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Syndrome of inappropriate antidiuretic hormone as the initial presentation in Guillain-Barré syndrome

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ABSTRACT

Background: Guillain-Barré syndrome (GBS) is an autoimmune disease damaging the peripheral nervous system. It commonly presents as rapidly progressing bilateral symmetrical motor weakness. There has been known association of syndrome of inappropriate antidiuretic hormone (SIADH) in patients with GBS though it is rare.
Case Presentation: We report a patient with rare clinical presentation of SIADH before the onset of motor deficits in GBS.
Conclusions: SIADH as an initial finding in patients with GBS is very rare. This case report emphasizes the importance of early detection of SIADH in GBS to avoid delay in treatment.

Case Report

Implication for health policy/practice/research/medical education:
Our case report emphasizes that SIADH could precede the development of profound motor deficit in patients with Guillain-Barré syndrome. Physicians should be aware of this rare presentation to prevent delays in diagnosis and proper treatment.
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1. Background

Guillain-Barré syndrome (GBS) is the most common cause of acute flaccid paralysis in the United States with incidence of 1-2 cases per 100 000 patients (1,2). The pathophysiological mechanism is hypothesized to be immune mediated where antecedent infection or other triggers provokes an immune response causing nerve injury (3). Autonomic dysfunction occurs in about 70% patients with GBS. Although there is a known association of syndrome of inappropriate antidiuretic hormone (SIADH) in patients with GBS, it usually occurs later when there is maximal motor deficit. SIADH as one of the initial presentations in GBS is very rare; therefore, this case report promotes physician awareness to the potential early presentation of GBS to prevent delays in diagnosis and treatment.

2. Case Presentation

A 72-year-old functionally independent female, presented with complaints of bilateral lower extremity weakness and difficulty ambulating. She also reports having numbness and tingling in bilateral feet, as well as nausea. Her past medical history is significant for dyslipidemia, left lower extremity sciatic pain. Her only medication is Simvastatin. Two days prior to presentation she was diagnosed with urinary tract infection and was given cefpodoxime. She denies any urinary incontinence, shortness of breath, or recent trauma. No history of recent travel or vaccinations.

On presentation, vitals were stable and the initial neurological exam showed no motor or sensory deficit. Sodium level on presentation was 128 mEq/L. The patient was placed on normal saline intravenous hydration despite which her sodium dropped to 115

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mEq/L. Her serum osmolality was (245 osm/L), with an elevated urine osmolality (702 osm/L), suggestive of SIADH. The patient was placed on fluid restriction to 1L per day, and her serum sodium level continued to decline to 113 mEq/L, and intravenous hydration of 3% saline was then initiated despite which her serum sodium continued to decline. She was later started on oral tolvaptan 15 mg daily for 7-days for management of SIADH. Hospital course was complicated by progressive lower extremity weakness. On day 6 of hospitalization, physical examination revealed bilateral lower extremity strength of 1/5. Non-contrast CT of the chest, magnetic resonance imaging (MRI) of brain and cervical, thoracic, lumbar spine did not show any acute changes. Lumbar puncture performed showed a CSF total protein 288, RBC 1000, WBC 3, clear, colorless – findings suggestive of GBS. Therapeutic plasmapheresis exchange (TPE) was initiated and patient completed 5 sessions. Her sodium level stabilized between 134-137 mEq/L. With aggressive treatment and inpatient physical therapy, patient regained her strength and was able to walk without assistance upon discharge.

3. Discussion

GBS commonly presents as a progressive symmetrical muscle weakness with absent or decreased deep tendon reflexes. Paresthesia and tingling is also common. The most dreaded complication however is severe respiratory muscle weakness requiring ventilatory support to protect the airway. Autonomic dysfunction occurs in about 70% of the patients causing symptoms such as urinary retention, diarrhea/constipation, tachycardia/bradycardia, hyponatremia and SIADH (4). The incidence of SIADH in GBS is about 4.8% (5).

Association between SIADH in GBS although rare, has been reported in previous case reports. Pathogenesis of SIADH in GBS is unknown, but is hypothesized to be due to resetting of the osmoreceptor response. It could also be caused by increased sensitization of the renal tubule to vasopressin's action (6). Recent studies have also shown interleukin (IL-6) as the cause of hyponatremia in GBS (7). Pseudohyponatremia can occur in GBS as an artifact as result of intravenous immunoglobulin (IVIG) administration, which is the mainstay of treatment in management of patients with GBS.

In our case, the patient was found to have hyponatremia on admission prior to the development of motor deficit in GBS and was diagnosed with SIADH requiring treatment with tolvaptan and plasmapheresis. Her sodium level stabilized only after the treatment of the underlying GBS was treated and addressed. After completion of five sessions of TPE, the patient's sodium level remained stable within normal range even after discontinuing tolvaptan. SIADH usually presents in GBS at later stages when there is profuse motor deficit. Ramanathan et al and Hoffmann et al described a case of SIADH and dysautonomia as the initial presentation in GBS (8,9). To our knowledge only two cases of GBS presenting with SIADH as the initial sign has been reported. Recent observational studies have shown that hyponatremia in patients with GBS is associated with poor outcomes and increase mortality (10,11). A prospective study done by Saifudheen et al found that SIADH is associated with the severity of the disease and is an indicator of poor prognosis (10).

4. Conclusions

Our case report emphasizes that SIADH could precede the development of profound motor deficit in patients with GBS. Physicians should be aware of this rare presentation to prevent delays in diagnosis and proper treatment. Further studies are necessary to understand the pathophysiology behind SIADH in GBS and its impact on the prognosis of the patients.

Authors' contribution

NF; drafting of manuscript, clinical consultant and acquisition of data. DA; drafting of manuscript, clinical consultant and acquisition of data. JW; critical revision of manuscript. BMM; critical revision of manuscript. EEC; contributing author and critical revision of manuscript for important intellectual content. All authors reviewed and approved the final manuscript.

Conflicts of interest

Authors declare no conflict of interests.

Ethical considerations

Informed consent was obtained before report of the patient as a case report.

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References

1. Ayscue P, Van Haren K, Sheriff H, Waubant E, Waldron P, Yagi S, et alAcute flaccid paralysis with anterior myelitis - California, June 2012-June 2014. MMWR Morb Mortal Wkly Rep. 2014;63(40):903-6.
2. Ropper AH. The Guillain-Barré syndrome. N Engl J Med. 1992;326(17):1130-6. Doi: 10.1056/NEJM199204233261706.
3. Yuki N. Molecular Mimicry and Guillain-Barré Syndrome.

Brain Nerve. 2015;67(11):1341-6. doi: 10.11477/mf.1416200304
4. Zochodne DW. Autonomic involvement in Guillain-Barré syndrome: a review. Muscle Nerve. 1994;17(10):1145-55.
5. Anandan C, Khuder SA, Koffman BM. Prevalence of autonomic dysfunction in hospitalized patients with Guillain-Barré syndrome. Muscle Nerve. 2017; 56(2):331-333. doi: 10.1002/mus.25551
6. Cooke CR, Latif KA, Huch KM, Wall BM. Inappropriate antidiuresis and hyponatremia with suppressible vasopressin in Guillain-Barré syndrome. Am J Nephrol. 1998;18(1):71-6.
7. Park SJ, Pai KS, Kim JH, Shin JI. The role of interleukin 6 in the pathogenesis of hyponatremia associated with Guillain-Barré syndrome. Nefrologia. 2012;32(1):114. doi: 10.3265/Nefrologia.pre2012
8. Ramanathan S, McMeniman J, Cabela R, Holmes-Walker DJ, Fung VS. SIADH and dysautonomia as the initial presentation of Guillain-Barré syndrome. J Neurol Neurosurg Psychiatry. 2012;83(3):344-5. doi: 10.1136/jnnp.2010.233767
9. Hoffmann O, Reuter U, Schielke E, Weber JR. SIADH as the first symptom of Guillain-Barré syndrome. Neurology. 1999;53(6):1365. -
10. Saifudheen K, Jose J, Gafoor VA, Musthafa M. Guillain-Barre syndrome and SIADH. Neurology. 2011;76(8):701-4. doi: 10.1212/WNL.0b013e31820d8b40PubMed PMID: 21339497.
11. Sipilä JO, Kauko T, Soilu-Hänninen M. Admission sodium level and prognosis in adult Guillain-Barré syndrome. Int J Neurosci. 2017;127(4):344-349. doi: 10.3109/00207454.2016.1163551.