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THE MANAGEMENT OF A PATIENT WITH TRAUMATIC BRAIN INJURY AND ACUTE NEUROLOGICAL CONDITION PROGRESSING TO STATUS EPILEPTICUS: EXPERIENCE REPORT

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ABSTRACT
This report describes the case of a patient with a history of chronic diseases, who suffered a traumatic brain injury and presented a complex clinical picture, with possible diagnosis of subarachnoid hemorrhage (SAH), viral encephalitis, brain metastasis, and paraneoplastic encephalitis. The diagnostic approach involved complementary tests and the progression of the condition, focusing on neurological management and complications associated with the patient's condition. After investigation, a diagnosis of status epilepticus was confirmed; however, the patient passed away after 18 days of hospitalization. It was possible to identify the importance of early diagnosis and timely treatment to prevent complications of the condition, such as death.

Keywords: Status epilepticus, Complications of status epilepticus, Death.

INTRODUCTION

Acute neurological conditions encompass a range of severe disorders that require immediate recognition and rapid intervention, as the patient's life is often at risk. Among these emergencies, status epilepticus (SE) stands out as a potentially fatal condition characterized by prolonged seizures that demand intensive management. SE is defined as a seizure lasting more than 30 minutes or multiple consecutive seizures without full recovery between them, representing one of the most critical manifestations of epilepsy.[1]

The pathophysiology of status epilepticus involves electrical dysregulation in the brain, resulting in excessive and persistent neuronal activity. This phenomenon can be triggered by various causes, such as traumatic injuries, central nervous system infections, metabolic disorders, or even as a complication of uncontrolled epileptic seizures. The lack of appropriate treatment can lead to a cascade of harmful effects, including neuronal death and irreversible brain tissue damage, thereby increasing the morbidity and mortality associated with this condition.[2]

Clinically, status epilepticus manifests as continuous seizures that can affect both motor and cognitive functions of the patient. In addition to motor seizures, patients may present with altered levels of consciousness, respiratory difficulties, and cardiovascular instability. Early identification and differentiation between status epilepticus and other acute neurological conditions are crucial for the implementation of effective treatment. In this context, rapid diagnosis, often based on clinical history and physical examination, is essential, as delays in therapeutic intervention can result in permanent damage to the central nervous system.[3]

The treatment of status epilepticus is one of the greatest challenges in neurological clinical practice. Proper management requires the use of high-dose anticonvulsants and, in some cases, adjunct therapies such as general anesthesia, especially when SE is refractory to medications. Controlling seizures is essential to prevent sequelae such as cognitive, motor, and behavioral deficits, as well as systemic complications like respiratory failure and shock. Treatment must be tailored to the patient's profile and the underlying causes of status epilepticus.[4]

This report aims to discuss the clinical and therapeutic aspects of status epilepticus in a patient in an intensive care unit, with an emphasis on early identification, emergency management, and specific interventions. It will address the main causes leading to the development of SE and the diagnostic challenges faced by healthcare professionals. A thorough understanding of status epilepticus is essential to improve prognosis and reduce the complications associated with this severe neurological emergency.

EXPERIENCE REPORT

A 67-year-old male patient with a history of chronic kidney disease (CKD), systemic arterial hypertension (SAH), and prostate cancer undergoing chemotherapy (goserelin acetate 10.8 mg every three months) presented to the hospital. His regular medications included amlodipine, allopurinol, bicarbonate, epoetin alfa, iron oxide saccharate, and acetylsalicylic acid. The patient was admitted following a fall from standing height 7 days prior, presenting with a facial hematoma, progressive alteration in consciousness, and difficulty ambulating. He reported no recollection of the fall and denied headache, chest pain, or dyspnea. He sought hospital care due to reported paresthesia on the right side of his face, which had led to the interruption of hemodialysis, prompting referral to the hospital.

At the time of evaluation, the patient was alert but confused, with a presentation possibly secondary to trauma or neurological complications. On physical examination, the patient appeared to be in fair general condition, somnolent, non-icteric, acyanotic, and afebrile. The remaining findings are detailed below:

- Blood Pressure: 177/97 mmHg
- Heart Rate: 89 bpm
- Respiratory Rate: 17 breaths per minute
- Glasgow Coma Scale: 14
- Oxygen Saturation on O2: 95%
- Neurological: Glasgow 14, no motor deficits, no signs of meningeal irritation
- Cardiovascular: regular heart rhythm, no murmurs
- Respiratory: bilateral vesicular breath sounds present, no adventitious sounds

- Abdomen: flat, no signs of peritoneal irritation
- Extremities: no edema, calves free of tenderness

The initial management for the condition was the transfer to the Intensive Care Unit (ICU). The patient was admitted to the ICU in fair general condition, with a Glasgow score of 14 and acute cognitive alteration, being monitored and maintained in an intensive support environment. The initial decision was for continuous observation with investigation into possible causes of his neurological manifestations. Investigations were conducted with the aid of imaging and laboratory tests, as outlined in the tables.

The patient maintained mental confusion and developed agitation within the first 24 hours of hospitalization but remained eupneic with 2 liters of oxygen via nasal cannula and a good respiratory pattern. In the ICU, in addition to vasoactive medications for vital sign control, acyclovir was initiated due to the diagnostic hypothesis of viral encephalitis. After 48 hours of hospitalization, the patient progressed to myoclonus and recurrent seizures, requiring orotracheal intubation. A neurology consultation was requested.

The patient exhibited coughing during tube aspiration and biting, but did not present new episodes of myoclonus. After 4 days of hospitalization, attempts to wake the patient by reducing sedation were made, but without effective results. According to the neurology consultation, valproic acid 250 mg/5 ml was initiated, prescribed at 5 ml every 12 hours, and clobazam 10 mg every 12 hours. On neurological physical examination: global hyporeflexia, absence of clonus, Hoffman sign absent, plantar cutaneous reflex indifferent. The patient also presented normal muscle tone, no rigidity, miotic pupils, isocoric, with poor photoreactivity, corneal-palpebral reflex present, and absence of the oculocephalic reflex (doll's eyes maneuver). There was no nystagmus, no ocular deviation, absence of pupillary hippus, and no involuntary movements were observed.

Laboratory tests and serologies were requested as per Table 3, considering the hypotheses of seizures to be clarified, paraneoplastic encephalitis, carcinomatous meningitis, and infectious encephalitis. Given the negative VDRL result in both blood and cerebrospinal fluid (CSF), the possibility of neurosyphilis (which could present with seizures and elevated protein levels in CSF) was ruled out. Therefore, following the negative neurosyphilis test, the patient continued on acyclovir for the differential diagnosis of herpes encephalitis. Elevated protein levels in CSF can also occur in isolation in cases of seizures of any etiology, even if caused by a metabolic disturbance, for example. After several attempts at extubation, the patient continued to exhibit a decreased level of consciousness (LOC) even after sedation was discontinued, leading to the consideration of toxic-metabolic encephalopathy or non-convulsive status epilepticus as potential diagnoses.

Upon re-evaluation after 9 days of hospitalization, the patient presented with new episodes of seizures accompanied by myoclonus. Laboratory tests revealed a reactive FTA-Abs IgG, and a consultation with infectious disease specialists was requested. They initiated crystalline penicillin for the treatment of neurosyphilis. During the physical examination, the patient experienced two generalized myoclonic seizures, each lasting 5 seconds.

Table 1 - Tests performed upon admission on July 22, 2024

Test	Result	References
Hemoglobin	11,1	12,8 - 17,8 g/dL
Hematocrit	33,0	40 – 50 %
Leukocytes	6.630	3600 - 11000/mm3
Platelets	132.000	150.000 - 400.000/mm3
Lactate	1,86	0,5 - 2,2 mmol/L
pH (venous)	7,42	7.350 – 7.450
PCO2 (venous)	42,5	35 – 45 mmHg
HCO3 (venous)	26,2	21 – 27 mEq/L
Saturation (venous)	98%	95 – 100%
Total Bilirubin	0,50	0,3 – 1,2 mg/dL
Direct Bilirubin	0,39	0,1 a 0,4 mg/dL
Indirect Bilirubin	0,11	< 1,2 mg/dL
Sodium	143	135 – 145 mmol/L
C-Reactive Protein	0,5	Menor que 6 mg/L
Calcium	1,16	1,17 - 1,30 mmol/L
Potassium	4,1	3,5 – 5,5 mmol/L
Magnesium	2,0	1,7 - 2,4 mg/dL
Prothrombin Time (PT)	11,1 seconds	12,7 to 15,2 seconds
Activated Partial Thromboplastin Time (aPTT)	22,7 seconds	24 the 40 seconds
INR	1,01	0,8 – 1
Creatine Phosphokinase (CPK)	22	29 – 168 U/L
Creatine Phosphokinase MB (CKMB)	9	< 5 ng/mL
Troponin	Negative	Negative
Creatinine	5,26	0,80 a 1,30 mg/dL
Urea	69	15 a 45 mg/dL

Source: Elaborated by the author.

Table 2 - Cerebrospinal Fluid (CSF) Test on 07/23/2024

Test	Result	References
Glucose	64	40–80 mg/dL
Protein	88	15–60 mg/dL
VDRL	Negative	Negative
Cell Count	4	0-5 lymphocytes/mcL

Source: Elaborated by the author.

Table 3 - Tests performed in July 2024

Test	Result	References
HbsAg	Non-Reactive	Non-Reactive
Anti-HBs	Non-Reactive	Non-Reactive
Anti-HBc IgM and IgG	Non-Reactive	Non-Reactive
Anti-HCV	Non-Reactive	Non-Reactive
VDRL	Non-Reactive	Non-Reactive
FTA Abs IgG	Reactive	Non-Reactive
CSF Viral Panel	Negative	Negative
CSF Cysticercosis	Negative	Negative

Source: Elaborated by the author.

CT Scan of the Brain 22/07/2024

Reduction in the volume of the encephalic parenchyma, with prominence of the cerebral sulci and fissures, as well as the cerebellar folia, leading to compensatory ectasia of the supratentorial ventricular system. Sparse hypodense foci in the supratentorial white matter, without significant atrophic or expansive effects, nonspecific, but possibly corresponding to gliosis/microangiopathy. Basal ganglia and capsular regions appeared preserved. Midline structures are centered. The fourth ventricle was in its usual location, with normal dimensions. No evident lesions in the posterior fossa by this method. Incipient atheromatous plaques in the carotid siphons. Absence of intracranial hematomas.

MRI of the Brain 24/07/2024

No acute ischemic vascular insults were characterized in the present study. Foci of signal alteration in the hemispheric white matter, nonspecific, usually resulting from chronic microvascular changes / gliosis (Fazekas 2). Image suggestive of an ischemic lacune in the left lentiform nucleus. Encephalic volume reduction, expected for the age group. Important clinical correlation.

Electroencephalogram performed on 02/08/2024

The EEG showed regular, continuous, symmetrical, and disorganized baseline activity with a burst-suppression pattern. During the bursts, there is a predominance of diffuse, irregular delta rhythms, interspersed with diffuse, irregular theta rhythms. Epileptiform activity of the slow-wave spike-and-wave type is frequent in the right frontal region. Three seizures were observed. The seizures had an ictal onset in the right cerebral hemisphere with higher amplitude in the temporal region in the theta range.

The electrographic end was characterized by two seizures with a slow-wave spike-and-wave pattern in the right temporal region and one seizure with a beta rhythm. The seizures lasted from 1 to 2 minutes. The intermittent photic stimulation did not alter the tracing. The

auditory and painful stimuli did not modify the tracing. Brain mapping and spectral analysis revealed a dominant rhythm of 1.50Hz and 25.1μV. Cerebral topography revealed the following predominant potentials with their respective locations: Delta 29.5μV in O2, Theta 18.1μV in F8, Alpha 10.8μV in O2, Beta 12.9μV in OZ. Conclusion: The EEG shows disorganization of the baseline activity with a burst-suppression pattern. Three electroclinical seizures were observed.

Case progression

The patient remained in the ICU for 18 days, evolving with a severe general condition, but hemodynamically stable and without the use of vasopressor drugs. Nevertheless, the patient continued on mechanical ventilation with the need for a tracheostomy and sedation. The patient remained 48 hours without vasopressor drugs and was on valproic acid, clobazam, phenobarbital, and levetiracetam due to persistent myoclonus. The patient also continued with dialysis as per nephrology guidance (dialysis-dependent chronic kidney disease).

After this period, the patient progressed in less than 10 hours to the absence of vital signs on the monitor. Central pulses were checked, and they were absent. The monitor showed a rhythm of asystole. In light of this situation, a cardiopulmonary resuscitation protocol was initiated as per Advanced Cardiovascular Life Support (ACLS). Additionally, sodium bicarbonate infusion was performed to correct metabolic acidosis. Despite the support, after 40 minutes of the protocol, the patient remained in asystole. Pupils were checked and found to be dilated and non-reactive to light, with no return of spontaneous circulation. Therefore, the patient's death was confirmed, and the death certificate was filled out.

DISCUSSION

Status epilepticus (SE) is a serious and potentially fatal neurological condition characterized by the persistence of seizures or the occurrence of repeated seizures without return to consciousness. Its pathophysiology involves an imbalance between excitatory and inhibitory mechanisms in the brain, leading to neuronal hyperexcitability and the propagation of epileptic discharges.[5]

The main cause of this imbalance is the deficiency of inhibitory neurotransmitters, such as GABA (gamma-aminobutyric acid), and the increased activity of excitatory neurotransmitters, such as glutamate. Understanding the mechanism of status epilepticus (SE) was crucial in deciding the appropriate treatment, as prolonged seizures, such as those observed in this patient, can lead to permanent brain damage if not rapidly controlled. This alteration results in disorganized electrical activity in the brain areas responsible for motor coordination and cognition, such as the cerebral cortex, hippocampus, and thalamus. If status epilepticus is not quickly controlled, irreversible brain damage can occur due to the high metabolic demand of neuronal cells, leading to neuronal cell death and impairment of cognitive and motor functions.[6]

The symptoms of status epilepticus vary depending on the affected brain area but typically include prolonged or repeated seizures. Additionally, the patient often experiences loss of consciousness and may develop autonomic changes such as tachycardia, hypertension, hypotension, or excessive sweating. In severe cases, the patient may experience hypoxemia and respiratory failure due to the disruption of normal ventilation. The postictal syndrome,

characterized by confusion, drowsiness, and memory difficulty, can persist for hours after the seizure. Furthermore, patients with status epilepticus may experience acute cognitive impairment, leading to difficulties with orientation and reasoning. The clinical picture may be exacerbated in non-convulsive seizures, where mental alteration may be the only visible sign of the condition.[3]

The diagnosis of status epilepticus is primarily clinical, based on the patient's history and observation of the seizures. To confirm the diagnosis and monitor brain activity, electroencephalography (EEG) is crucial. The EEG reveals continuous epileptic activity, which is characteristic of status epilepticus. In cases where the seizure is not easily detected, such as in non-convulsive status epilepticus, the EEG may be the only indicator of abnormal brain activity. As presented by the patient in question, in the reported experience, after excluding other diagnostic possibilities, abnormal activity was identified in the EEG.[7]

Additionally, imaging exams such as computed tomography (CT) or magnetic resonance imaging (MRI) can be useful in identifying underlying structural causes, such as tumors or brain infections, that may be contributing to the condition. Other laboratory tests, such as complete blood count, blood gas analysis, liver and kidney function, and glucose levels, are important to assess the metabolic and systemic conditions that may exacerbate the epileptic status. The EEG is the diagnostic test of choice for confirming the diagnosis of status epilepticus. It helps identify patterns of epileptic discharges that are indicative of continuous abnormal neuronal activity. CT or MRI of the brain are important to rule out structural causes such as tumors, abscesses, or brain hemorrhages, which may predispose to epileptic episodes.[1]

Additionally, laboratory tests, including sodium, calcium, glucose, and renal and liver function, are essential to detect metabolic or toxic disturbances that may contribute to the status epilepticus. Arterial blood gas analysis is relevant to assess respiratory changes, such as hypoxemia, which can worsen the patient's condition. Finally, toxicological tests may be necessary to rule out intoxications with drugs that induce seizures.[4]

The differential diagnosis of status epilepticus should include several neurological and systemic conditions that may present with similar symptoms. Syncope (brief loss of consciousness) can mimic epileptic seizures, but it is usually of short duration and is not associated with convulsive movements. Stroke (CVA), especially when it involves motor areas, can result in loss of consciousness and involuntary movements, but it is not typically accompanied by continuous electrical activity as seen in status epilepticus.[3]

Metabolic disorders, such as hypoglycemia or hyponatremia, can also cause neurological symptoms similar to seizures, but the difference is that there are no EEG changes typical of seizures. Hypoxia, caused by severe respiratory failure, can lead to loss of consciousness, but it is not associated with continuous epileptic discharges. Finally, psychoses or panic attacks may result in mental confusion, but they lack the objective signs of seizures or abnormalities on the EEG.[8]

The treatment of status epilepticus aims to quickly and effectively stop the seizures to prevent severe complications, such as permanent brain damage. The initial approach involves the intravenous administration of benzodiazepines, such as lorazepam or diazepam, which have a rapid and effective effect on epileptic activity. If status epilepticus persists, additional medications, such as phenytoin or fosphenytoin, may be used for long-term control. In refractory cases, where seizures do not respond to conventional treatments, barbiturates

(such as phenobarbital) or anesthetic agents, like propofol, may be required.[8,9]

The biggest challenge was dealing with refractory status epilepticus. The conventional treatment, which initially included benzodiazepines and antiepileptic drugs, was not sufficient to stop the seizures. This required a more aggressive approach with the use of barbiturates and propofol, which are often used in refractory status epilepticus cases but also present risks, such as respiratory depression and hypotension.[10]

This experience gave me a deeper understanding of therapeutic options in refractory status epilepticus, as well as highlighting the importance of well-structured protocols and interdisciplinary collaboration to ensure the success of treatment. More recent strategies include the use of cannabinoids for refractory epilepsies and therapy with topiramate to prevent subsequent seizures.[4,8] Careful monitoring of vital signs, respiratory function, and metabolic conditions is essential during treatment.

Status epilepticus can lead to severe complications if not treated appropriately. These complications include irreversible neuronal damage, hypoxia due to disruption of normal ventilation, respiratory failure, and hypotension. The patient may also develop cardiovascular complications, such as arrhythmias, and a stroke (CVA) due to the increased metabolic demand in the brain.[4,8]

Additionally, refractory status epilepticus (when treatment is ineffective) can lead to permanent cognitive sequelae, such as memory difficulties, disorientation, and motor deficits. The mortality associated with status epilepticus is significant, particularly in patients with comorbidities or in refractory states, and can be exacerbated by conditions such as infections and uncorrected metabolic disorders2. Furthermore, the need for adjustments in long-term medications was discussed with the medical team, considering the patient had a history of hypertension, chronic kidney disease, and prostate cancer being treated with chemotherapy. The use of antiepileptics could interact with other ongoing therapies, requiring close monitoring of renal function and drug levels. Despite the team's efforts, the patient progressed to death after 18 days.

FINAL CONSIDERATIONS

This clinical case illustrates the complexity of managing patients with traumatic brain injury in the presence of comorbidities and oncological treatments, making the experience complex and requiring delicate management. Early identification of potential causes of cognitive and neurological alterations is crucial for appropriate treatment and prevention of complications. The patient's progression depends on the integrated management of underlying conditions, such as prostate cancer, kidney disease, and blood pressure control, in addition to strict monitoring in the ICU.

Status epilepticus is a neurological emergency with an imminent risk of severe complications, including irreversible brain damage, respiratory failure, and mortality. Its pathophysiology is related to an imbalance between excitatory and inhibitory activity in the brain, leading to continuous convulsive activity. The diagnosis is primarily confirmed through EEG, with early recognition and rapid treatment being essential to prevent damage. Initial therapies include benzodiazepines, followed by other antiepileptic drugs in refractory cases.

This experience was crucial for gaining a deeper understanding of status epilepticus and therapeutic approaches in a real-life scenario. The use of benzodiazepines and antiepileptic drugs, followed by more invasive interventions such as propofol and phenobarbital, proved effective but also highlighted the challenges associated with treating refractory status epilepticus. Continuous monitoring of respiratory function, vital signs, and careful observation of postictal complications were essential in managing the patient. Collaboration among intensive care teams, neurologists, and other specialists was key to patient care, and this experience provided a more holistic view of managing this neurological emergency.

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