Dear Editor, 

Glutamate is a major excitatory neurotransmitter in the central nervous system. Overstimulation of postsynaptic receptors causes excitotoxicity, which underlies neuronal loss resulting in injury in many seemingly unrelated disorders, including ischemia, trauma, hypoglycemia, hypoxia, status epilepticus, Wernicke syndrome, and even neurodegenerative disorders such as Alzheimer’s disease, Parkinson’s disease, and Huntington’s disease.1,2

In status epilepticus, neuronal seizure activity increases the release of glutamate at presynaptic terminals. Encephalopathy with status epilepticus often involves the hippocampus, other parts of the limbic system, the thalamus, and the cerebellum.3

We describe a case of a 53-year-old woman presenting with acute diffuse headache followed by two generalized tonic-clonic seizures admitted to our emergency department.

She had no previous illnesses, but she was a heavy smoker (40 cigarettes per day), referred regular ingestion of high doses of ethanol for the last 10 years, and had been using crack for the last two years, once or twice a day, two days a week.

Her examination in the emergency room revealed impaired level of consciousness, Glasgow Coma Scale of 10 out of 15, no focal neurological signs, and moderate psychomotor agitation.

Her laboratory results were within normal limits. There was no evidence of infectious or metabolic abnormalities. A spinal tap was unremarkable.

The MRI demonstrated hyperintensities in T2 and FLAIR in temporal, occipital, and parietal regions, thalamus, brainstem, and cerebellum, with outstanding symmetry.

An EEG was performed and confirmed status epilepticus. The patient was admitted to an intensive care unit (ICU), receiving intravenous midazolam, diazepam, and phenytoin. A new EEG examination had normal results.

Figure 1. MR images revealing symmetrical hypersignals in FLAIR sequence during status epilepticus (A, B and C) and after 30 days of drug treatment and recovery (D, E and F).
References: