## Nonconvulsive Status Epilepticus in Schizophrenia: Focus on Early Detection

*To the Editor:* Nonconvulsive status epilepticus (NCSE) as a condition with a change in consciousness associated with continuous epileptiform discharges in the EEG is easily treated but often underdiagnosed.[1] This is partially explained by its absence of major motor signs and protean manifestations seen in nonepileptic disorders. We present a case of NCSE and highlight early detection of this condition in patients with schizophrenia.

**Case Report**

A 64-year-old woman with schizophrenia, which was well managed by risperidone 4 mg/day, fludiazepam 0.75 mg/day, and flunitrazepam 2 mg/day in recent years, was brought to the emergency department because of an abrupt onset of an altered level of consciousness, irritability, and psychosis. Laboratory results showed hyponatremia (115 mmol/liter) and hypokalemia (2.6 mmol/liter). Otherwise, abdominal ultrasonography, brain CT, CSF analysis, and other examinations revealed no remarkable findings. The family reported that for the past few days, there had been a marked excess of water intake. With a diagnosis of polydipsia-induced hyponatremia, she was admitted for electrolyte replacement.

Upon admission, the treating physicians instantly discontinued all psychotropics. Because her condition did not improve even as her serum sodium and potassium concentrations were restored, psychiatric consultation was requested on Hospital Day 4. A diagnosis of delirium was made, and risperidone 4 mg/day was resumed. Her condition, however, gradually worsened. On Hospital Day 7, an EEG was obtained, which revealed generalized epileptiform discharges. The brain MRI showed negative findings. Consultation with a neurologist resulted in a diagnosis of NCSE; a regimen of levetiracetam 1,500 mg/day and intravenous valproic acid 1,200 mg/day was started, and risperidone was withheld. Four days after the initiation of antiepileptic agents, the patient's condition was notably improved. An EEG performed subsequently did not show any further epileptiform discharges.

**Discussion**

This case illustrates the complexities involved in distinguishing NCSE from other causes contributing to the development of delirium. Considering NCSE as a cause of delirium without an EEG is a great challenge because of the significant overlap in clinical features between NCSE and delirium. Misdiagnosis of NCSE could place psychiatrists at risk of iatrogenic error, because delayed treatment or exposure to antipsychotic agents that could lower seizure threshold might aggravate NCSE. Benzodiazepine (BZD) withdrawal, antipsychotic treatment, and delay in diagnosis might have worked in combination to exacerbate this patient's NCSE. Evidence demonstrates that patients with schizophrenia are more likely to experience seizures because of comorbid substance abuse, long-term exposure to psychotropic agents, and frequent episodes of head injury.[2] Also, the behavioral and mental status changes of NCSE may incorrectly be ascribed to psychotic symptoms. A variety of precipitating factors have been implicated in NCSE, including polydipsia-induced hyponatremia,[3] a previous history of seizures,[1] infection,[1] ECT,[4] and BZD withdrawal,[5] all of which are commonly associated with either the treatment of or are common comorbidities with schizophrenia.

In conclusion, when evaluating patients with schizophrenia who have developed altered mental status and behavior, psychiatrists should pay more attention to whether an emergent EEG is needed in order to confirm or exclude the diagnosis of NCSE.

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