Leuprolide Acetate Induced Non-convulsive Status Epilepticus: Case Report

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INTRODUCTION

Nonconvulsive status epilepticus (NCSE) can result from metabolic disorders, central nervous system infections, brain tumors, drugs and toxins that affect the brain. Leuprolide acetate (LA) is a long-acting gonadotropin-releasing hormone (GnRH) analog used for the treatment of central precocious puberty. Adverse neurologic reactions rarely occur with LA treatment. Seizure exacerbations and absence seizures have been reported during LA therapy, especially in patients with brain damage. However, NCSE associated with LA has not been reported until now. Although its mechanism is not well known; GnRH excites neurons with expression of human GnRH receptors, and changes in estradiol and progesterone levels, secondarily, lower the seizure threshold. We report a leuprolide acetate-induced NCSE in a patient who had been treated for precocious puberty.

METHODS

The clinical and electroencephalography (EEG) findings of the patient who developed NCSE due to LA treatment are presented.

CASE

A 9-year-old girl presented with decreased responsiveness and impaired consciousness for 3 days. She had a history of prematurity (32th GW) and right middle cerebral artery territory infarct at newborn period. Consequently left spastic hemiparesis, psychomotor retardation and symptomatic epilepsy were developed. Brain MRI revealed cystic encephalomalacia at the right MCA territory. Carbamazepin was prescribed at the age of 6, and seizures were partially controlled. LA was prescribed at the age of 7 due to puberty precocious . In conclusion, after LA initiated, frequency of seizures significantly increased, and the patient had two NCSE attacks (one was at the first month of LA treatment).

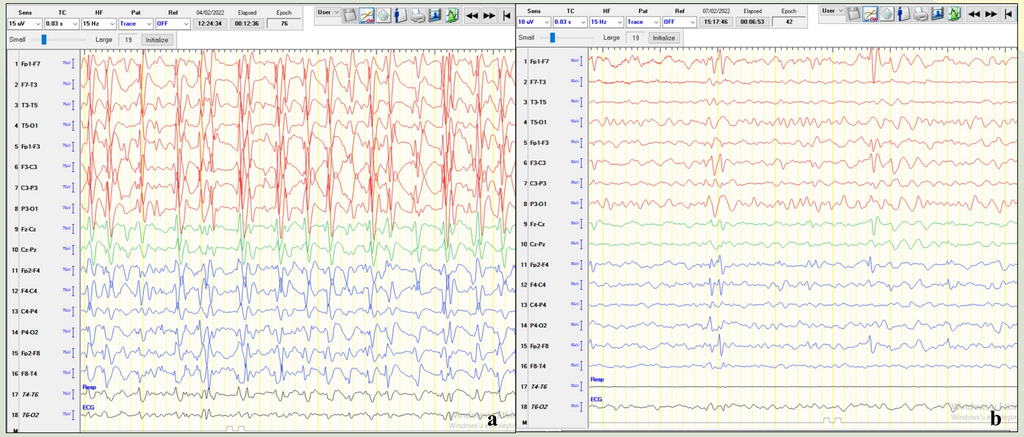

Figure 1a: After initiating LA, EEG shows rhythmic, generalized >2,5 Hz polyspike and slow wave discharges, 1b: After discontinuation of LA , bilateral focal frontal sharp discharges.

There was no other triggering factor. There was no lateralizing finding in her neurological examination. Continuous generalized epileptiform discharges were detected on EEG and suggested NCSE. Discharges disappeared with midazolam infusion. After discontinuation of LA treatment, no findings were observed in the EEG except for the initial interictal findings (focal frontal sharp discharges) and cognitive performance improved.

CONCLUSION

Some drugs may trigger epileptic seizures even convulsive or non-convulsive SE. Discontinuation of the responsible drug is critical to control seizure activity in these patients. Therefore, caution is required for NCSE when using GnRH analogues in pediatric patients with brain damage.