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New-onset refractory status epilepticus after first dose of tozinameran

[1]You-Jiang Tan, [2]Chun Yang Sim, [1]Kaavya Narasimhalu, [3]Kheng Seang Lim

[1]Department of Neurology, National Neuroscience Institute, Singapore; [2]Faculty of Medicine and Health Sciences, University Malaysia Sarawak, Sarawak, Malaysia; [3]Division of Neurology, Department of Medicine, Faculty of Medicine, University of Malaya, Kuala Lumpur, Malaysia

Abstract

As the worldwide vaccination effort against COVID-19 gains traction, complications attributed to the vaccines are increasingly reported in medical literature. Herein, we describe the first two cases of new-onset refractory status epilepticus after receiving their first doses of tozinameran. These were attributed to underlying autoimmune responses against the vaccine given the temporal proximity to them receiving tozinameran, the absence of alternative diagnoses, the refractoriness of their seizures, and their clinical improvement only after administration of immunotherapies. Importantly, their cases supplement existing reports on the rare neurologic complications of the COVID-19 vaccines and encourage continued vigilance amongst the physicians amidst the pandemic.

Keywords: NORSE; COVID-19; vaccination; status epilepticus; encephalitis

INTRODUCTION

COVID-19 was identified in 2019 as a novel cause of severe acute respiratory distress syndrome and has been associated with neurologic complications involving both the central and peripheral nervous systems.[1] Declared a pandemic by the World Health Organization in March 2020, close collaboration between the pharmaceutical industry and government bodies saw the introduction of multiple vaccine platforms, ranging from mRNA-based to viral vectors.[1,2] As the global vaccination efforts intensify, reports on neurologic complications after vaccinations are increasingly reported in literature, although a causal relationship remains unclear.[2] Herein, we describe the two rare cases of new-onset refractory status epilepticus (NORSE) beginning five and eight days after receiving tozinameran (Pfizer-BioNTech) respectively.

CASE REPORT

Patient 1

A 28-year-old female presented with generalized tonic-clonic seizures (GTCS) after receiving tozinameran. She was without pre-existing medical disorders or drug allergies. She had just received her first dose of tozinameran eight days ago. She was afebrile, but was photophobic and lethargic, scoring 10 on the Glasgow Coma Scale (GCS; E3V2M5). Kernig sign was absent. Suspecting acute meningoencephalitis, intravenous (IV) antimicrobials were started together with IV levetiracetam for her seizures. Blood inflammatory markers, microbiologic cultures and assays, toxicology, and tests for metabolic, thyroidal, and electrolyte derangements, all returned normal. Lumbar puncture (LP) revealed a normal opening pressure (10cmH_2O). Cerebrospinal fluid (CSF) analysis however demonstrated elevated white cell counts (WCC; 8/uL) and protein levels (0.43g/L). CSF microbiologic assays and cultures were negative. SARS-CoV2 polymerase chain reaction (PCR) tests of nasopharyngeal swabs performed two days apart were negative. Brain magnetic resonance imaging (MRI) revealed bilaterally swollen medial temporal lobes which were hyperintense on T2 and fluid-attenuated (FLAIR) sequences (Figure 1A). Post-contrast T1 sequences demonstrated no abnormal enhancement.

Despite initial treatment measures, she developed myoclonic status epilepticus and required induction of barbiturate coma at the intensive care unit (ICU). Electroencephalography (EEG) demonstrated near-continuous seizures over both hemispheres (Figure 2, with description

Address correspondence to: Dr. Tan You-Jiang MRCP, National Neuroscience Institute, Department of Neurology, Outram Road, 169608, Singapore. Tel: +65 6326 5656, +65 93872969, Email: tan.you.jiang@singhealth.com.sg

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