

Non-Convulsive Status Epilepticus Following Hemodynamically Unstable Atrial Fibrillation

S Blitshteyn, D Chabolla, F Rubino

Citation

S Blitshteyn, D Chabolla, F Rubino. *Non-Convulsive Status Epilepticus Following Hemodynamically Unstable Atrial Fibrillation*. The Internet Journal of Neurology. 2004 Volume 4 Number 2.

Abstract

Non-convulsive status epilepticus (NCSE) is an increasingly recognized condition in non-epileptic intensive care patients and is seen in up to 8% of patients in coma. NCSE can be a challenging diagnosis because clinical features include altered mental status in the absence of convulsive activity.

In critical care patients, NCSE may be precipitated by a variety of conditions, such as infection, severe electrolyte disturbance, acute intoxication, sedative-hypnotics withdrawal and traumatic brain injury. However, NCSE following hemodynamically unstable atrial fibrillation has not been reported. This article describes a patient who developed NCSE after an episode of hemodynamically unstable atrial fibrillation resulting in systemic hypotension and cerebral hypoperfusion. Fosphenytoin failed to produce clinical improvement despite resolution of abnormal pattern on EEG, and complete recovery was achieved only after administration of lorazepam. In the intensive care patients, NCSE may occur following hemodynamic compromise, and cerebral hypoperfusion may play a role in precipitating NCSE. A high index of clinical suspicion is necessary to diagnose and treat NCSE, which may be rapidly reversed by the administration of benzodiazepines.

INTRODUCTION

Non-convulsive status epilepticus (NCSE) is an increasingly recognized condition in non-epileptic intensive care patients, and is seen in up to 8% of patients in coma who have no signs of seizure activity [1]. The etiology of de novo NCSE in the intensive care setting may include infection, severe electrolyte disturbance, acute intoxication, sedative-hypnotics withdrawal and traumatic brain injury [2,3]. Diagnosis and identification of precipitating factors can be challenging since NCSE is often mimicked by other conditions [4]. We report a patient who developed NCSE following an episode of hemodynamically unstable atrial fibrillation, which resulted in systemic hypotension and cerebral hypoperfusion. To our knowledge, atrial fibrillation or cerebral hypoperfusion as possible NCSE precipitating factors have not been described previously.

CASE REPORT

A 65-year-old woman presented to the emergency department with severe abdominal pain and was hospitalized for suspected diverticulitis. On hospital day 3, she became unresponsive, hypotensive, with a measured systolic blood pressure at less than 80 mm Hg, and was found to have atrial

fibrillation with rapid ventricular response. Direct current cardioversion was successfully performed, as a result of which she became awake and alert, and was transferred to the intensive care unit for further monitoring. On hospital day 4, she remained hemodynamically and neurologically stable and at her baseline mental status. On hospital day 5, she again became unresponsive despite stable vital signs and sinus rhythm. She did not respond to verbal stimuli and did not follow simple commands. There were no visible signs of convulsive activity, and her Glasgow coma scale was estimated at 9. MRI of the brain revealed no evidence of acute infarct, hemorrhage or intraparenchymal lesions. CSF analysis did not show evidence of meningitis. Laboratory tests revealed mildly elevated liver function tests and serum creatinine level, which were felt to be secondary to tissue hypoperfusion caused by hemodynamically unstable atrial fibrillation. EEG recording on hospital day 5 demonstrated intermittent spikes and polymorphic rhythmic slowing over all head regions with no response to auditory or photic stimulation. This pattern was felt to be compatible with a diagnosis of NCSE by an epileptologist. The patient was administered 15 mg/kg of intravenous fosphenytoin, which failed to produce any clinical improvement. On hospital day

7, the patient remained unresponsive, and a second EEG was obtained. It revealed a significant change from previous recording, with normal appearing sleep patterns and a responsiveness of cerebral activity during auditory stimulation. During the second EEG, 2 mg of intravenous lorazepam was administered as part of the NCSE protocol, and no direct EEG changes were observed following administration of lorazepam. However, several hours later, the patient was found to be fully awake, alert and oriented. She was discharged several days later and has remained seizure-free and without neurologic deficits at a 4-week follow-up.

DISCUSSION

Non-convulsive status epilepticus occurs in up to 8% of comatose patients [1] and may present a diagnostic challenge when precipitating factors are not easily identified. In the intensive care setting, other commonly encountered conditions can mimic NCSE and may include substance intoxication, psychiatric conditions, metabolic encephalopathy, traumatic brain injury and stroke [3,4]. In the case presented here, the differential diagnostic considerations for the unresponsive state included metabolic encephalopathy due to hepato-renal dysfunction, but this diagnosis was refuted by the EEG response to fosphenytoin and her rapid clinical improvement following administration of lorazepam. Fountain and Waldman found that unresponsive patients with metabolic encephalopathy did not arouse and some

actually became less responsive after benzodiazepine administration, despite resolution of rhythmic sharp waves on EEG [5]. Other etiologies of acute mental status change in this patient included cardioembolic stroke, in the setting of new-onset atrial fibrillation, infection and severe electrolyte disturbance. These were ruled out by MRI of the brain, CSF analysis, and other diagnostic tests showing no evidence of systemic infection or significant electrolyte abnormalities. Thus, NCSE was the most likely etiology of the unresponsive state in our patient, and this diagnosis was supported by a significant change in EEG pattern after administration of fosphenytoin and a rapid clinical response to lorazepam.

We propose, based on the patient's evidence of hepato-renal dysfunction from systemic hypoperfusion, that cerebral hypoperfusion caused by hemodynamically unstable atrial fibrillation may have played a role in NCSE. Vespa et al. reported convulsive and non-convulsive seizures in 22% of

patients with moderate-to-severe traumatic brain injury. In more than half of the patients, the seizures were non-convulsive and occurred during the first week after the injury [3]. Although in their study, brain injury resulted from traumatic subdural hematoma, subarachnoid hemorrhage and cerebral contusion, the combined effect of hypotension and hypoxemia during the initial resuscitation was considered as causally related to status epilepticus. In our patient, systemic hypotension in the setting of hemodynamically unstable atrial fibrillation occurred one day prior to the onset of unresponsive state. We hypothesize that systemic hypotension may have resulted in an increased susceptibility to NCSE via cerebral hypoperfusion causing transient neuronal injury and increased cortical excitability.

Therefore, in the intensive care patients, NCSE may occur following hemodynamic compromise, and cerebral hypoperfusion may play a role in precipitating NCSE. Further investigation is necessary to delineate if there is a causative relationship between cerebral hypoperfusion caused by non-traumatic, commonly encountered conditions in the intensive care unit and NCSE.

This case also underscores the importance of prompt recognition of NCSE, identification of its precipitating factors and institution of the most effective therapy. The patient was initially treated with fosphenytoin, which failed to produce clinical improvement despite resolution of abnormal wave pattern on EEG. Recovery from the unresponsive state three days after its onset was achieved following administration of lorazepam, which has been consistently reported as the first line therapeutic agent in NCSE [6,7].

Furthermore, despite earlier reports of potentially favorable outcomes [7,8], NCSE has been found to cause substantial morbidity and mortality in a study by Shneker and Fountain [9]. NCSE was associated with an 18% mortality rate in a series of 100 consecutive patients and a 39% mortality rate in a sub-group of patients with severe mental state impairment [9]. Therefore, clinicians should be vigilant to diagnose and treat NCSE, a potentially life-threatening condition.

CONCLUSION

This is the first case report describing a patient who developed NCSE following an episode of hemodynamically unstable atrial fibrillation, which resulted in systemic hypotension and cerebral hypoperfusion. Since clinical features of NCSE include altered mental status without signs

of convulsive activity, identification of NCSE in a comatose patient may present a diagnostic challenge. Clinicians should have a high index of clinical suspicion to diagnose and treat NCSE, which may be rapidly reversed by the administration of benzodiazepines.

CORRESPONDENCE TO

Svetlana Blitshteyn, MD Department of Neurology Mayo Clinic Jacksonville 4500 San Pablo Road Jacksonville, Florida 32224 Phone: 904-953-2623 Fax: 904-953-0757 E-mail: Blitshteyn.Svetlana@mayo.edu

References

1. Towne AR, Waterhouse EJ, Boggs JG, et al. Prevalence of nonconvulsive status epilepticus in comatose patients. *Neurology*. 2000; 54: 340-345.
2. Dunne JW, Summers QA, Steward-Wynne EG. Non-convulsive status epilepticus: a prospective study in an adult general hospital. *Q J Med* 1987; 62:117-126.
3. Vespa PM, Nuwer MR, Nenov V, et al. Increased incidence and impact of nonconvulsive and convulsive seizures after traumatic brain injury as detected by continuous electroencephalographic monitoring. *J Neurosurg* 1999; 91: 750-760.
4. Kaplan PW. Assessing the outcomes in patients with nonconvulsive status epilepticus: nonconvulsive status epilepticus is underdiagnosed, potentially overtreated, and confounded by comorbidity. *J Clin Neurophysiol* 1999; 16: 341-352.
5. Fountain NB, Waldman WA. Effects of benzodiazepines on triphasic waves: implications for nonconvulsive status epilepticus. *J Clin Neurophysiol* 2001; 18:345-352.
6. Waterhouse EJ, DeLorenzo RJ. Status epilepticus in older patients: epidemiology and treatment options. *Drugs & Aging* 2001; 8:133-142.
7. Scholtes, FB, Renier WO, Meinardi, H. Non-convulsive status epilepticus: causes, treatment, and outcome in 65 patients. *J Neurol Neurosurg Psychiatry*. 1996; 61: 93-95.
8. Kaplan PW. No, some types of nonconvulsive status epilepticus cause little permanent neurologic sequelae. *Neurophysiol Clin*. 2000; 30:377-82.
9. Shneker BF, Fountain NB. Assessment of acute morbidity and mortality in status epilepticus. *Neurology*. 2003; 61:1066-73.

Author Information

Svetlana Blitshteyn, M.D.

Department of Neurology, Mayo Clinic Jacksonville, Mayo College of Medicine

David R. Chabolla, M.D.

Department of Neurology, Mayo Clinic Jacksonville, Mayo College of Medicine

Frank A. Rubino, M.D.

Department of Neurology, Mayo Clinic Jacksonville, Mayo College of Medicine