

HELPFUL DATA, BUT LESS CERTAINTY

Childhood Mesial Temporal Sclerosis. Ng YT, McGregor AL, Duane DC, Jahnke HK, Bird CR, Wheless JW. *J Child Neurol* 2006;21(6):512–517. The prevalence and clinical characteristics of mesial temporal sclerosis have not been well studied in children. All brain magnetic resonance imaging (MRI) reports of children less than 14 years of age were reviewed from two tertiary institutions. A 52-month period from one institution and a 37-month period from the other were reviewed. All reports of definite or possible mesial temporal sclerosis were noted. These patients' MRIs were then reviewed to confirm the MRI diagnostic criteria of mesial temporal sclerosis. The charts of the patients who satisfied these criteria were reviewed in detail. Three thousand one hundred brain MRI reports were reviewed. Twenty-six reports of mesial temporal sclerosis were found. Twenty-four of the 26 films satisfied the criteria of mesial temporal sclerosis by MRI after the films were reviewed. The prevalence among all pediatric brain MRI studies was 0.77%. All patients had presented with seizures, that is, there were no "incidental" findings of mesial temporal sclerosis. Four patients had a history of febrile seizures. Mesial temporal sclerosis is an uncommon finding in children, but when it occurs, it is always associated with epilepsy. Asymptomatic mesial temporal sclerosis or mesial temporal sclerosis not associated with a seizure disorder did not occur in our series. Febrile seizures can occur in association with mesial temporal sclerosis presenting in childhood.

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COMMENTARY

This careful, retrospective study by Ng and colleagues of a patient MRI database serves as a reminder that mesial temporal sclerosis (MTS) may already be present in childhood. The mean age of children whose MRI disclosed abnormalities consistent with MTS was 6.9 years (range: 0.7–12.7 years). Two factors likely contributed to detection of MTS in 24 (3.9%) of the 623 pediatric epilepsy patients: (a) use of a specific epilepsy protocol with fine-cut coronal T₂-weighted sequences perpendicular to the hippocampi (supplemented by coronal fluid-attenuated inversion recovery (FLAIR) images from one institution), and (b) the severity of seizure disorders of these 24 patients. Among 22 patients for whom therapeutic data were available, 11 (50%) were daily taking more than one antiepileptic drug. Moreover, 13 of the 24 patients had a potentially epileptogenic MRI lesion in addition to MTS, a significant neurological antecedent (e.g., encephalitis, trauma), or both. Curiously, febrile seizures occurred in only 4 of the 24 children, 2 of whom had potentially epileptogenic factors. These data support the concept that MTS may result from one or more of several pathophysiological factors (1).

Although MTS appeared only among epilepsy patients and not among the 2,477 children with other conditions, the implied high specificity is negated by their employment of the MTS-revealing epilepsy protocol only for seizure disorder patients. Subsequent application of the epilepsy protocol to a set of sequential patients referred for MRI for any reason may more confidently establish specificity. Results may relate to findings by Benbadis et al., which identified both hippocampal atrophy and T₂ signal abnormalities in 8 (4%) of 204 patients without a seizure history who underwent MRI (2). Thus, the Ng et al. study (pediatric epilepsy patients) and the Benbadis study (nonepilepsy patients) disclosed abnormalities compatible with MTS in 3.9% and 4%, respectively!

Some epileptologists have proposed that two conditions must coexist for MTS to develop: (a) an initial precipitating injury (e.g., prolonged febrile seizure, CNS infection, head trauma) and (b) any factor that increases vulnerability to neuronal injury. Accumulating evidence supports various candidates for this second mechanism—among them, hippocampal dysgenesis and a genetic component (3,4). As only 4 of the

24 MTS patients in the Ng et al. study had febrile seizures, other tandems (such as trauma and hippocampal dysgenesis) may suffice.

Despite the presence of MTS, clinical descriptions and video-telemetry disclosed complex partial seizures in only 16 of 22 patients for whom data were available in the Ng et al. study. It is known that temporal lobe seizures in young children (< 6 years) may manifest as symmetrical tonic or clonic events of the limbs (5). In fact, EEG-documented seizures originating in a temporal lobe that contained MTS were identified in only 8 of 18 patients with an identified epileptogenic focus. This finding may reflect the dual pathology of MTS and a separate epileptogenic area. (6).

Advances in neuroimaging disclose abnormalities, such as MTS, that may have escaped earlier techniques. Data of this study underline the need for clinicians to realize that lesion-based temporal lobe epilepsy may begin in childhood. However, establishment of specificity, that is, clinical significance of any abnormality revealed in a laboratory test, remains a challenge.

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