Complex partial seizure mimicking psychotic reaction in an adolescent

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SUMMARY: Serdaroğlu A, Gücüyener K, Bilir E, Soysal Ş. Complex partial seizure mimicking psychotic reaction in an adolescent. Turk J Pediatr 2001; 43: 251-253.

A previously healthy 15-year-old boy initially diagnosed to have acute psychotic reaction had a history of a single generalized seizure and prolonged amnestic states of varying intensity and duration. An ictal electroencephalogram (EEG) showed bitemporal ictal discharges starting from the left side. Carbamazepine was started. A magnetic resonance imaging (MRI) obtained on the 10th day of the antiepileptic therapy showed increased signal intensity on the T2 weighted images. The patient's memory function markedly improved during 10 months' follow-up with antiepileptic treatment, although he described brief attacks of dizziness. A repeat MRI examination showed normal findings. The amnestic states were thought to be due to frequent complex partial seizures, and transient MRI changes to hippocampal edema. This case illustrates the importance of epileptic disorders in the differential diagnosis of psychiatric conditions.

Key words: complex partial seizure, psychotic reaction, MRI changes.

The hippocampus, a deep structure of the mesiotemporal lobe, is critically important for the regulation of short-term memory[1]. Electrical stimulation or transient ischemia of the mesiotemporal lobe causes transient global amnesia (TGA)[2,3]. The pathophysiology of TGA is still unclear; factors other than ischemia influencing the deep temporal structures may be responsible[4]. We present a 15-year-old boy with complex partial seizures and prolonged amnesia who was misdiagnosed with a psychotic reaction.

Case Report

A 15-year-old right-handed boy was admitted to the emergency room with a history of a single generalized seizure and loss of consciousness. He had generalized tonic-clonic movements lasting for two or three minutes followed by 30 minutes of somnolence. No aura was described. The history included memory disturbances for the past two months: the patient could not remember most of the events that had occurred during this period. Once he had entered another classroom instead of his own. He had been seen by a child psychiatrist and diagnosed with an acute psychotic reaction triggered by his grandmother's death. His past family and medical histories were significant for an uncomplicated febrile convulsion at age two. He had been a healthy child otherwise. Two previous electroencephalograms (EEG) and cranial computerized tomography (CT) were normal.

In the emergency room he was an alert pleasant boy with stable vital signs. Physical examination was normal. On mental status examination, he was oriented to person, place, year and month, but not to date. Serial events and calculations were intact. Judgement and interpretation were normal. He had some disturbance in immediate- and short-term memory, and he could not remember any details of subjects told in the class. However, he was not seriously concerned with his memory impairment. The rest of the neurological examination was normal. Laboratory studies including complete blood count (CBC), electrolytes, cerebrospinal fluid antibodies, herpes simplex type I-II, rubella, rubeola, adenoviruses, Epstein-Barr virus (EBV) and oligoclonal band were all negative. A routine scalp 16 channel EEG and cranial CT scan with contrast injection were normal. During long term EEG monitoring there were bitemporal ictal discharges starting from the left side and quickly spreading to other parts of both hemispheres. The initial abnormality consisted in 4-5 Hz, 30-

70 microvolt rhythmic sharp waves beginning in the left temporal region and becoming bilateral in one second, with maximal amplitude at electrodes T3 and T5. The discharges lasted 30 seconds and were followed by diffuse 2-3 Hz irregular slowing with no lateralization. He was unable to carry on a conversation during ictal discharges. Medical treatment with carbamazepine (CBZ) (400 mg/day) was initiated. Magnetic resonance imaging (MRI) was obtained at the 10th day of the CBZ treatment. T_2 weighted CTR/TE/NER showed increased signal intensity at the hippocampi bilaterally (Fig. 1). Three months later, his memory function was improved. On nine months' follow-up he had no recurrent amnestic episodes, but had infrequent, brief attacks of dizziness with transient impairment of consciousness. EEG after 10 months was completely normal and the follow up MR examination demonstrated no signal change of the hippocampus (Fig. 2).

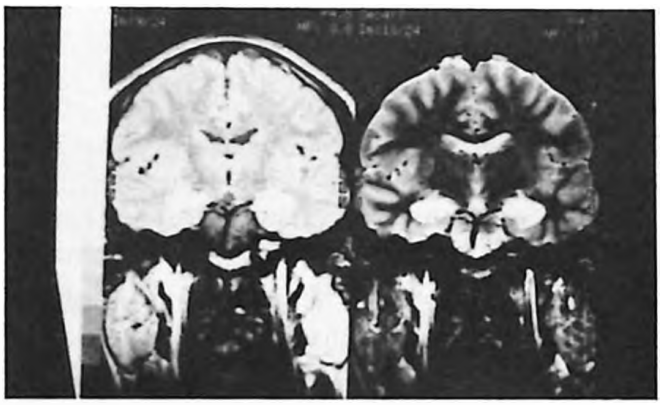

Fig. 1. T2 weighted images of the initial MRI demonstrated high signal intensity of the bilateral hippocampal region with no apparent structural change.

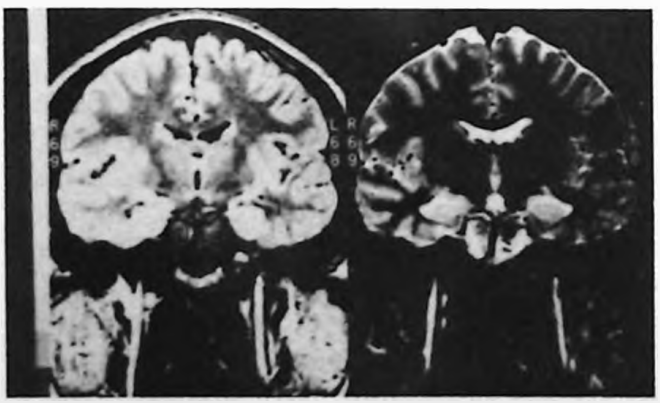

Fig. 2. Ten-month follow-up MRI demonstrated marked improvement with no obvious signal range of the hippocampus.

Discussion

This patient's amnestic states can be explained by both ictal and postictal changes. The hippocampus has been known for a long time to be critical for the formation and recall of short-term memory[1]. A previous study has suggested interictal epileptiform discharges due to transient global amnesia in epileptic patients[4].

The hippocampal lesion in our patient's MRI was a focal signal intensity change; no mesiotemporal sclerosis or hippocampal atrophy was observed. The differential diagnosis of focal signal intensity changes in patients with complex partial seizures includes vascular, neoplastic and inflammatory disorders. This differentiation can be especially difficult in patients with status epilepticus. The reversibility of the findings on consecutive MRIs in accordance with clinical improvement of memory supports hippocampal edema[6-8]. Reversible focal CT or MRI abnormalities have been reported in epilepsy due to cerebral edema resulting from increased energy requirement during repetitive ictal discharges, increased regional cerebral blood flow, or regional breakage of the blood brain barrier[5,6]. Sammantino et al.[5] described three patients with striking focal cerebral edema whose neurological deficits resolved as the edema regressed gradually, although the resolution of images on serial CT scans usually lagged behind clinical improvement.

This patient probably had frequent temporal lobe complex partial seizures in the previous two months. He presented with gradually increasing memory disturbance and unusual behavior as the most prominent complaints. More subtle symptoms such as brief episodes of unconciousness or dizziness were probably missed by his parents, pediatrician and child psychiatrist. He therefore had received the diagnosis and treatment of acute psychotic reaction. This case exemplifies the importance of careful investigation for epileptic disorders in patients presenting with psychiatric symptoms.

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