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CASE STUDY

Executive Dysfunction in Frontotemporal Epilepsy Due to Traumatic Brain Injury

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Abstract

Traumatic brain injury has been recognized as a cause of epilepsy. Epilepsy could impair cognitive function and the cognitive profiles are as heterogenous as the epileptic syndromes themselves. In most idiopathic epilepsies, cognition is only mildly deteriorated or even normal, whereas symptomatic epilepsy disorders are accompanied by focal deficits according to the specific functions of the respective areas. Poor cognitive outcome is generally associated with an early onset and a long duration of the seizure and with poor seizure control. We report a 31-year-old man, with a specific generalized tonic clonic seizure during sleep since one year ago. MRI shows cystic encephalomalacia in left frontal lobe due to head trauma 3 years ago. This patient had executive function disorder which was caused by uncontrolled frontotemporal epilepsy due to traumatic brain injury. We propose AED and cognitive stimulation to be performed routinely to prevent further cognitive deterioration.

Keywords: *Executive function, Frontotemporal epilepsy, Traumatic brain injury.*

Background

As proposed by the International League against Epilepsy (ILAE) and the International Bureau for Epilepsy (IBE) 2005, epilepsy is a disorder of the brain characterized by an enduring predisposition to generate epileptic seizures, and by the neurobiological, cognitive, psychological and social consequences of this condition. Epilepsy is a variety of disorders reflecting underlying brain dysfunction that may result from many different causes; one of them is traumatic brain injury [1].

The term traumatic brain injury (TBI) is referred to damage to the brain, either on impact or subsequently due to processes initiated by that impact [2].Post-traumatic epilepsy is defined as one or more unprovoked seizures that occur at least a week after TBI [3].Most post-traumatic epilepsy occurs during the first year after injury, although they can also occur for many years afterwards. The incidence of seizures due to trauma ranges from 1.9-30%. As many as 9.1 per 100 persons developed epilepsy in the first three years after TBI [2, 4]. Like many other changes of cerebral processes, chronic epilepsy influences cognition, which is defined as the totality of capacities underlying complex adaptive behaviour. In most idiopathic epilepsies, cognition is only mildly deteriorated or even normal, whereas symptomatic epilepsy disorders are accompanied by focal deficits according to the specific functions of the respective areas. The poor cognitive outcome is generally associated with an early onset and a long duration of the seizure and with poor seizure control [5].

Case

A 31 years old man complained forgetful since six months ago. He complained forgets things in his daily routines, had difficulties in imagine and compare things, and also difficult to do even simple arithmetic's. These complain did not disturb his activity daily living, he could perform his daily routines by himself. Since a year ago he was known to have seizure during night sleep. The seizure always preceded by screaming, followed by stiff and jerking in his entire body, considered a generalized, tonic-clonic seizure. The seizure lasted less than 5 minutes, repeatedly about twice a month. Following the seizure patient looked disoriented for a relatively short time and then back to his normal consciousness. The patient never looked for medication before because there

was no disturbance to his daily life. The patient had a history of brain injury about three years before the first seizure due to motorcycle accident. He was admitted to the hospital but no serious brain injury found. Head CT scan was within normal limit, and he gained his full consciousness by day after the accident. There was no neurologic deficit found. We performed several diagnostic examinations for the patient. We did not find any neurologic deficit in physical examinations. Neurobehavior examinations showed patient failed in executive function examinations such as calculation, phonemic and animal verbal fluency test, abstraction, trail making test form B, and Luria's motor sequences. He also had difficulties in backward digit span and go-no-go test but passed the clock drawing test. There was no obvious disturbance in memories evaluation including immediate, recent, and remote memories using the CERAD examination. Head MRI (Figure 1) with and without contrast showed cystic encephalomalacia in the left frontal lobe. It may be due to head trauma before. Electroencephalogram (Figures 2 and 3) showed epileptiform discharge in left frontotemporal as 1 Hz, high amplitude, spike-wave in F7-T3 during hyperventilation.

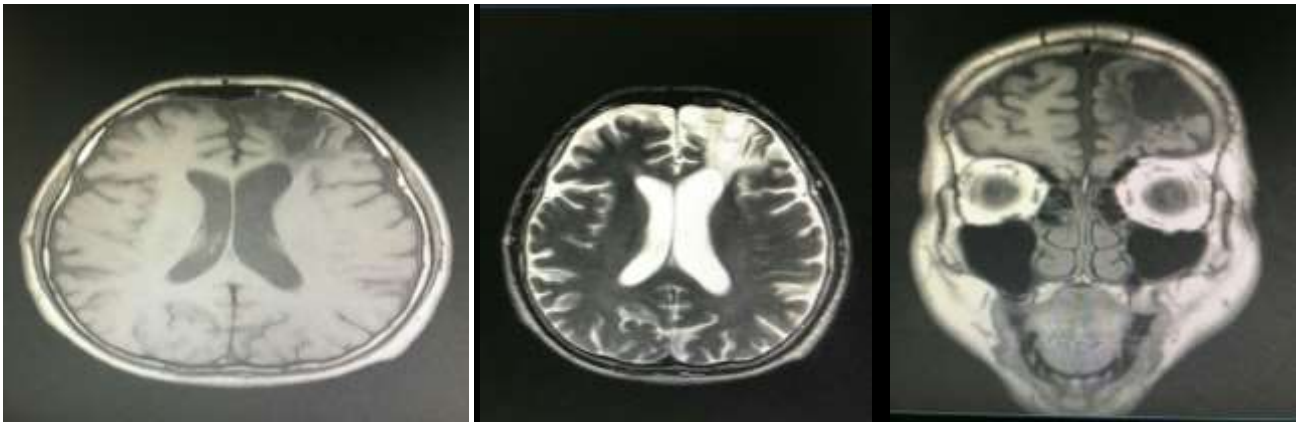

Figure 1: Head MRI (Axial T1, Axial T2, Coronal T1)

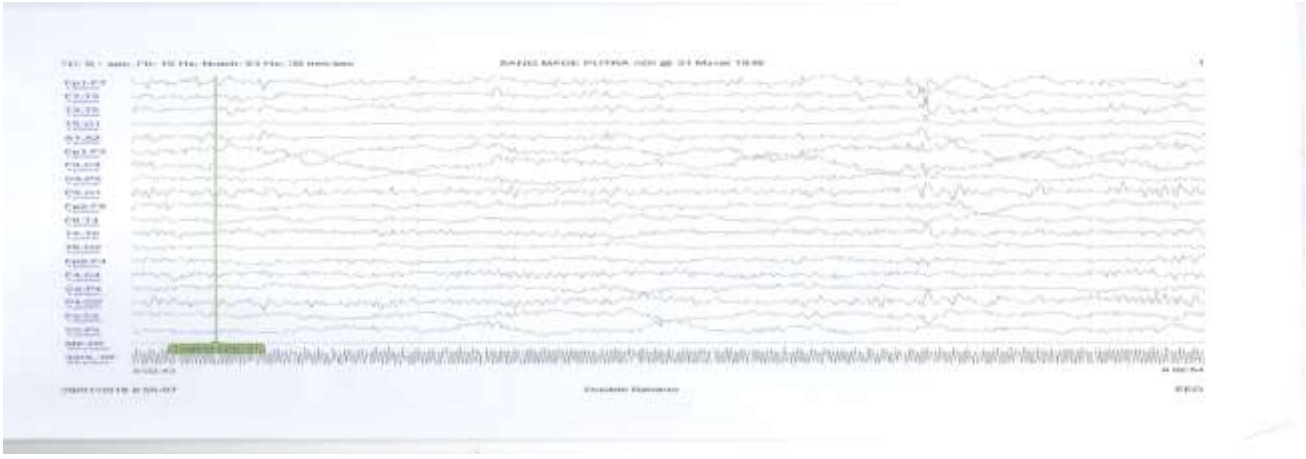

Figure 2: Bipolar EEG

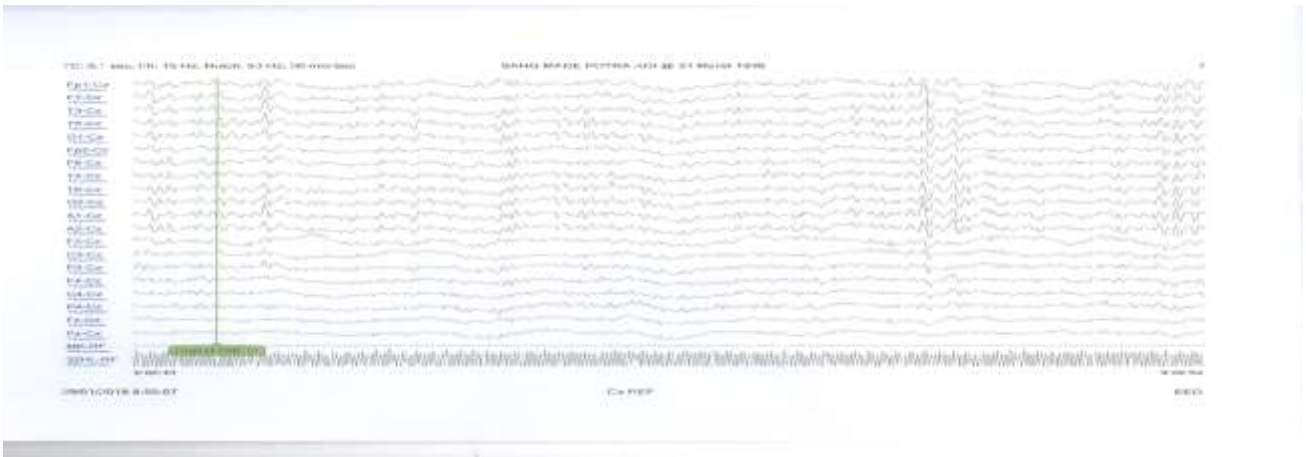

Figure 3: Unipolar EEG

Discussion

The concept in the definition of epilepsy is an enduring alteration in the brain that increases the likelihood of future seizure. According to ILAE, epilepsy is diagnosed clinically, but the head MRI and electroencephalogram (EEG) are supporting to show whether there is anatomical or functional disturbance [1].This patient got motorcycle accident three years before his first seizure. He gained his full and clear consciousness the day after the accident, indicating there was no severe brain injury. The seizure repeated more than ten times in a year.

Then he is diagnosed with post-traumatic epilepsy. Patients head MRI showed cystic encephalomalacia in left frontal lobe, and the EEG showed epileptiform discharge in left frontotemporal, supporting the diagnosis. Some studies show the incidence of epilepsy after brain injury is related to age, the severity of disease, imaging results, treatment, early-stage seizures, and persistent focal abnormalities on EEG more than one month after injury [2, 6].

The other older studies also show that premorbid chronic alcoholism likely increases the risk of the development of seizure, but the results were not tested for significance, and still need further investigation [2, 7].This patient had nocturnal, generalized, tonic-clonic seizure preceded by screaming.

This sleep activation pattern could be seen in frontal and temporal lobe epilepsy. According to the theory, motor semiology is characteristic of frontal lobe seizures. This motor semiology can be divided into elementary motor signs which consist of clonic movements and tonic or dystonic contraction and/or posturing as well as head and/or eye version, and gestural motor behaviour which commonly observed in prefrontal seizures and produce complex motor behaviours [8].

On the other hand, cardinal semiology of temporal lobe seizures consists of prodrome, aura, altered consciousness, amnesia, and automatism. These form of epilepsy sometimes overlapped, frontal lobe epilepsy can be mistaken with temporal lobe epilepsy, and so does the other way. Temporal lobe seizures are the most frequent site of origin of partial seizures, while frontal lobe seizures are the second one [9]. EEG showed spike-wave in F7-T3 which located in left posterior frontal lobe and left anterior temporal lobe. This area is border between frontal and temporal lobe. Considering the patient's symptoms that overlapped between frontal lobe and temporal lobe features, we referred the patient to have frontotemporal MRI. About six months after he developed epilepsy, patient complained forgetful in his daily routines. He had difficulties in imagine, compare things, and arithmetic's even the simple one, but still could perform his daily activities. The disturbance is suspected because of the recurrent and uncontrolled epilepsy in past year because of the time the symptom appeared was more related to the timing of seizure.

Age at onset of the disease, duration of the disease, the total number of seizures, seizure frequency and severity are the possible factors influence cognitive function in patient with epilepsy. General tonic-clonic seizures are more likely to impair cognition than simple and complex partial seizures, and interictal epileptic activity accounts for a much smaller part of the cognitive effects than previously thought [5].

The frontal lobes, especially the prefrontal cortex, play an important role in higher-level cognitive processes, such as executive functioning skills. Executive functions involve several types of processing including decision making, planning, focused attention, concept formation, response inhibition, cognitive flexibility, and working memory.

According to the role of the frontal lobes, it is not surprising that studies have consistently shown that adults with epilepsy involving frontal cortex would exhibit impairments in several executive functioning skills [5, 10]. A number of neuropsychological tests have been designed to evaluate executive functions and are in widespread use, such as Digit Span Forward and Backward, Verbal Fluency Test, Trail Making Test form A and B, and Luria's Motor Sequences [11].

In further studies on subfunctions of memory, there is tight interaction of temporal and prefrontal areas in memory processes, therefore patients with temporal lobe epilepsy may also have impaired frontal-lobe functions, particularly when they have secondarily generalised tonic-clonic seizures.[5]

Neurobehavior examinations in this patient reflect executive dysfunction. Studies reported relative impairment on a number of cognitive tasks in patients with frontal lobe epilepsy compared to temporal lobe epilepsy, including measures of manual motor skill as well as tests of executive function, and greater impairment is noted in the left frontal lobe group compared to the right. However, some of the measures used did not effectively differentiate the frontal lobe epilepsy patients from those with temporal lobe epilepsy [11, 12].

Thus the executive dysfunction in this patient may be caused by the frontal lesion and also by uncontrolled epilepsy. Memory disturbances are one of the main problems in both frontal lobe epilepsy and temporal lobe epilepsy. It has been postulated that the mechanism underlying working memory dysfunction in patients with frontal lobe epilepsy and temporal lobe epilepsy is the propagation of seizure activity from the temporal lobe to the frontal lobe through these numerous connections [10, 13].The memory examinations in this patient was normal, not consistent with the literature which has found impaired attention and working memory in patients with frontal lobe epilepsy and temporal lobe epilepsy.

To prevent any further cognitive deterioration, this patient has to take an antiepileptic drug (AED) to control the seizure. Androsova et al [14].Reported that carbamazepine and valproate were the most commonly used first AEDs in focal epilepsy patients, while lamotrigine and levetiracetam were the most second or third AEDs used. Treatment of epilepsy should start with AED monotherapy. Older-generation AEDs are effective but have tolerability and pharmacokinetic disadvantages. Giving medication of epilepsy patients largely remains a process of trial and error, and any evidence of superiority of one AED over another is lacking [15].

Another method to prevent cognitive deterioration is cognitive stimulation. The executive functions were known to have a good response to stimulation. The executive functions that responded best to training were attention shifting and working memory, while planning and execution speed did not seem to change with stimulation. Moro et al [16]. Indicated that an improvement in executive functions may have a positive effect on memory and general cognitive abilities.

It shows that without specific stimulation, the performance of patients with MCI declines over time and that after stimulation the recovery is only partial, so executive functions need to be stimulated right from the early stages of mental deterioration and that the precocity of cognitive stimulation may represent a crucial factor for the intervention to be successful because a decline in executive functions is considered to be a significant risk factor in terms of the fast development of dementia.

Conclusion

Cognitive dysfunction in people with epilepsy is a frequently encountered but also easily overlooked problem. Patients with localised epilepsies generally have deficits in the cognitive functions controlled by the respective areas, as exemplified by memory impairment in temporal lobe epilepsy and executive deficits in frontal lobe epilepsy. In this patient, we proposed AED to control the seizure, and cognitive stimulation should be performed routinely to prevent further cognitive deterioration. Executive functions need to be continuously stimulated over time since cognitive stimulation has a strong impact in the short term and it has effect to maintain efficient functioning in the long term.

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