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Case Report

Post COVID-19 Guillain Barre syndrome with syndrome of inappropriate secretion of antidiuretic hormone

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ABSTRACT

Guillain Barre syndrome (GBS) is a rare but potentially fatal immune mediated disorder of peripheral nerves and nerve roots usually triggered by infections characterized by ascending paralysis with or without sensory symptoms, hyporeflexia to areflexia. Usually preceded by gastrointestinal or respiratory infection. Post COVID-19 neurological manifestation include GBS, transverse myelitis etc., occur at varying incidence rates at various places. Here we report a 42-year-old lady who had COVID-19 recovered presented with quadriparesis with absent deep tendon reflexes with electro-diagnostically proven AMSAN variety of GBS treated successfully with IVIg. Patient was having hyponatremia which was diagnosed to be due to SIADH and was successfully treated with fluid restriction and tolvaptan. This case is being reported due to combination of COVID-19, COVID vaccination shortly before GBS and hyponatremia due to syndrome of inappropriate secretion of antidiuretic hormone (SIADH) which is quite rare combination.

Keywords: COVID-19, SIADH, hyponatremia, vaccine, Acute inflammatory demyelinating polyneuropathy, GBS

INTRODUCTION

Guillain Barre syndrome (GBS) is acute inflammatory demyelinating polyradiculoneuropathy, characterized by rapidly evolving ascending paralysis with mild sensory symptoms, hyporeflexia to areflexia reaching nadir within 4 weeks. Worldwide incidence is around 1-2 cases per 100000. 2/3 are associated with antecedent infection.[1] Campylobacter gastrointestinal infection has been identified predominantly. Following COVID-19 infection many GBS cases are reported.[3] Post COVID vaccine related GBS have also been reported.[5] In GBS hyponatremia has been found around in 5% of patient. Various mechanisms for hyponatremia in GBS have been proposed.[7-9] Here we report a 42-year-old lady who had been vaccinated against COVID-19 first dose and had COVID-19 just prior to GBS with SIADH having significant hyponatremia

CASE REPORT

A 42-year-old well controlled hypothyroid female patient presented to Kasturba medical college, Manipal, Karnataka medicine out patient department on 31/5/2021 with complaints of tingling paresthesia in both lower limbs since 5 days along with progressive weakness in both lower limb. Weakness started distally and progressed proximally. Within two days of onset, patient noticed she was unable to get up from sitting position. She was unable to lift her upper limbs and she was unable to roll in her bed. She had cough while drinking water and taking food along with hoarseness of voice. She could able to sense bladder fullness and had bowel control. Her recent history had included COVID-19 vaccination (inactivated whole virion vaccine) on April 17, 2021. She had mild COVID-19 between 10/05/2021 to 22/05/2021. Her examination showed bilateral lower limb distal power 3/5 and proximal power 1/5. Upper

limb proximal power was 3/5 and distal power 4/5. She had bilateral lower motor neuron type of facial palsy. She had neck muscle weakness. Her deep tendon reflexes were absent bilaterally. Sensory system examination showed bilateral decreased sensation in both feet. Hence a provisional diagnosis of GBS was made. Her CSF analysis showed 9 cells/mm3 with protein >200 mg% (Table 2). Nerve conduction study was consistent with bilateral lower limb and upper limb polyradiculoneuropathy with conduction block (AMSAN) (Figure 1 and 2). Hence patient was treated with IVIG at 2 gm/kg body weight over a period of 5 days. She had subtle signs of respiratory failure in the form of decreased single breath count. She was monitored for worsening respiratory failure closely in intensive care unit. She was having intermittent tachycardia with maximum heart rate reaching up to 140/minute. She gradually showed improvement in general condition and muscle power. On 8th day she was shifted to general ward. Her laboratory values have been depicted in Table 1.

Table 1: Laboratory parameters of the patients.

Parameters	Dates					
	31/05/2021 (admission)	1/06/2021	2/06/2021	3/06/2021	4/06/2021	5/06/2021
Random blood sugar	89 mg%					
Blood urea	28 mg%					
Serum creatinine	0.57 mg%					
Serum sodium	132 meq/l	127 meq/l	124 meq/l	118 meq/l	125 meq/l	124 meq/l
Serum potassium	4.5 meq/l	4.7 meq/l	4.8 meq/l	5 meq/l	4.8 meq/l	4.6 meq/l
T3	1.02 ng/ml					
T4	11.46 ugm/dl					
TSH	3.450 uIU/l					
Hemoglobin	13.4 gm%					
WBC count	9300 cells /mm^3					
Platelet count	4.12 lakhs/mm^3					
Urine spot sodium		212 mmol/l				13 mmol/l
Urine osmolality		769 mosm/kg				
Serum osmolality		270 msolm/kg				
Cortisol			12.7 ugm/dl			
Uric acid			3.3 mg/dl			
Serum sodium	6/6/2021	7/6/2021	8/6/2021	9/6/2021	16/06/2021	19/07/2021
	123 meq/l	127 meq/l	131 meq/l	130 meq/l	137 meq/l	139 meq/l

Table 2: Cerebrospinal fluid analysis report.

Cells	9 cells/mm^3 100% lymphocytes
Protein	>200 mg%
Sugar	68 mg%
Chloride	117 mmol/l
Lactate	20.6 mg/dl

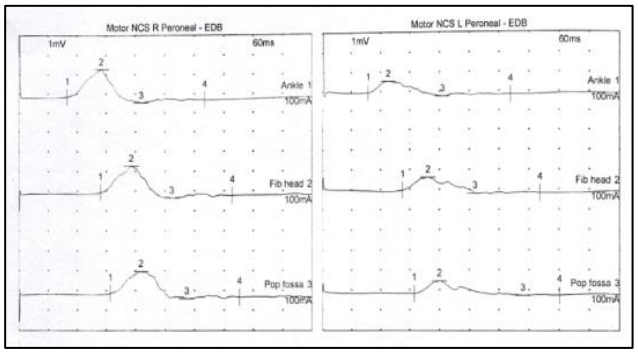

Figure 1: Nerve conduction study tracings of bilateral peroneal nerves showing reduced CMAP (compound muscle action potential).

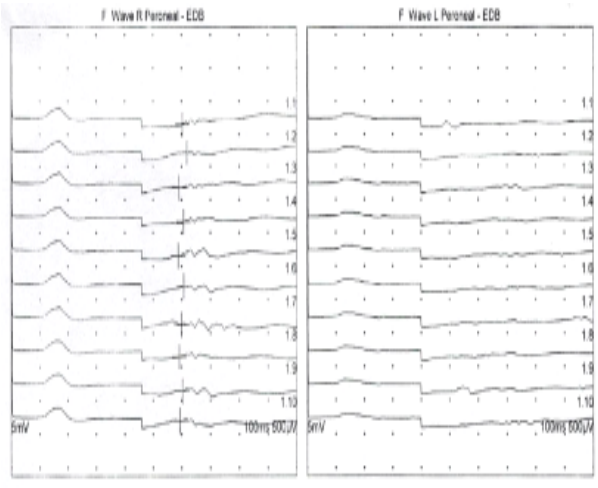

Figure 2: Nerve conduction study of peroneal nerves showing prolonged F wave latency.

She was found to have significant hyponatremia during treatment in intensive care unit which worsened after 2nd day of treatment with IVIg which reached lowest level of

118 meq/l on 3rd day of treatment (admission serum sodium 132 mmol/l). Her urine Na+ and urine and serum osmolarity were consistent with SIADH (urine sodium 212 mmol/l, urine osmolarity 769 mosmol/kg, serum osmolarity 270 mosmol/kg). Other work up for hyponatremia like thyroid function test, serum cortisol, levels were within normal limits. She had not received any diuretics recently before admission. She was treated with fluid restriction and tolvaptan which improved her serum sodium level (serum sodium 137 meq/l at the time of discharge). She was offered a regular physiotherapy and discharged in a good condition with an advice to review in outpatient department after specified period. On follow up at one month she had significantly regained power in limbs with normal serum sodium level and at 3 month she had regained power in both upper and lower limb to almost to the full extent.

DISCUSSION

GBS is a serious rapidly evolving ascending polyradiculoneuropathy of autoimmune origin manifesting as areflexic motor paralysis with or without sensory disturbance. Occurs in 1 to 2 per 100,000 population worldwide.[1] Spectrum of SARS CoV-2 (COVID-19) infection include various neurological manifestations of central and peripheral nervous system.[2,3] Many cases of GBS have been reported following COVID-19. Mechanism of neurological damage by SARS CoV-2 virus is still debated. Possible mechanisms are direct damage to specific receptors, cytokine related injury secondary hypoxia or retrograde travel along nerve fibres.[4] COVID-19 is asymptomatic for a variable period and this makes it difficult to predict time period between onset of infection and development of GBS. Our patient had mild COVID-19 and was in home isolation and recovered completely with symptomatic treatment. Post SARS CoV- 2 vaccine induced various neurological manifestations including GBS, transverse myelitis have also been reported.[5] But still large data are pending. Our patient had also received first dose of SARS CoV-2 vaccine (inactivated whole virion vaccine) in the third week of April 2021. In GBS about 5% of hospitalized patients have SIADH manifesting with significant hyponatremia.[6] Various mechanisms have been postulated for hyponatremia in GBS patients. Hyponatremia correlates with clinical course of GBS and its severity is an indicator of poor prognosis.[6] Hyponatremia may be due to SIADH or due to pseudohyponatremia or due to true hyponatremia. Pathogenesis of SIADH in GBS is incompletely understood. SIADH may precede GBS or develop during course of illness.[7] One mechanism is damage to hypothalamic cells leading to leakage of ADH in to circulation.[7] Another mechanism is resetting of the osmoreceptors in hypothalamus.[8] Recently IL-6 has been implicated in pathogenesis of SIADH in GBS. IL-6 has been found to augment the release of ADH. Second mechanism is true hyponatremia caused by movement of intracellular water into extracellular compartment due to sucrose content in the IVIg solution.[9] Third possible etiology of hyponatremia in GBS is pseudohyponatremia following treatment with IVIg due to large volume of IVIg infused causing dilution of serum sodium by increased protein and lipid content of IVIg with an elevated serum osmolar level. Differentiating between SIADH and pseudohyponatremia is important. In SIADH serum osmolarity will be low and urine sodium and urine osmolarity will be higher. But in pseudohyponatremia due to IV Ig therapy serum osmolarity will be higher.[6] Current patient was having low serum osmolarity and high urine sodium and urine osmolarity. Hence a diagnosis of SIADH in GBS has been made and patient was treated with fluid restriction and tolvaptan. Patient responded well and serum sodium improved and maintained within normal range and gradually tolvaptan was stopped before discharge.

CONCLUSION

Post COVID-19 neurological manifestations are reported in large numbers. Post SARS CoV-2 vaccine induced GBS have also been reported in many case series. Neurological symptoms should be addressed with much concern in COVID-19 recovered patient and also in those who receive vaccine. SIADH leading to significant hyponatremia in GBS patient is common and serum sodium should be monitored in GBS patient to prevent permanent neurological deficit.

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