signal intensity in left frontotemporoparietal area, internal capsule, external capsule, and basal ganglia, extending to right frontal lobe through corpus callosum.**

***C*, Contrast-enhanced CT scan shows slight thickening of genu of corpus callosum in left side with subtle mass effect on frontal horn of lateral ventricle. There is no definite area of abnormal attenuation.**

and white matter were commonly affected, but involvement of the white matter was more prominent and extensive. The distinction between gray and white matter in the involved region was poor, particularly in the swollen frontotemporal area. Follow-up MR images obtained 2 months after initial studies in two patients showed no interval changes.

Discussion

The principal pathologic characteristics of gliomatosis cerebri are diffuse neoplastic proliferation of glial elements among neural structures and destruction of myelin sheaths with slight or no damage to nerve cells and axons [1–3]. Abnormal glial cells infiltrate along the anatomic pathways, mainly throughout the white matter. Commissural structures such as the corpus callosum are frequently affected and expanded. Gray matter may be preserved [10], but dense subpial expansion and edema are common [3, 8].

The disease usually is slowly progressive, although its course can span from a few days to more than 20 years. Nonspecific personality and mental changes are the most frequent clinical manifestations [1], and clinical findings are disproportionate to the extent of the brain involvement [11]. Thus, diagnosis usually is difficult and based on neuroimaging findings.

The MR findings in our cases were similar to those of cases previously reported [7–9]. The lesion was either isointense or slightly hypointense on T1-weighted images and diffusely hyperintense on proton density–weighted and T2-weighted images. The abnormal signal intensity might be attributable to tumor cell infiltration or demyelination or both. Localized areas of high signal intensity in the midbrain and pons on T2-weighted images might be caused by either true tumor infiltration or wallerian degeneration. Proton density–weighted and T2-weighted images were superior to T1-weighted images for evaluating the extent of the lesion. Sagittal and coronal images were useful particularly for the evaluation of focal or diffuse enlargement of the corpus callosum (Fig. 2). Expansion of affected areas and mass effect were well delineated, particularly on coronal images. Therefore, coronal and sagittal images can help in differentiating gliomatosis cerebri from other white matter demyelinating diseases.

Contrast enhancement reflects disruption of the blood-brain barrier, presumably in areas of more dense infiltration of tumor cells. In our study, focal areas of contrast enhancement were seen on MR in four patients, but enhancement was seen on CT in only one of these. Better detection of these enhancing lesions with MR seems to have resulted from the intrinsic higher sensitivity of MR and gadopentetate dimeglumine. On CT scans, many lesions appeared as poorly defined, subtle hypodense or isodense areas [4–7]. In nine cases of gliomatosis cerebri reported by Artigas et al. [12], CT findings were normal in three. In the other six, CT scans showed midline shifting, hydrocephalus, and diffuse hypodense areas in the white matter, as in our cases. When CT shows only subtle hypodense areas in the white matter, gliomatosis cerebri may resemble a demyelinating disease [4]. In another case [7], CT scans showed multifocal hypodense areas corresponding to cystic degeneration of the white matter with intervening, apparently normal brain tissue. Because many seemingly uninvolved areas contained neoplastic cells, the tumor extended far beyond the limits suggested by gross examination or CT. Pathologic examination and MR images can show the continuity between the lesions and enable correct estimation of disease extent and diagnosis.

Because MR affords better tissue contrast than CT does, MR can show the extent of tumor much better. Involvement of gray matter or the corpus callosum and mass effect are well visualized with MR, partly owing to its good tissue characterization properties and multiplanar imaging capability. CT does not show these features, and it is suboptimal for lesion detection and differentiation from other white matter disease.

In summary, lesions of gliomatosis cerebri appear as diffuse, poorly defined hyperintense areas on proton density–weighted and T2-weighted MR images and as isointense or hypointense areas on T1-weighted images. In our series, focal contrast enhancement of the affected portion was seen in a limited number of cases. MR images were superior to CT scans for evaluating the extent of gliomatosis cerebri. Multiplanar images such as coronal and sagittal scans also provided better information of involved areas and helped to distinguish gliomatosis cerebri from other white matter demyelinating diseases. On CT scans, the lesions showed isodensity or subtle hypodensity and appeared much smaller than lesions on T2-weighted MR images. Accordingly, MR is expected to play a major role, surpassing CT, in the primary diagnosis of gliomatosis cerebri.

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