International Journal of Research in Medical Sciences
Davis E et al. Int J Res Med Sci. 2017 May;5(5):2251-2253
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20171881

Case Report

Non-convulsive status epilepticus: an often-overlooked etiology of syndrome of inappropriate antidiuretic hormone secretion

Elizabeth Davis, Rima Chakraborty*

University of Minnesota, Minneapolis, MN, USA

Received: 26 February 2017
Accepted: 25 March 2017

***Correspondence:**
Dr. Rima Chakraborty,
E-mail: chak004@umn.edu

ABSTRACT

Altered mental status is a common presenting complaint in adult medicine with a broad differential diagnosis. When found in the context of chronic medical conditions, less common etiologies can be overlooked. We present a case of acute altered mental status thought to be secondary to acute on chronic hyponatremia in the context of syndrome of inappropriate antidiuretic hormone secretion (SIADH), eventually diagnosed as non-convulsive status epilepticus, partial type. We report the case of a 67-year-old patient with known SIADH of unknown etiology, hypertension, chronic pancreatitis and chronic obstructive pulmonary disease (COPD) who presented with fatigue, myalgia, decreased urine output. On presentation patient also had profound acute on chronic hyponatremia. During sodium correction, the patient developed an acute, progressive decline in mental status. Vital signs remained stable and workup including LP and MRI were negative. Initial electroencephalographic (EEG) showed no definitive seizure activity, but did show bifrontal focal continuous slowing. The patient's mental status continued to decline and upon further evaluation it was suggested that the EEG findings and the patient's progressive AMS could be compatible with non-convulsive status epilepticus. The patient received loading doses of IV lorazepam and levetiracetam and within 48 hours after initial treatment was back to baseline. Non-convulsive status epilepticus is a common, but heterogeneous subclass of status epilepticus that is difficult to diagnose. This case demonstrates the difficulty of diagnosing normalized corrected Shannon entropy (NCSE) in the context of other chronic medical conditions and the importance of including it on any differential diagnosis for acute change in mental status.

Keywords: EEG, Hyponatremia, NSCE, SIADH

INTRODUCTION

SIADH is a cause of euvolemic hyponatremia.[1] SIADH was first described in 1957 in a patient suffering from hyponatremia in which no physiologic stimulus for ADH release was identified.[2] SIADH has historically been associated with malignancy, many other associations exist including infections, CNS demyelinating disorders and medications.[1]

The severity and duration of hyponatremia plays a large role in presentation. Hyponatremia is defined as a plasma sodium concentration of less than 135mmol/L, but symptomatic hyponatremia generally does not occur until profound hyponatremia occurs, defined as a plasma sodium level below 120mmol/L.[3]

Acute changes (<48hours) and profound hyponatremia are strongly correlated with onset of neurologic symptoms, while chronic hyponatremia may be asymptomatic due to the ability of the brain to compensate over time.[1] Neurologic symptoms of hyponatremia include headache, difficulty concentrating, impaired memory, seizures and weakness.[4]

Status epilepticus (SE) is defined as continuous seizure activity for greater than five minutes or more than one seizure within five minutes without recovery between episodes.[5] SE can be subcategorized based on seizure etiology or pathophysiology. One category, non-convulsive status epilepticus (NCSE), is defined as continuous seizure activity that lacks the classic motor and sensory findings of SE and often presents with acute changes in mental status.[6,7] NCSE is very challenging to diagnose and there is little consensus regarding clinical or EEG diagnostic criteria.[7] In this case report, we discuss a patient who presented with acute on chronic hyponatremia secondary to SIADH who developed progressively worsening mental status. While the changes in mentation were initially attributed to the patient's severe hyponatremia, the patient was eventually diagnosed with non-convulsive status epilepticus. This case report illustrates the challenge of diagnosing SE as a cause of altered mental status in a patient presenting with SIADH.

CASE REPORT

A 67-year-old patient with past medical history of SIADH of unknown etiology (baseline Na 128-130 mmol/L), COPD, chronic pancreatitis and hypertension presented to the Emergency Department with a one week history of fatigue, weakness, myalgia and decreased urine output. On presentation, vital signs were stable and within normal limits. Exam was pertinent for mild bilateral inspiratory wheezes and dry mucous membranes. Patient's outpatient medications included Amlodipine, Hydrochlorothiazide (HCTZ), Lisinopril and Lorazepam.

Laboratory investigation was significant for sodium of 108 mmol/L, chloride 69 mmol/L and serum osmolarity (Osm) of 229. Urine osmolarity (UOsm) was 343 and urine sodium 48. Chest x-ray and labs were otherwise negative.

Initially the patient's' overall presentation was suggestive of weakness and fatigue secondary to acute on chronic hyponatremia. The history suggested that the chronic hyponatremia had been acutely compounded by poor oral intake and use of HCTZ. HCTZ was a relatively new medication for the patient and upon admission HCTZ was held and patient was started on normal saline overnight. The next morning the sodium had corrected to 115 mmol/L (serum Osm 229, UOsm 343). IV fluids were discontinued and free water intake was restricted to 2L/d. Over the next day the sodium corrected to 120 mmol/L in 24 hours. Patient mentation did not change with the correction from 108 to 120 in 48 hours.

On hospital day 3 the patient began feeling increasingly confused, with disorganized thinking and began displaying deficits in short term memory. There was initial concern for demyelination syndrome secondary to possible sodium overcorrection, however an MRI of the brain was negative for acute changes. Patient mental further deteriorated with disorganized thinking and fluctuating orientation throughout hospital days 3-8. During the same time sodium stayed stable in 120-122. LP was negative for acute process. A repeat MRI was negative and an EEG showed bilateral focal frontal continuous slowing, but no distinct epileptiform discharges. Further neoplastic lab workup and imaging were negative.

On hospital day 9 it was suggested that the nonspecific findings on EEG and the patient's acute progressive altered mental status could be compatible with non-convulsive status epilepticus. The patient was treated with loading doses of IV lorazepam and levetiracetam and within 24 hours became alert and oriented and was significantly more interactive. By 48 hours' patient was back to prior to admission cognitive function. The sodium improved to 128 within 24 hours. Patient remained on 1000mg levetiracetam as anti-seizure prophylaxis and repeat EEG showed resolution of the bifrontal slowing and no further abnormalities.

DISCUSSION

The differential diagnosis for acute onset altered mental status is broad and includes neurologic, infectious and metabolic etiologies. Given the underlying SIADH, the team initially focused on the correction of the acute on chronic hyponatremia as the etiology for the altered mental status. Profound hyponatremia (serum sodium <120 mmol/L), especially in the acute setting (<48hours), can lead to cerebral edema and neurologic dysfunction including headache, nausea, vomiting, disorientation, muscle cramps, lethargy and decreased reflexes.[4]

The optimal correction of profound hyponatremia has been a source of debate, but current standards suggest a maximum correction of 10 mmol/24hours with an initial correction closer to 5 mmol/24hours.[8] Osmotic demyelination syndrome (ODS) is a rare syndrome that can occur if sodium is corrected too rapidly, especially if sodium is corrected at a rate greater than 12-14 mmol/24hours.[4]

The concern for ODS as the cause of AMS in our patient was due to the fact that the sodium was initially overcorrected with an increase of 15 mmol in 26 hours. The overcorrection was adjusted with boluses of D5W. Risk of ODS due to overcorrection of profound hyponatremia ranges in the literature from 1% to as high as 11% and is assessed using either CT or MRI brain imaging, which was negative in our patient.[8-10]

Routine EEG was recommended by neurology to assess for seizure activity and showed bifrontal slowing, but no definitive epileptiform activity. Given the clinical context and workup, it was suggested the EEG findings could be a sign of atypical seizure activity caused by non-convulsive status epilepticus (NCSE). This diagnosis was

confirmed by marked clinical improvement with loading doses of levetiracetam and lorazepam and resolution of bifrontal slowing on subsequent routine EEG. NCSE is a heterogeneous process defined as continuous seizure activity that generally lacks stereotypical alteration in motor function or sensation. Presentation can vary widely, but the primary clinical presentation is altered mental status that usually manifests as slowed mentation, disorientation and bizarre or inappropriate behavior.[6,7]

Subtle motor symptoms may be present, including eye deviation, nystagmus or rhythmic twitching that can be overlooked.[7] Despite the fact that NCSE may make up 20-25% of all cases of status epilepticus, it is often difficult to diagnose.[12,6,7]

In present case, the EEG findings did not show classic seizure activity, but given the context there was high clinical suspicion that the findings were consistent with NCSE. Like all seizures, triggers for NCSE are widely varied and etiology is often multifactorial. In our case, it was likely a combination of acute on chronic profound hyponatremia and prior to admission discontinuation of lorazepam that lowered his seizure threshold.

Our patient's chronic SIADH, despite extensive workup, is still of unknown etiology. At this point it is difficult to elucidate whether the patient's chronic SIADH was a contributing factor to lowering his seizure threshold, or a consequence of the underlying NCSE itself and possibly a chronic, previously undiagnosed, seizure disorder. This case illustrates the difficulty in diagnosing NCSE, especially in the context of other chronic medical conditions like SIADH. It also demonstrates that diagnosis with SIADH is incomplete without determination of the etiology and appropriate workup should always be pursued.

CONCLUSION

Non-convulsive status epilepticus is a common, but heterogeneous subclass of status epilepticus that is difficult to diagnose. This case demonstrates the difficulty of diagnosing NCSE in the context of other chronic medical conditions and the importance of including it on any differential diagnosis for acute change in mental status.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

REFERENCES

1. Ellison DH, Berl T. The Syndrome of Inappropriate Anti-diuresis N Engl J Med. 2007;356:2064-72.
2. Schwartz WB, Bennett W, Curelop S, Bartter FC. A syndrome of renal sodium loss and hyponatremia probably resulting from inappropriate secretion of antidiuretic hormone. Am J Med. 1957;23:529-42.
3. Goh KP. Management of Hyponatremia. Am Family Physician. 2002;69:2387-94.
4. Adrogué HJ, Madias NE. Hyponatremia. N Engl J Med. 2000;342:1581-9.
5. Lowenstein, DH, Alldredge, BK. Status Epilepticus N Engl J Med. 1998;338:970-6.
6. Kaplan PW. Nonconvulsive Status Epilepticus. Neurology. 2003;61:1035-6.
7. Chang AK, Shinnar, S Nonconvulsive Status Epilepticus, Emergency Med Clin N Am. 2011;29:65-7.
8. Geoghegan P, Harrison AM, Thongprayoon C, Kashyap R, Ahmed A, Dong Y, et al. Sodium Correction Practice and Clinical Outcomes in Profound Hyponatremia. Mayo Clin Proc. 2015;90:1348-55.
9. Vu T, Wong R, Hamblin PS, Zajac J, Grossman M. Patients Presenting with Severe Hypotonic Hyponatremia: Etiological Factors, Assessment and Outcomes. Hosp Pract. 2009;37:128-36.
10. DeWitt LD, Buonanno FS, Kistler JP, Zeffiro T, DeLaPaz RL, Brady TJ, et al. Central pontine myelinolysis: demonstration by nuclear magnetic resonance. Neurology. 1984;34:570-6.
11. Celesia GG, Modern Concepts of Status Epilepticus. JAMA. 1976;235:1571-4.
12. Dunne JW, Summers QA, Stewart-Wynne EG. Non-convulsive status epilepticus: a prospective study in an adult general hospital. Q J Med. 1987;62:117-126.

Cite this article as: Davis E, Chakraborty R. Non-convulsive status epilepticus: an often-overlooked etiology of syndrome of inappropriate antidiuretic hormone secretion. Int J Res Med Sci 2017;5:2251-3.