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SHORT COMMUNICATION

Pathophysiological implications of focal cortical dysplasia of end folium for hippocampal sclerosis

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Summary Dual pathology is a well-known cause of temporal lobe epilepsy (TLE), but TLE associated with dual pathology within the hippocampus has rarely been reported. We describe a patient with medically refractory TLE due to a focal cortical dysplasia of the end folium of the left hippocampus (i.e. CA4) and left hippocampal sclerosis (HS) that was successfully treated with left anterior temporal lobectomy and speculate on the interaction between end folium sclerosis and HS.

Temporal lobe epilepsy (TLE) is the most common cause of medically intractable partial epilepsy, which is commonly associated with hippocampal sclerosis (HS) (Engel, 1998). Dual pathology is increasingly recognized among the TLE patients that undergo surgery with up to 20% in some series (Li et al., 1999). Dual pathology in most such cases refers to the presence of HS and an extrahippocampal lesion. However, dual pathology with HS plus focal cortical dysplasia restricted to the end folium of the hippocampus (i.e. CA4 sector) has rarely been reported (Thom et al., 1999). We describe a patient with medically intractable left TLE associated with such dual pathology, which may have histopathological implications for the development of HS.

Case report

A 20-year-old, right-handed woman experienced two generalized tonic—clonic seizures (GTCS) 1 month apart at age 14 years, a third GTC seizure 2 years later plus recurrent daily stereotyped complex partial seizures with motionless stares and loss of awareness lasting up to 30 s each that were variably associated with oral or manual automatisms. One non-convulsive status epilepticus (NCSE) occurred at

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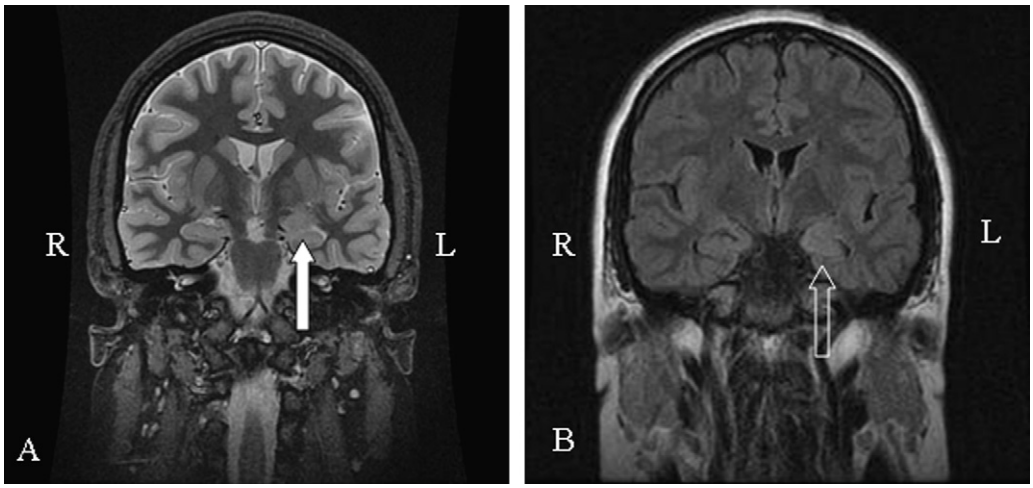

Figure 1 Coronal T_2-weighted (A) and coronal FLAIR (B) anatomical cranial MRI shows increased signal intensity in the left hippocampus (arrows).

age 18 years. A combination of daily carbamazepine 600 mg, clobazam 20 mg, and levetriacetam 1000 mg was ineffective. Medical and family history plus physical examination were non-contributory. In particular, she had no prior history of perinatal complications, childhood febrile convulsions, or head trauma. MRI showed increased signal intensity in the left anterior mesial temporal region (Fig. 1). FMRI and intraoperative electricocortical stimulation showed left hemispheric representation for language. Neuropsychological testing showed low average range intelligence and impaired verbal memory. Video-EEG telemetry with scalp electrodes recorded frequent interictal epileptic spikes, focal slowing, and seizures (12 clinical, 5 electrographic) over 5 days that were all localized to the left temporal region as the principal focus (Fig. 2). She has remained seizure free since her left anterior temporal lobectomy 3.5

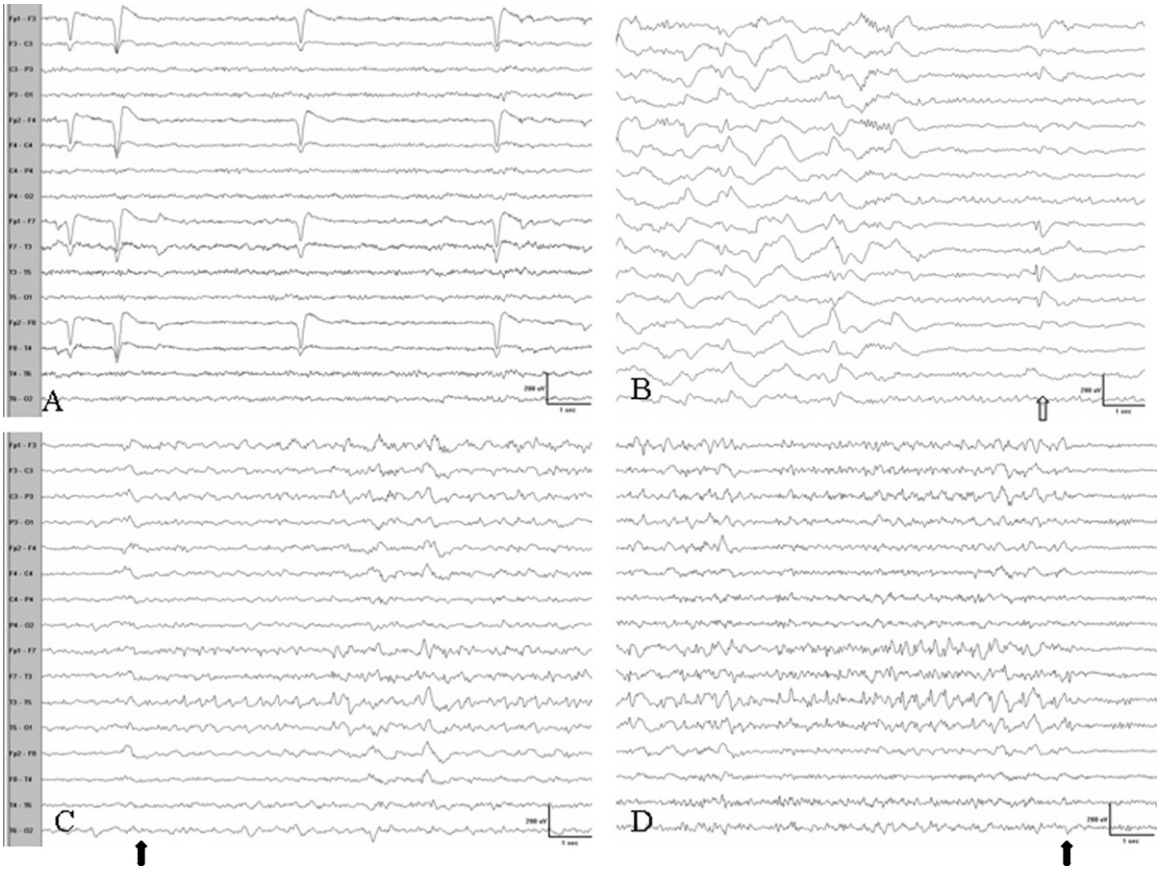

Figure 2 EEG with scalp electrodes using 10—20 International System of electrode placement: (A) focal slowing of the left temporal region during wakefulness. (B) An epileptic spike in the left temporal region during sleep (open arrow). (C and D) A seizure with predominant involvement of the left temporal region lasting about 18 s (solid arrows indicate the onset and ending of this seizure).