

Anti-Voltage-Gated Potassium Channel Antibody Syndrome: A Rare Cause of Hyponatremia in Intensive Care Unit

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Abstract

Hyponatremia causing seizure is a common cause for admission in the critical care unit. Here, we describe a peculiar case of seizure due to hyponatremia, associated with anti-voltage-gated potassium channel antibody syndrome. This case emphasizes that how a proper workup can unveil unusual but potentially treatable causes of hyponatremia. The hallmark of this syndrome is that neurological symptoms may relapse or progress if the disorder is not recognized in time. This case report emphasizes the point that how a keen observation may decode subtle signs of the grave but potentially treatable pathologies.

Keywords: Anti-voltage-gated potassium channel antibody syndrome, hypokalemia, hyponatremia, seizure, syndrome of inappropriate antidiuretic hormone secretion

INTRODUCTION

Dyselectrolytemia frequently gets complicated with seizures or arrhythmia requiring admission to critical care units. Sodium disorders are common electrolyte disturbances in clinical medicine, associated with increased rates of morbidity and mortality.^[1] Here, we describe a peculiar case of seizure due to hyponatremia that got complicated with aspiration pneumonitis. This case emphasizes that besides electrolyte correction, how a proper workup can unveil some very unusual but potentially treatable causes of hyponatremia, or it may indicate some grave pathology like a malignancy. In this case, syndrome of inappropriate antidiuretic hormone secretion (SIADH) was the cause of hyponatremia. In-depth history of the patient and further testing suggested anti-voltage-gated potassium channel antibody syndrome (anti-VGKC) to be the cause which was successfully treated. Still, it is frequently underdiagnosed and hence undertreated condition. In the Indian subcontinent, it is relatively new, as the first case report was published in 2007.^[2] Such cases usually report in neuro or psychiatry departments, but the presentation in intensive care with seizure, hyponatremia, hypokalemia, and polyuria was rather unique to our case.

CASE REPORT

A 58-year-old male, nonsmoker, nonalcoholic, nondiabetic but hypertensive (on amlodipine 10 mg O.D), presented in emergency with convulsions. Relatives denied fever, headache, vomiting but admitted bilateral upper and lower limb weakness with generalized fatigue, and irrelevant talk progressively increasing over the past 15 days. No significant medical or surgical history was seen.

Systemic examination was normal except low Glasgow coma scale (E1M4V2) and bilateral crepitations on chest auscultation. Initial impression was the postictal state with aspiration. His hemogram, renal and liver function tests were normal except for hyponatremia and hypokalemia. Random blood sugar was 129 mg/dl, and arterial blood gases showed pH 7.45 PaO₂ 58 mm Hg PaCO₂ 29 mm Hg. The patient was severely tachypneic and was managed with mechanical ventilation, electrolyte correction and intravenous (i.v.) piperacillin-tazobactam, pantoprazole and fosphenytoin.

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Access this article online

Quick Response Code:



Website:
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DOI:
10.4103/ijccm.IJCCM_480_17

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How to cite this article: Sulakshana S, Prakash S. Anti-voltage-gated potassium channel antibody syndrome: A rare cause of hyponatremia in intensive care unit. Indian J Crit Care Med 2018;22:746-8.

Brain magnetic resonance imaging ruled out cerebrovascular accident, the cerebrospinal fluid analysis was normal. Thyroid function tests, serum cortisol, protein, and lipid profile were normal. Serum osmolality was 235 mosm/kg, urine osmolality 149 mosm/kg serum and urine sodium were 109 and 149 mmol/L, respectively, suggesting SIADH as a diagnosis.

After regaining consciousness, the patient stated that he had frequent episodes of twitching in both lower limbs for past 1 month, but he ignored them. Further testing showed positive for anti leucine-rich glioma inactivated 1 and anti-contactin associated protein 2 antibodies, N-acetyl-cysteine-activated creatine kinase-474U/L and serum lactate dehydrogenase-391U/L. This laboratory report opened a new window for the management of the patient, and the patient was managed with three cycles of plasmapheresis and i.v. corticosteroids followed by oral corticosteroids. Knowing the association of anti-VGKC syndrome with thymoma or small cell adenocarcinoma, contrast-enhanced computed tomography abdomen and thorax was done which was normal. Patients' symptom resolved significantly after treatment. The patient was counseled for follow-up as anti-VGKC syndrome may precede the detection of malignancy, but they did not turn up.

DISCUSSION

Hyponatremia is an important metabolic cause of the seizure.^[3] SIADH accounts for about one-third of all hyponatremia presentations and is the most common cause of hyponatremia.^[4,5] yet remains commonly under-diagnosed and, therefore, under-managed.^[6] Diagnosis is made by clinical euvolemic state with low serum sodium and osmolality, raised urine sodium and osmolality, and exclusion of pseudohyponatremia and diuretic use.^[7] However, SIADH can be multifactorial, and it may indicate some rare but potentially treatable causes or some grave pathology like malignancy. A timely diagnosis will have a profound effect on the patients' outcome.

Anti-VGKC syndrome is a very rare cause of hyponatremia. However approximately 80% of cases of VGKC encephalitis develop hyponatremia at some time during the illness, generally as a result of SIADH.^[8,9] Limbic system has an important role in sodium and water balance.^[5] Limbic encephalitis is an important component of the anti-VGKC syndrome.^[8] Hence, there might be a hidden link between anti-VGKC antibody syndrome and SIADH via the limbic pathway, yet to be confirmed. Patients with anti-VGKC antibody syndrome often report in neuro and psychiatry departments due to twitching or memory fluctuations and hallucination. Presentation in intensive care with seizure, critical hyponatremia, hypokalemia, and polyuria was rather unique to our case. SIADH is not commonly associated with hypokalemia, but it might indicate the severity of water intoxication.^[10] Correction of hyponatremia was initially done with 3% saline and later on normal saline. Probably, this normal saline was responsible for polyuria and hypokalemia, as it normalized when further

tests showed a diagnosis of SIADH and patient was managed with fluid restriction.

Since 2007 when the first case report from India was published, it is still an under-recognized condition possibly due to low index of suspicion and paucity of diagnostic test availability.^[2] It would be more evident from a recent article published in 2017 whereby authors reported a case of psychogenic polydipsia which was managed with vasopressin antagonists and behavior therapy. However, the laboratory reports in that article itself do not suggest it to be a case of psychogenic polydipsia rather high urine osmolality, and high urine sodium suggested it to be a case of SIADH. The patient was well managed by water restriction as it is also the mainstay of therapy in SIADH.^[11] This imposes the need of always looking for red-flag signs of seizure, autonomic instability and movement disorder in patients presenting with first episode psychosis that may have an auto-immune etiopathogenesis.^[12]

The presentation with twitching, subtle movement disorder, memory impairment, and hyponatremia provides an early opportunity for diagnosis, which if missed may lead to an acute life-threatening process. The fact that such patients present initially to primary care highlights the importance of the understanding this syndrome by general practitioners, psychiatrists, and intensivists as well.^[13]

CONCLUSION

The hallmark of this syndrome is that neurological symptoms may relapse or progress if the disorder is not recognized in time. This case report is also unique in the sense that none of the previous anti-VGKC syndrome and SIADH association showed hypokalemia and polyuria as an important consideration, which was seen in our case. Anti-VGKC antibody syndrome is a rare disease, which, if detected, is amenable to treatment. This case report emphasizes the point that how a keen observation may decode subtle signs of the grave but potentially treatable pathologies.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understand that name and initials will not be published and due efforts will be made to conceal identity, but anonymity cannot be guaranteed.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Braun MM, Mahowald M. Electrolytes: Sodium disorders. *FP Essent* 2017;459:11-20.
2. Bajaj BK, Shrestha S. An interesting case report of Morvan's syndrome from the Indian subcontinent. *Neurol India* 2007;55:67-9.

3. Castilla-Guerra L, del Carmen Fernández-Moreno M, López-Chozas JM, Fernández-Bolaños R. Electrolytes disturbances and seizures. *Epilepsia* 2006;47:1990-8.
4. Laville M, Burst V, Peri A, Verbalis JG. Hyponatremia secondary to the syndrome of inappropriate secretion of antidiuretic hormone (SIADH): Therapeutic decision-making in real-life cases. *Clin Kidney J* 2013;6:i1-20.
5. Pillai BP, Unnikrishnan AG, Pavithran PV. Syndrome of inappropriate antidiuretic hormone secretion: Revisiting a classical endocrine disorder. *Indian J Endocrinol Metab* 2011;15 Suppl 3:S208-15.
6. Huda MS, Boyd A, Skagen K, Wile D, van Heyningen C, Watson I, *et al.* Investigation and management of severe hyponatraemia in a hospital setting. *Postgrad Med J* 2006;82:216-9.
7. Tee K, Dang J. The suspect-SIADH. *Aust Fam Physician* 2017;46:677-80.
8. Newey CR, Sarwal A. Hyponatremia and voltage gated potassium channel antibody associated limbic encephalitis. *J Neurol Neurophysiol* 2014;5:195.
9. Irani SR, Michell AW, Lang B, Pettingill P, Waters P, Johnson MR, *et al.* Faciobrachial dystonic seizures precede Lgi1 antibody limbic encephalitis. *Ann Neurol* 2011;69:892-900.
10. Padfield PL, Morton JJ. Potassium in the syndrome of inappropriate antidiuretic hormone secretion. *Postgrad Med J* 1979;55:721-2.
11. Bhatia MS, Goyal A, Saha R, Doval N. Psychogenic polydipsia-management challenges. *Shanghai Arch Psychiatry* 2017;29:180-3.
12. Rickards H, Jacob S, Lennox B, Nicholson T. Autoimmune encephalitis: A potentially treatable cause of mental disorder. *Adv Psychiatr Treat* 2014;20:92-100.
13. Yaxley J. Confusion, faciobrachial dystonic seizures, and critical hyponatremia in a patient with voltage-gated potassium channel encephalitis. *Korean J Fam Med* 2017;38:99-101.