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Case Report

Neurosurgical treatment of nonconvulsive status epilepticus due to focal cortical dysplasia

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ABSTRACT

We present a rare case of focal cortical dysplasia (FCD) and nonconvulsive status epilepticus (NCSE) treated successfully with early surgical intervention. Our case is a 9-year-old boy whose seizures, characterized by short episodes of loss of consciousness, appeared at the age of 7, and he showed progressive cognitive decline in the following years. NCSE was diagnosed, and his MRI revealed FCD in the left frontal region which was the same side as his EEG abnormality. Following lesionectomy, his NCSE disappeared and cognitive functions improved. Histopathologic analysis of the resected tissue revealed type-IIB FCD. This case illustrates the importance of early surgery to help restore cognitive functions by eliminating the clinical and electrophysiological features of NCSE.

1. Introduction

Nonconvulsive status epilepticus (NCSE) presents with clinical signs such as unexplained changes in behavior and mental status, confusion, or even a severe tendency to sleep, accompanied by almost continuous epileptiform activity in electroencephalography (EEG) [1]. NCSE is observed mainly during the clinical course of patients with drug-resistant epilepsy. Recognition of NCSE especially in pediatric patients with epilepsy is very important for optimal mental development. We present a case of NCSE, which developed in a 9-year-old male with focal cortical dysplasia (FCD) and was successfully treated with neurosurgery.

2. Case report

A 9-year-old boy was born at term without any remarkable perinatal problems. There was no history of epilepsy in his family. His motor development was also normal. He began to have episodes of unconsciousness lasting a few minutes when he was 7 years old. Despite the administration of valproate, carbamazepine, phenytoin, levetiracetam, clobazam alone or in combination, seizures recurred 2 or 3 times a month. At 8 years of age, sharp wave discharges were seen over the frontal regions predominantly over the left side in his EEG but the report for his magnetic resonance imaging (MRI) was normal. On The Wechsler Intelligence Scale for Children-Revised (WISC-R), he scored 81 on his total intelligence quotient (TIQ) testing which is in normal range of intellectual functioning at that time. Since he was 8 years old, he had been taking levetiracetam, clobazam and phenytoin. His family indicated that he had episodes of loss of consciousness lasting for 20 min, 5 to 6 times daily and he began to have a marked decline in school achievement at the age of 9. On the WISC-R, given at the time, he achieved a TIQ of 68, which is indicative of mild mental retardation. This finding suggested that the episodes of NCSE had a negative impact on mental activity even over a short time. Originally a patient at the pediatrics department, he was coincidentally referred to our laboratory for EEG. His EEG was recorded during wakefulness and showed almost continuous generalized spike–wave discharges compatible with left frontal secondary bilateral synchrony, consistent with NCSE (Fig. 1A-B). During this EEG recording, his communication was limited and he was slow in responding to verbal commands. His spontaneous speech was not fluent and he could not even do simple mathematical calculations. He was able to perform only some basic tasks. After intravenous diazepam administration abnormal electrophysiological activity was suppressed, although there was no prominent clinical change. Although

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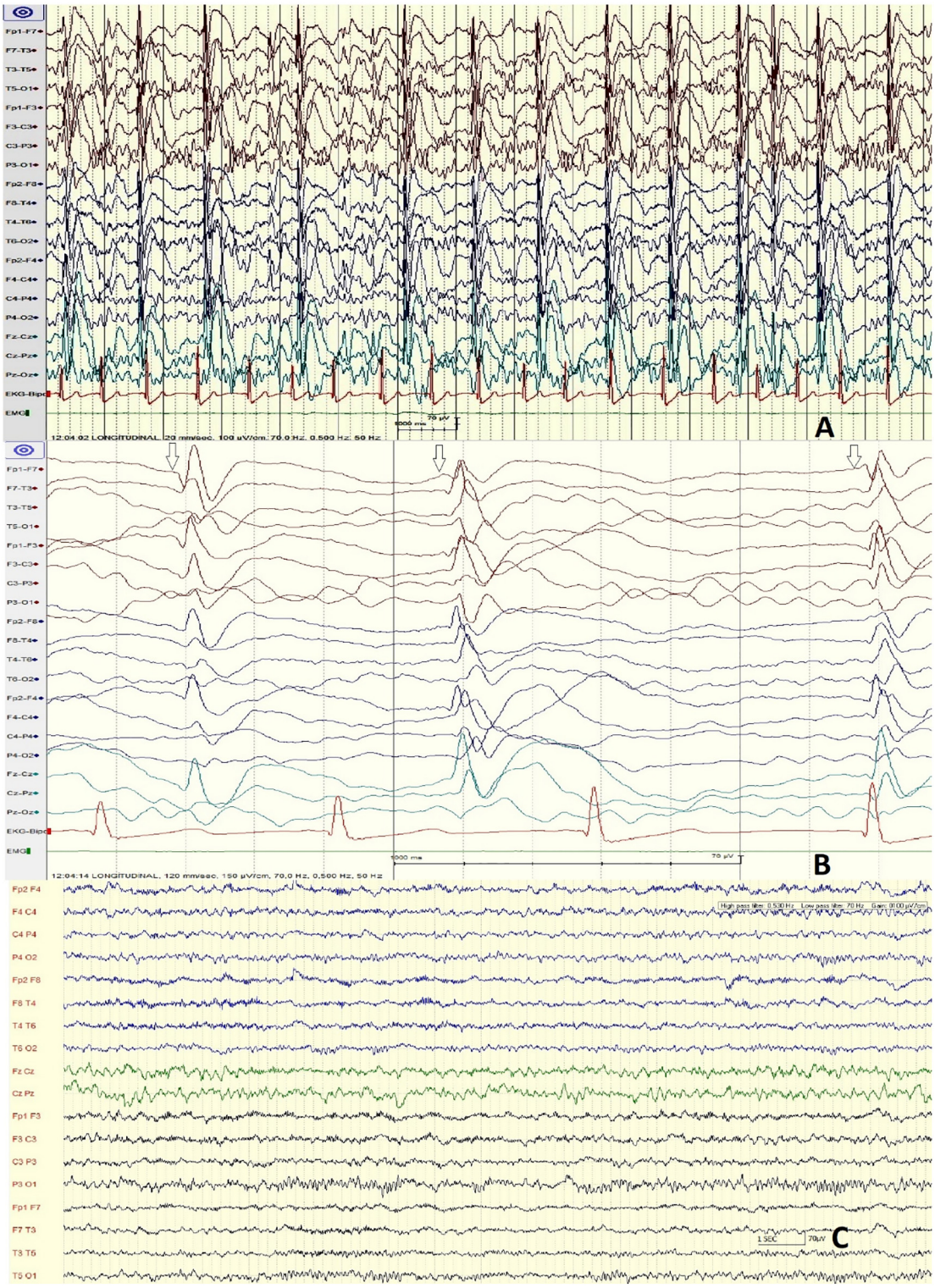

Fig. 1. Preoperative EEG(A–B). A: Continuous generalized spike–wave discharges with left frontal secondary bilateral synchrony and high amplitude. B: Left frontal discharges antecedent and extending to the right hemisphere when amplitude is decreased. C: Postoperative EEG showed no epileptiform discharges.

there was no evidence of electrical status epilepticus during sleep, spikes and/or sharp waves were observed in the frontal regions predominantly on the left side in NREM sleep.

His MRI demonstrated a lesion suggestive of FCD in the anterior cingulate gyrus of the left frontal lobe (Fig. 2A). There were no abnormalities in his blood tests. After preoperative evaluations, he underwent complete lesionectomy (Fig. 2B). Neuropathology of the resected brain tissue revealed FCD type IIB. The specimen was cut into 2–3 mm slices, perpendicular to the cortical surface. Histopathological examination showed a distorted cortical lamination and absence of important amount of the neurons; besides, there were large, bizarre shaped dysplastic neurons and balloon cells with eosinophilic glassy cytoplasms at unusual locations. Immunohistochemically, using the neurofilament (NF) antibody, dense accumulation of NF was observed in these large neurons (Fig. 2C–D). During the postoperative EEG recording, his communication was better, his speech was more fluent and he was giving