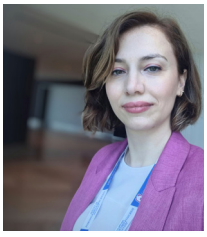
Non-convulsive Status Epilepticus in a Child During Treatment of Central Nervous System Infection

Esra Ülgen Temel

Cengiz Gökçek Obstetrics and Gynecology and Children's Diseases Hospital, Clinic of Child Neurology, Gaziantep, Türkiye



Esra Ülgen Temel MD

Cite this article as: Esra Ülgen Temel. Non-convulsive status epilepticus in a child during treatment of central nervous system infection. *Arch Epilepsy.* 2025;31(2):74-75.

Corresponding Author: Esra Ülgen Temel MD, Cengiz Gökçek Obstetrics and Gynecology and Children's Diseases Hospital, Clinic of Child Neurology, Gaziantep, Türkiye, E-mail: ulgenesra@yahoo.com

Received: 07.03.2025 **Accepted:** 07.03.2025 **Epub:** 07.04.2025 **Publication Date:** 14.05.2025

DOI: 10.4274/ArchEpilepsy.2025.25181

Dear Editor,

I read with interest the manuscript by Gezegen et al.[1] reporting a rare presentation of non-convulsive status epilepticus (NCSE). While the report offers valuable insights, I would like to discuss a different presentation about this disorder.

I present a toddler with normal renal function who had NCSE during treatment of a central nervous system (CNS) infection with ceftriaxone.

A 22-month-old girl had a history of two simple febrile seizures, and presented with a refractory fever, nausea, and vomiting. She was hospitalized, and 100 mg/kg/d IV ceftriaxone was administered. She was referred to the pediatric neurology clinic on the third day of therapy, owing to persistent fever and inability to speak. She was alert and had no focal motor deficits. She was making noise but not speaking. She had mild unsteadiness.

Twenty white blood cells, glucose 49 mg/dL, and protein 35 mg/dL were observed after lumbar puncture. She was diagnosed with meningitis that was partially treated. The therapy was augmented with vancomycin. Despite her heightened awareness and decreased fever, she was still speechless and had an unsteady gait. Her mother's recordings showed she was twitching her mouth and eyes while sleeping. IV midazolam was administered for possible NCSE, which improved her speech. Contrast-enhanced cerebral magnetic resonance imaging was normal. The next-day sleep electroencephalogram (EEG) was normal.

After 8 days, her mother reported a recurrence of speech impairment and unsteady gait. The cerebrospinal fluid (CSF) culture was negative. The ceftriaxone treatment was discontinued. The EEG of the patient revealed rhythmic delta/theta activity with typical spatiotemporal evolution, which is considered NCSE based on Salzburg EEG criteria for the diagnosis of NCSE. IV levetiracetam was administered. She regained the ability to speak the same day, and her gait improved. Her EEG has become normal for her age. During the two-year follow-up, she experienced no seizures and had typical development. Genetic testing for epilepsy was unremarkable.

In our case it was observed that the patient's clinical condition occurred after administering ceftriaxone and disappeared entirely when the medication was discontinued, suggesting that ceftriaxone was the cause of this condition. There are numerous articles in the literature that relate cephalosporins to NCSE.[2,3] The incidence of CNS adverse effects is higher in individuals with renal failure, primarily attributed to elevated medication levels in the systemic circulation. When the blood-brain barrier is compromised, as observed in our patient with meningitis, the CSF concentration of cephalosporins may increase, and CNS toxicity may ensue.[4] Speech was the most impacted in our case. Speech impairment is a recognized consequence of disorders affecting the electrical activity of the brain, such as Landau-Kleffner syndrome and electrical status epilepticus in sleep in children.[5] NCSE should be considered in the presence of neurological symptoms during cephalosporin treatment, irrespective of renal failure.

Ethics

Informed Consent: Written and verbal informed consent was obtained from the parents for the publication of this report.

Acknowledgments

I would like to express our gratitude to the patient and her family for cooperating in this report.

Footnotes

Financial Disclosure: The author declared that this study received no financial support.

REFERENCES

1. Gezegen H, İlgezdi Kaya İ, Kalaycı T, et al. Seeing clowns with a ring 20 chromosome. *Arch Epilepsy.* 2024;30(1):31-35. **[Crossref]**
2. Grill MF, Maganti R. Cephalosporin-induced neurotoxicity: clinical manifestations, potential pathogenic mechanisms, and the role of electroencephalographic monitoring. *Ann Pharmacother.* 2008;42(12):1843-1850. **[Crossref]**
3. Bora I, Demir AB, Uzun P. Nonconvulsive status epilepticus cases arising in connection with cephalosporins. *Epilepsy Behav Case Rep.* 2016;6:23-27. **[Crossref]**
4. Schliamser SE, Cars O, Norrby SR. Neurotoxicity of beta-lactam antibiotics: predisposing factors and pathogenesis. *J Antimicrob Chemother.* 1991;27(4):405-425. **[Crossref]**
5. Caraballo RH, Cejas N, Chamorro N, Kaltenmeier MC, Fortini S, Soprano AM. Landau-Kleffner syndrome: a study of 29 patients. *Seizure.* 2014;23(2):98-104. **[Crossref]**