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MR Imaging in Patients with Temporal Lobe Seizures: Correlation of Results with Pathologic Findings

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Thirty-nine consecutive patients with medically intractable complex partial seizures were studied with electroencephalography and MR imaging to localize an epileptogenic focus for temporal lobectomy. The patients were divided into three groups on the basis of pathologic findings after lobectomy: Group 1 comprised 13 patients with neoplasms, hamartomas, or cysts; group 2 comprised 13 patients with moderate and severe mesial temporal sclerosis (one patient was included in both groups 1 and 2); and group 3 comprised 14 patients who underwent aspiration lobectomy, which yielded limited tissue for pathologic study so no pathologic diagnosis was made. The majority of the patients in group 3 were assumed to have mesial temporal sclerosis. Abnormal MR signal in the temporal lobe on T2-weighted images was graded as minimal increase (1+), intermediate or moderate increase (2+), and very significant increase (3+). An abnormal signal was demonstrated in 26 (67%) of the 39 patients. In group 1, the tumor/cyst subgroup, an abnormal signal was seen in all 13 patients. Most had 3+ signal. There was increased signal in eight (62%) of 13 patients in group 2 and in six (43%) of 14 patients in group 3.

This study suggests that MR can detect almost all tumors and a significant number of mesial temporal sclerosis lesions in individuals with complex partial seizures. On the basis of this small series, individuals who exhibit significant signal (3+) can be expected to have neoplasms, hamartomas, or cysts, and patients who exhibit minimal signal (1+) will usually have mesial temporal sclerosis.

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Seizures that arise from a focus in the mesial temporal lobe are frequently refractory to drug therapy. When there is accurate preoperative localization of the epileptogenic focus, temporal lobectomy can result in complete cure or a significant improvement in seizure activity [1]. Electroencephalography (EEG) has provided the major data concerning localization of such seizures. Imaging techniques including skull series, pneumoencephalography, and angiography have not been useful for evaluation. CT scans, which can be correlated with functional electrographic abnormalities, have not been helpful. Recently, positron emission tomography has given new information about metabolic changes at the focus [2]; however, such equipment is restricted in number and accessibility. More recently, MR imaging has been shown to be useful in establishing the location of epileptogenic foci [3–6].

We studied 39 consecutive patients with intractable temporal lobe seizures undergoing temporal lobectomy. The patients were studied between January 1984 and December 1987. The imaging studies in these patients were correlated with the surgical and pathologic findings and the clinical response after lobectomy.

Materials and Methods

Patient Population

Thirty-nine consecutive patients with complex partial seizures who had temporal lobectomy at the Duke University Medical Center were included in this study. Presurgical investigations

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in all patients included CT, MR, scalp EEG, video/EEG cable telemetry, neuropsychological testing, and intracarotid Amytal examination. In 10 patients, localization of the epileptiform focus by noninvasive measures was not possible. Invasive monitoring techniques using implanted depth electrodes were required for adequate localization in these patients.

Imaging Studies

CT scans were obtained on GE 8800 and 9800 units without and with contrast material. A 10-mm slice thickness was used in the axial plane. Selected patients had coronal studies.

MR studies were performed on a 1.5-T GE Signa unit. T1-, intermediate-, and T2-weighted images using a spin-echo (SE) sequence, 2500/40,80 (TR/TE), were obtained. Axial slices parallel to the temporal horn with 5-mm slice thicknesses and coronal images also were obtained in all patients. The phase-encoding gradient was changed to the cephalocaudad direction for all seizure patients to avoid artifacts over the temporal lobe related to carotid arterial pulsation. The scans of the 39 patients were mixed randomly with eight control scans and reviewed by two experienced neuroradiologists without knowledge of the diagnosis. The control patients were chosen for the study from among seizure patients in whom the seizure focus was located specifically outside the temporal lobe; this meant that the technique for these MR scans was identical to that for the temporal lobe patients, including a change in the phase-encoding direction.

Abnormalities on MR images were recorded as (1) increased signal intensity on intermediate- and T1-weighted images in the absence of structural deformity; (2) mass deformity, including extension of cortical signal into adjacent white matter; (3) calcification; and (4) atrophy. Atrophy was said to be present when there was dilatation of the temporal horn and/or dilated CSF spaces with loss of temporal lobe volume.

Abnormal MR signal was classified into very significant increased signal intensity (3+), moderately high signal (2+), and minimal but definitely increased signal (1+) in the temporal lobe. When the signal was recorded as 3+, the increased signal was approximately as intense as that of CSF in the lateral ventricle on some but not necessarily all late T2-weighted scans. When increased signal was accompanied by mass effect or distortion of normal anatomy suggesting structural mass lesions, this was so recorded.

Surgical Procedures

After electrocorticography and electrical recordings from the amygdala and hippocampus, the temporal lobe was resected in one of two ways. After the anterior 5 cm of the lateral temporal lobe was resected, one surgeon removed the uncus, amygdala, and hippocampus by subpial resection. In the other cases, the other surgeon removed these areas en bloc.

Pathology Studies

Tissue stains used included hematoxylin and eosin, with or without Luxol fast blue for counterstain. The patients were divided into three groups on the basis of pathologic findings at lobectomy (Table 1). The first group (group 1) comprised patients with neoplasms, hamartomatous malformations, or cysts. All patients in the second group (group 2) had severe or moderate mesial temporal sclerosis (MTS). The third group (group 3) comprised patients who had normal neocortex but in whom, with one exception, no histologic tissue was available from the hippocampus. These patients were considered likely to have MTS. Groups 1 and 2 were chosen to correspond to groups A and B in the study of Kuzniecky et al. [7].

Results

Group 1: Neoplasms or Other Abnormal Tissue

In 13 patients (33%), neoplastic or other structural lesions were found in the temporal lobectomy specimen. Six patients had astrocytomas, two had oligodendrogliomas, one had an undifferentiated tumor, one had an epidermoid, one had a cyst, and one had multiple hamartomas and associated MTS (Figs. 1 and 2; Table 1). MR revealed significant increases in signal intensity (3+) in all but five patients. A moderate increase in signal intensity (2+) was seen in four of these and a minimal increase in signal (1+) in one. However, in the latter patient, an exophytic tumor projected off the temporal lobe tip, which appeared to be an obvious intrinsic brain tumor even though the MR signal was not very dramatic. Some deformity was seen in 12 of 13 patients in this group. In two, significant calcification was seen on CT.

Group 2: Moderate to Severe Gliosis and Neuronal Loss

Moderate to severe neuronal loss and gliosis of the neocortex and/or mesial temporal structures were seen in 13 patients (Fig. 3; Table 1). The mesial temporal structures were involved in each of these patients. These patients had classical MTS. In eight (62%) abnormal MR signal was seen in the temporal lobe. Moderately increased signal (2+) was seen in one patient; in the other patients with MTS and increased MR signal in the temporal lobe the signal was graded as 1+. In none was mass deformity or calcification established. Atrophy of the temporal lobe was seen in one patient.

Group 3: Incomplete Pathologic Data

In 13 of these 14 patients the temporal lobe neocortex was examined histologically, but in only one patient was the hippocampus examined histologically. In this one patient it was normal; however, the MR scan showed a 2+ signal (Fig. 4). On the basis of our findings and those of others, it is likely that the majority of these patients had MTS. MR studies showed increased signal in six of the 14 patients. In five of the six a 1+ increase in signal was seen; in one, a 2+ signal was seen. The 2+ signal occurred in the single patient in whom the neocortex and hippocampus were normal histologically. In one patient, significant atrophy was seen in the temporal lobe.

Control Subjects and Follow-up

Eight control subjects were scattered through the series; all were regarded as normal by the reviewers.

Postoperatively, patients were followed for a mean of 28 months (range, 12–45 months). The outcome was determined by incidence of seizures during the most recent year of follow-up. Patients were categorized as seizure-free if they had no

TABLE 1: MR and Pathologic Findings in Patients with Temporal Lobe Epilepsy

Group/Case No.	MR Signal	Deformity	Pathologic Findings
1: Neoplasms or other abnormal tissue			
1[a]	3	2	Anaplastic astrocytoma
2	3	2	Oligodendroglioma
3	3	2	Astrocytoma
4	3	2	Undifferentiated neoplasm
5	3	2	Astrocytoma
6	3	3	Epidermoid
7	3	2	Astrocytoma
8[a,b]	3/1	1	Hamartoma/MTS
9	2	2	Ganglioglioma
10	2	0	Cyst
11	2	1	Astrocytoma
12	1[c]	1	Astrocytoma
13[d]	2	1[e]	Oligodendroglioma/MTS
2: Moderate to severe gliosis and neuronal loss			
8[b]	3/1	–	Hamartoma/severe MTS
14	2	–[f]	Severe MTS
15	1	–	Severe MTS
16	1	–	Moderate MTS
17[g]	1	–	Severe MTS
18	1	–	Moderate MTS
19	1	–	Severe MTS
20	1	–	Moderate MTS
21	0[f]	–	Severe MTS
22	0	–	Severe MTS
23	0	–	Severe MTS
24	0	–	Moderate MTS
25	0	–	Severe MTS
3: Incomplete pathologic data			
26[h]	2	–	Normal
27	1	–	Normal
28	1	–	Normal
29[g]	1	–	Normal
30	1	–	Normal
31	1	–	Normal
32	0	–	Normal
33	0	–	Normal
34	0	–	Normal
35	0	–	Normal
36	0	–	Normal
37	0	–	Normal
38	0	–	Normal
39	0	–	Normal

Note.—For MR signal, 0 = normal signal; 1 = minimal but definite increase; 2 = intermediate; 3 = signal similar to that of CSF. For deformity, 0 = no deformity; 1 = minimal deformity; 2 = moderate deformity; 3 = severe deformity. MTS = mesial temporal sclerosis.

[a] Calcification was seen on CT.

[b] This patient is listed in both groups 1 and 2.

[c] A 6-mm exophytic tumor was found on the surface of the temporal lobe tip. One observer considered it a small mass (grade 1); another considered it normal (grade 0).

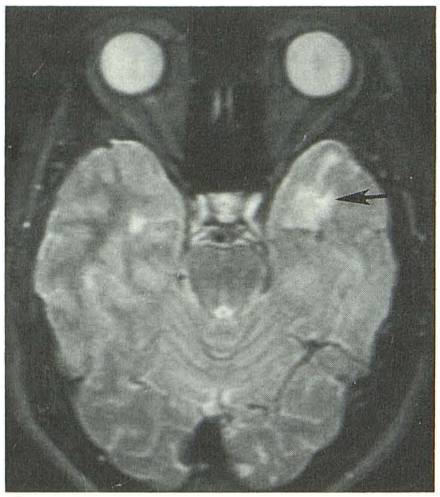
[d] Neoplasm was not found on block resection; however, microscopic examination of the entorhinal cortex detected a very small oligodendroglioma.

[e] Graded 2 by one observer and 1 by another.

[f] Regarded as minimal mass effect by one observer. Mass deformity was not seen in any other patients in groups 2 or 3.

[g] "Atrophy" was assigned to case 17 by one observer. Minimal to moderate atrophy was found in case 29. Atrophy was not seen in any other patients.

[h] Histologic examination showed normal neocortex and hippocampus. The other 13 patients in group 3 were studied by aspiration technique, which showed normal cortex. The hippocampus was not available for study in these patients; it is likely that most of these patients had MTS.



A

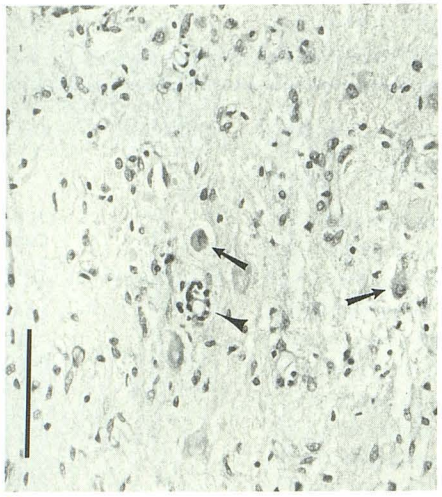


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Fig. 1.—Case 9: 28-year-old man with 27-year history of complex partial seizures. Temporal lobe resection revealed ganglioglioma.

A, Axial MR image (2500/40) through temporal lobe. Hyperintense focus (*arrow*) in subcortical white matter compartment has very minimal extension anteriorly to cortex. Signal was graded as 2+.

B, Pathology of left lobectomy specimen was considered to be ganglioglioma. Neoplastic neurons (*arrows*) are interspersed among neoplastic astrocytes in a fibrillary background. Perivascular lymphocytic cuffing (*arrowhead*) is seen. No more seizures were seen during 24 months following lobectomy. Scale bar = 100 μm. (H and E)

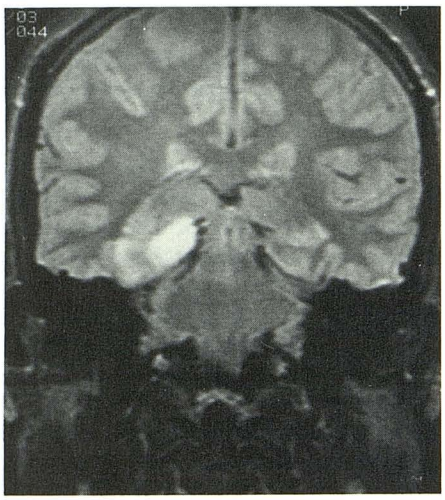
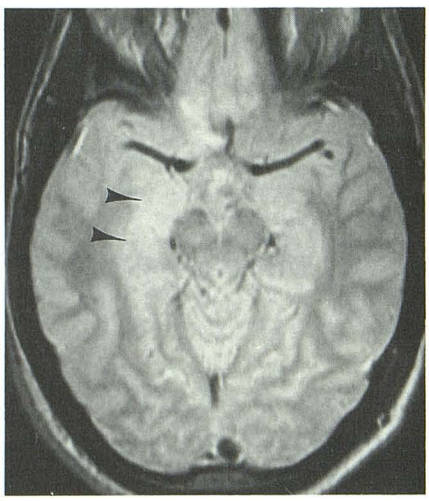


Fig. 2.—Case 7: 26-year-old man with 1-year history of simple and complex partial seizures. Right temporal lobectomy revealed astrocytoma. Coronal MR image (2500/40) shows severe increase in signal intensity (3+), confined largely to the hippocampus.



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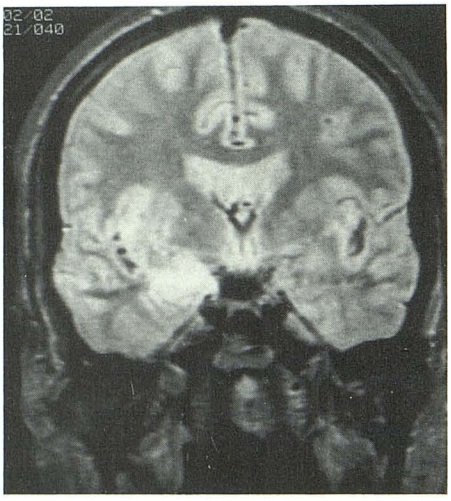

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Fig. 3.—Case 15: 25-year-old man with 9-year history of complex partial seizures. Temporal lobe resection revealed severe mesial temporal sclerosis. T2-weighted axial image (2500/80) shows subtle but definite increase in signal intensity in medial third of temporal lobe (*arrowheads*). Increased signal was rated as 1+.

Fig. 4.—Case 26: 29-year-old man with 10-year history of complex partial seizures. Temporal lobe sections were interpreted as normal at pathology. Incomplete surgical samples may have been sent as patient is thought to have mesial temporal sclerosis. Coronal MR image (2500/40) shows increased signal in hippocampus. This was rated as a 2+ increase in signal.

seizures (excluding auras). Patients were considered significantly improved if they had less than 10 seizures per year *and* at least a 90% reduction in seizures as compared with the preoperative year. All other patients were considered to be not significantly improved.

Overall, 29 (74%) were seizure-free, five (13%) were significantly improved, and five (13%) were not significantly improved. Within pathologic subgroups, 77% of group 1 patients (neoplasms), 90% of group 2 patients (MTS), and 100% of group 3 patients were seizure-free or significantly improved.

Discussion

Historically, neuroimaging for the detection of epileptogenic foci in the temporal lobe has had poor results. CT, while demonstrating the capacity to detect structural lesions such as brain tumors and focal atrophy, has a low yield in detecting the offending temporal lobe in MTS patients. MR has consistently demonstrated a higher sensitivity than CT to potential seizure foci in the temporal lobe [2–9]. MR not only provides an altered signal more often, but provides artifact-

free axial and coronal scans of the temporal lobe, depicting the underlying anatomy more clearly.

Pathologic studies over many years have demonstrated that MTS is associated with seizures arising from the temporal lobe [10, 11]. With rare exceptions [12], MTS cannot be detected on CT scans. Despite the findings of some studies [13], MR does appear to offer localization of the epileptogenic focus in a substantial number of seizure patients. Kuzniecky et al. [7] demonstrated abnormal signal in 11 of 14 patients with severe sclerosis pathologically, and in six of 12 patients with mild to moderate sclerosis. High-intensity signals ipsilateral to the epileptogenic focus were documented also in 65% of patients studied by this group [7].

In our previous study of 59 patients in whom seizures arose throughout the brain, EEG was positive in 67%, MR was positive in 53%, and CT was positive in 42% [9]. In the present study, abnormal signal was demonstrated in 27 (69%) of 39 patients with temporal lobe seizures. Maximum increased signal (3+) was displayed in eight, moderate increased signal (2+) in six, and minimal increased signal (1+) in 13. In group 1, the tumor/cyst subgroup, an increased signal was seen in all 13 patients. Mass deformity was seen in 12 of 13 patients in group 1.

If we analyze only the patients with proved and presumed MTS, an increased signal was displayed in 14 (52%) of 27, which is somewhat less than previously reported [7]. Since the populations are small in both series, the differences between them probably are not significant. However, if the differences are confirmed by larger series over time, one might have to consider that yield on a 0.5-T field strength is higher despite the theoretical advantage of increased signal to noise with increasing field strength [14]. Schorner et al. [8] reported one patient with proved MTS who exhibited what we would have described as a 3+ MR signal. Their group used a 0.35-T MR unit with an SE 1600/70 sequence. It can be inferred from these observations that it is difficult to define universal standards for signal intensity that would be applicable to MR units of varying field strengths. One can suggest that SE pulse sequences with fairly long TRs (2000–2800 msec) and long TEs (80–100 msec) would be most likely to show increased signal.

The severity of the pathologic changes appears to correlate roughly with the signal intensity on MR. If one notes a significantly increased signal intensity (3+), this indicates that the patient most likely has a neoplasm. In contrast, if the patient has a minimal increase in signal (1+), the lesion is less likely to be a neoplasm and more likely to be MTS. Patients with an intermediate signal increase (2+) could have either tumor or, less commonly, MTS. One of the patients in our series could not be categorized so neatly. That patient (case 26) was found to have an intermediate signal (2+) on MR, yet on pathologic study was found to have normal neocortex and normal hippocampus (Fig. 4). Probably the best explanation for this is that there was a sampling error and the pathology was omitted. While such an observation is disturbing, it is important to note that the patient is seizure-free postoperatively and the MR abnormality did correlate with the side of the epileptogenic focus.

Calcification was seen on CT in two patients, one with an astrocytoma and the other with hamartomas plus MTS. While calcifications are seen much better on CT, frequently they can be suspected by MR. Calcification was recognized on MR in one of our two patients. On the basis of our observations and those of others, when calcification is detected, the first diagnosis should be tumor.

Minimal dilatation of the temporal horn is not helpful in establishing the offending temporal lobe. Two patients in the entire series were recorded as having atrophy. One patient was in group 2 and one was in group 3. In only one patient (case 29) did the atrophy correlate with the pathologic findings in the temporal lobe.

Increased signal in the temporal lobe as described in our series has been helpful in clinical decision making. When the signal alteration is very intense it offers strong support for a structural lesion that is likely to be approached surgically. Second, when the EEG data are not clear-cut as to the side of the lesion, demonstration of increased signal may obviate depth electrodes. The detailed data for this decision will be presented in a separate report.

MR appears to be an excellent imaging method for studying patients with partial complex seizures. While demonstration of abnormal MR signal is important in the assessment of the need for potential lobectomy, it must be correlated closely with the electrophysiologic localization of a focal temporal lobe abnormality.

In summary, we have compared the MR and pathologic findings in 39 patients who had lobectomy for medically intractable temporal lobe seizures. Thirteen (33%) of the patients had neoplasms/cyst. Generally, very-high-intensity signals were seen in the temporal lobe in these patients. In 14 (52%) of the remaining 27 patients, a less intensely increased signal was seen in one temporal lobe that corresponded to the side of the epileptogenic focus as shown by EEG. One patient was entered into both group 1 (hamartoma with 3+ signal) and group 2 (MTS with 1+ signal). Those patients with very high signal intensity all turned out to have neoplasms (and one glial-lined cyst); those with subtle but definite tissue signal abnormality (1+) all turned out to have either MTS, or the pathologic examination was incomplete. This study along with others suggests that MR is an excellent neuroimaging method in temporal lobe epilepsy.

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