Case Report

Indian Journal of Emergency Medicine
Volume 5 Number 3, July - September 2019
DOI: http://dx.doi.org/10.21088/ijem.2395.311X.5319.9

Misleading Features of an Unwitnessed First Episode of Status Epilepticus; Causing Confusion with Assault and Head Injury

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Received on 15.06.2019,
Accepted on 11.07.2019

Abstract

The diagnosis of first episode of status epilepticus associated with head injury can be quite difficult unless it is witnessed. Such status epilepticus can lead to serious complications due to its prolonged seizure activity leading to hypoxia, hypotension, metabolic acidosis, cardiac dysrhythmias, neurogenic pulmonary edema, hyperthermia, rhabdomyolysis, and pulmonary aspiration and permanent brain damage and death. We hereby describe a case of a 41-year-old male who was found unresponsive in his balcony with multiple facial injuries initially mimicing assault with head injury but eventually confirmed to be a status epilepticus. He was well-managed by the emergency team and he was discharged in a stable condition with no focal neurological deficit.

Keywords: Head injury; Status epilepticus; Suspected assault; Seizure.

How to cite this article:
Dhileeban CM, Sarat Kumar Naidu, Dheeraj Bhaskaran Nair. Misleading Features of an Unwitnessed First Episode of Status Epilepticus; Causing Confusion with Assault and Head Injury. Indian J Emerg Med. 2019;5(3):205-208.

Introduction

Status epilepticus (SE) is a common neurological emergency associated with high morbidity and mortality. It is definedas single seizure persisting for >30 min or multiple seizures without neurological recovery in between, impending status epilepticus is defined as continuous or intermittent seizures lasting >5 min. Therefore, any seizure activity lasting for more than 5 minutes is significant.[1,2]

Mortality from SE varies from 3 to 50% in different studies. In old age, refractory status epilepticus (RSE) may lead to mortality in over 76% cases. The lifetime prevalence of SE in persons with epilepsy range from 1 to 16%. Precise epidemiological data for SE are not available for India.[2,3] It is essential that all physicians are able to identify and treat SE promptly and efficiently. Because it may sometimes mislead into trauma as in our case, wherein mortality and morbidity may increase.

Case Study

An ambulance call was received by our ER for a middle-aged male who was found lying unresponsive in his balcony with bleeding from his head and face. EMS team with doctor was immediately rushed to that place and the police was informed as well. On scene, this 40-something gentleman was found unresponsive with E1V1M4 and with multiple lacerations over forehead and blackish discoloration of right eyelid. In the balcony there was a cot and a concrete beam which was blood stained. His vitals were, pulse 100 bpm, RR 7 (Labored breathing), BP 70/50 mmHg, SpO_2 80% at RA, Random blood sugar was 140 mg/dl. Immediately he was cannulated and 1L normal saline was pushed, and simultaneously he was intubated due to low GCS and unstable hemodynamics. The oropharynx was full of blood. After initial stabilization, he was shifted to our

emergency department which was 10 minutes of drive. On taking further history, the patient was apparently separated from his wife and was living alone in his house. Initialy the whole scenario mimicked as assault and head injury.

In the ER, he was taken for urgent CT scan which showed small hemorrhagic contusion involving the right temporal lobe and fracture of roof of right orbit. His hemodynamics started improving with the initial resuscitation and there was no evidence of any other injuries.

After sometime his neighbours, friends and relatives arrived to the hospital. His friend stated that he met him that day morning when he was apparently normal. He also mentioned that he was a known case of ulcerative colitis and chronic multiple joint pains and there was no other significant past medical history. His blood investigations showed no significant findings except raised lactates. Other radiological imaging did not show any relevant findings either. Without any other clue, he was being managed as a case of assault and head injury. He was admitted under neurosurgeon in the ICU.

The next day the patient improved neurologically and was extubated successfully. MRI brain and venography were done which revealed a focal area of T2 hyperintensity with surrounding T2 hypointense rim and showing blooming on GRE sequence was seen involving the right middle temporal lobe. The surrounding brain parenchyma was otherwise normal. In view of the possible history of trauma for this patient, this representation of focal hemorrhage needed to be considered, though a close differential diagnosis was a cavernoma. Yet another punctuate T2 hypointensity, most likely non-specific dystrophic mineralization is also was seen involving the left high frontal lobe.

When he was conscious and oriented, on further history-taking from him, he revealed that he was alright that morning and after his breakfast he went out to his balcony when he suddenly became dizzy and had facial twitches and did not remember thereafter. He also revealed that he had similar episode once in the past when he was provisionally diagnosed as seizure but did not take any medications then.

It was only after taking detailed history from the patient himself, that the scenario of an episode of status epilepticus became more clear. It was possible that he had SE due to which there was airway compromise leading to hypoxia. During the seizure episode he probably fell from the cot accidently and hit on the concrete beam which was probably the reason for his head and facial injuries. He also had a minor tongue-bite which was the reason for bloody secretion in the throat during intubation.

The patient was managed conservatively with anticonvulsants, antibiotics and other supportive measures. Neurology, surgical and orthopedics opinions were sought and he was shifted to ward for further management. He was finally discharged in a stable condition after 4 days of hospital stay. On his follow-up visits, he was doing well.

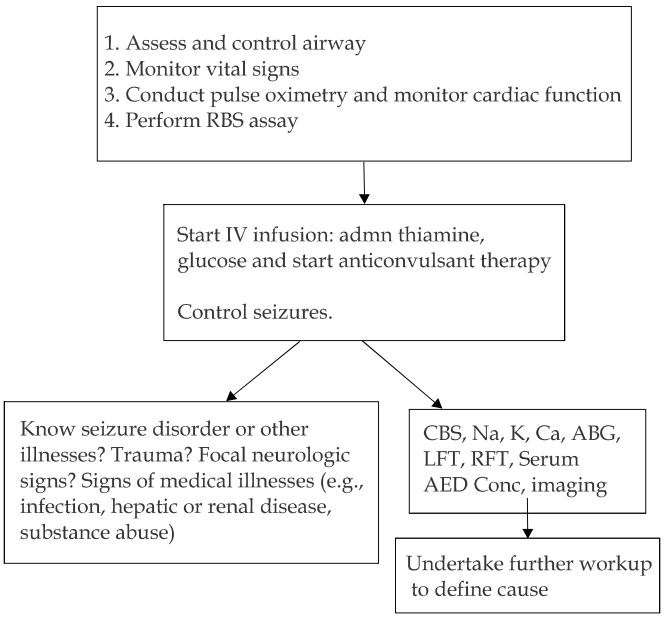

Fig 1: Approach to status epilepticus

Even in the police investigations, nothing suspicious of assault was ascertained which further confirmed that this was not a case of assault.

Discussion

Status epilepticus is a life-threatening neurological emergency associated with significant morbidity and mortality. It has been estimated that up to 150,000 cases of SE occur annually in the US, with 55,000 associated deaths.[2,3] Sex, age, geography, and race influence the epidemiology of SE. An incidence of 6.2–18.3 per 100,000 population has been reported in the US.[2,4]

Gaba aminobutyric acid (GABA) is the major inhibitory neurotransmitter in the CNS. It is released from GABA minergic neurons and binds to several types of GABA receptors [i.e., GABA-A (GABA type A), GABA-B, and GABA-C receptors]. GABA receptors are macromolecular proteins that form a chloride ion channel complex and contain specific binding sites for GABA and a number of allosteric regulators, including barbiturates, benzodiazepines, and a number of anesthetic agents. GABA receptor–mediated inhibition may be responsible for the normal termination of a seizure. In addition, the activation of the NMDA receptor by the excitatory neurotransmitter glutamate may be required for the propagation of seizure activity. The activation of NMDA receptors results in increased levels of intracellular calcium, which may responsible for the nerve cell injury seen in patients with SE.[2,5]

As seizure crosses the 5-minutes mark, drastic change occur in the cellular level decreased expression and internalization of GABA additionally with increased expression of both glutamine NMDA receptor. These changes greatly decreased seizure threshold. The blood brain barrier is also compromised, cause CNS penetration of potassium and albumin both are excitatory neurotransmitter.

After 20 minutes hypoxia, hypotension, metabolic acidosis, cardiac dysrhythmias, rhabdomyolysis and pulmonary edema can develop.[6,7]

After 2 hours of seizure activity neurotoxic aminiacid and calcium are released into cells leading permanent neuronal damage.[5,8]

The longer duration of SE, the more refractory to treatment and greater chances of complications. More prolonged seizures carry higher risk of mortality.[8,9]

The following Figures 1 and 2 demonstrate the guidelines for the management of SE.

CBC-complete blood count, Na–sodium, k–pottasium, Ca–calcium
ABG-Arterial blood gas, AED–anti-epileptic drug
Guidelines for management of active seizure and status epilepticus

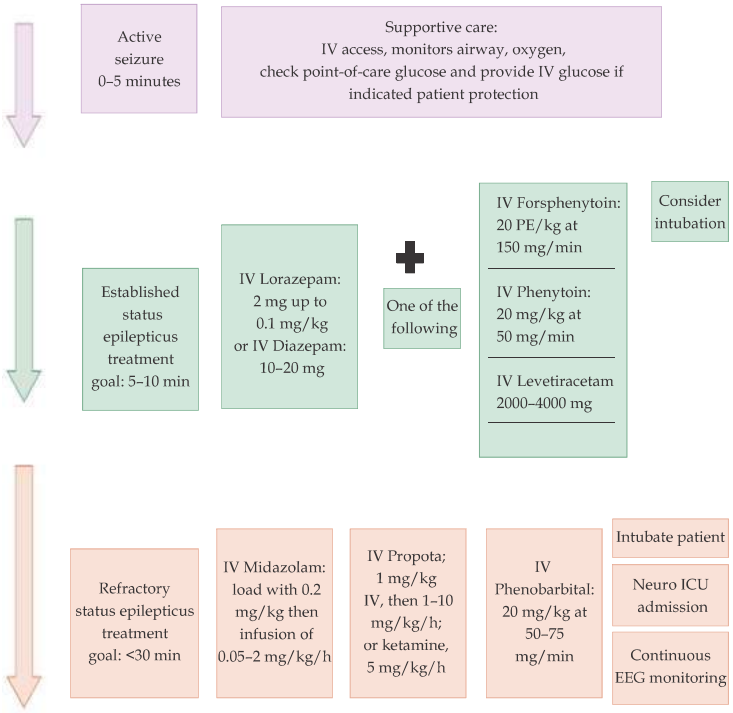

Fig 2: Guidelines for management of active seizure and status epilepticus ICU–intensive care unit, PE–phenytoin equivalents.[10]

Conclusion

Status epilepticus is a neurological emergency associated with health-care costs, morbidity, and mortality.[2] For a known case of seizure disorder, it becomes easier to diagnose a new episode. However, it can lead to misdiagnosis if it is a first episode and that too if it is unwitnessed as seen in our case described above. Our patient presented with unresponsive state with multiple trivial facial injury and head injury and was being initially managed as head injury secondary to assault, for 2 days until he became conscious to reveal the clear history; CT head also suggested small hemorrhagic contusion and fracture of right orbit roof, all favoring head injury.

The primary aim for publishing this article is to make the emergency physicians aware that all cases mimicking head injury and assault need not to be assault; it can have an alternate diagnosis like status epilepticus. As emergency physicians, we should keep our mind open to a wide range of possible diagnoses, obviously attempting to manage the most probable diagnosis first, as we did in our case.

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