

CHRONIC EPILEPSY DOES NOT CAUSE MEASURABLE STRUCTURAL CHANGES ON MRI

The Structural Consequences of Newly Diagnosed Seizures

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Ann Neurol 2002;52:573–580

Intractable epilepsy may be associated with widespread structural cerebral damage. We determined whether structural damage occurs to the hippocampus, cerebellum, and neocortex in the first few years after a diagnosis of seizures. Sixty-eight patients older than 14 years with newly diagnosed seizures and 90 matched controls underwent serial magnetic resonance imaging (MRI) brain scans 3.5 years apart. Using quantitative analysis of serial scans, we determined changes in hippocampal volume, hippocampal T₂ relaxometry, and total and regional brain volumes. Thirty-four (50%) patients had recurrent unprovoked seizures between baseline and follow-up scans. One patient with preexisting hippocampal sclerosis (HS) did not develop progressive hippocampal damage. Group analyses found no difference in change in cerebral measures between patients and controls or between patients with and without recurrent seizures. Significant quantitative changes in individuals were largely attributable to preexisting cerebral lesions or alcohol abuse. Subtle changes detected in individuals over a 3.5-year period but were not related to a history of overt seizures. Our results show patients with newly diagnosed seizures are not generally at increased risk of seizure-induced structural cerebral damage as detected with MRI. Cerebral damage may occur before the onset of seizures or develop insidiously over a more prolonged period

preoccupation among patients and clinicians alike. Prospective data on this issue, however, are sparse and remain the source of controversy. In this study, Liu et al. began to provide some answers. They studied MRI changes occurring within 3.5 years from the onset of the seizure disorder in patients with newly diagnosed epilepsy beginning after age 14 years. These authors compared magnetic resonance imaging (MRI) changes of 90 outpatients, recruited from a local population to those of 90 age- and gender-matched controls, recruited from the same community base, over the same time period. Sixty-eight patients and 90 controls had a second MRI after a median period of 42 months. The authors measured hippocampal, cerebellar, total brain, gray matter and white matter, and intracranial volumes, as well as hippocampal T₂ maps (HCT2). Eighteen patients had cryptogenic, and 24, symptomatic partial epilepsy. Twenty-six patients had generalized epilepsy (four idiopathic, 16 cryptogenic, and 16 symptomatic). Patients with temporal lobe epilepsy (TLE) were significantly more likely to have recurrent seizures than were those with extratemporal or generalized epilepsy. There were no group differences in any of the volumetric measures. Furthermore, the seizure frequency, by seizure type had no impact on any volumetric or signal change, with the exception of patients with partial epilepsy without secondary generalization, who were more likely to have a higher HCT2 combined signal with a higher seizure frequency.

The data from this study suggest an absence of structural changes within the first 4 years of the seizure disorder, even in patients with recurrent seizures. These findings contrast with the evidence of hippocampal, cerebral, and cerebellar atrophy reported in cross-sectional studies of patients with refractory epilepsy. The differences between these studies and that of Liu et al. are that the former included patients with childhood-onset seizures [and hence, with mesial temporal sclerosis (MTS)] and patients with a history of status epilepticus. For example, O'Brien et al. (1) reported progressive hippocampal atrophy over a 4-year period in a 28-year-old man with intractable TLE of long-standing duration.

The aim of this study was to focus on the structural changes resulting from recurrent seizures. Does the absence of such changes imply an absence of functional changes ex-

COMMENTARY

A progressive cerebral damage resulting from the cumulative effect of epileptic seizures has been a source of great

pressed as cognitive deterioration or de novo psychiatric comorbidity? Some studies suggest that patients with TLE and normal MRI are less likely to have cognitive deficits compared with those with atrophy of mesial structures (2). Whether this remains true in extratemporal or generalized epilepsies may not follow these rules. For example, Mirsky et al. (3) demonstrated disturbances in attention in a group of children with absence seizures. Furthermore, Janz (4) reported a higher frequency of psychiatric disturbances in patients with juvenile myoclonic epilepsy compared with controls. Finally, the absence of structural changes is not necessarily predictive of a good seizure outcome, as intractable epilepsy in patients with normal MRI studies is recognized in most epilepsy surgical programs. Paradoxically, the absence of atrophic hippocampal volumes in intractable TLE is predictive of a less favorable postsurgical seizure outcome than in those with MTS (5).

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References

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