

Case Report

Syndrome of Inappropriate Antidiuretic Hormone (SIADH) in Traumatic Spinal Cord Injury

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Abstract

Hyponatraemia is a known complication associated with neurosurgical conditions including acute spinal injury. The prevalence of hyponatraemia in acute spinal cord injury has been known to be much higher than in the general population. Hyponatraemia is a marker of different underlying diseases and it can be a cause of morbidity itself; this implies the importance of a correct approach to the problem. The syndrome of inappropriate antidiuretic hormone (SIADH) is one of the most common causes of hyponatraemia; it is a disorder of sodium and water balance characterised by urinary dilution impairment and euvolaemic/hypotonic hyponatraemia, in the absence of renal disease or any identifiable non-osmotic stimulus able to induce antidiuretic hormone (ADH) release. It is a diagnosis of exclusion. We are reporting a case of hyponatraemia in a patient with lumbar spinal cord injury who was initially managed as any other hyponatraemia and was later diagnosed as suffering from SIADH.

Key words: SIADH, Hyponatraemia, spinal cord injury.

Introduction:

Hyponatraemia is the most common electrolyte disorder encountered in clinical medicine¹. The prevalence of hyponatremia in spinal cord injury has been known to be much higher than in the general medical or surgical patient population. The prevalence of hyponatraemia in the neurosurgical population has been reported as high as 50%². Uncontrolled hyponatraemia may lead to lethargy, seizures, coma, cardiac arrhythmia and death. Therefore, the complication of hyponatremia should be paid attention after the spinal cord injury. Balance of sodium in blood is complicated and is influenced by many factors. Autonomic nervous system, neuroendocrine dysfunction and haemodynamic changes after spinal cord injury (SCI) play a key role in occurrence of electrolytes abnormality³.

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Cite as:

Chethan. C, Vinay Goyal, Diganta Borah, Rajendra Sharma, Nonica Laisram. Syndrome of Inappropriate Antidiuretic Hormone (SIADH) in Traumatic Spinal Cord Injury, IJPMR, September 2015; Vol 26(3) : 76-8

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Received on 12/08/2013, Accepted on, 11/12/2015

Case Report:

In October 2011, a 25-year-old married female suffered SCI following an accidental fall from roof. She temporally lost consciousness and was immediately shifted to a tertiary care trauma centre, where she was diagnosed to have fracture L1 with traumatic SCI, paraplegia and neurogenic bowel and bladder. She was managed surgically with pedicle screw fixation and was discharged subsequently. On 13/02/2012 she presented to PMR OPD of Safdarjang Hospital with complaints of weakness and loss of sensation in lower limbs, bowel and bladder incontinence and pressure ulcers on both trochanteric and sacral region. Patient was subsequently admitted to rehab ward for comprehensive management.

On examination her cardiorespiratory (PR- 94 beats/minute regular in rhythm, BP - 106/60 mm Hg, RR - 12 breaths/minute, regular) and gastro-intestinal system showed no abnormality. Pallor was present. There were grade IV pressure ulcers on bilateral trochanteric and sacral region; measuring 10 X 10 cm on right, 7 X 5 cm on left trochanter and 18.5 X 9 cm on sacrum. On neurological examination her higher mental functions and cranial nerves were normal, she had complete SCI with AIS – A. Motor, sensory and neurological levels were D11.

Management :

Baseline investigations showed Hb to be 10.8g/dl and serum sodium - 141meq/l. SCI rehabilitation consisting

of two hourly position changes, ROM exercises of bilateral lower limbs and strengthening exercises of bilateral upper limbs, gradual training in tilt table along with daily pressure ulcer dressing was started. Culture and sensitivity of sacral pressure ulcer showed pseudomonas, sensitive to ciprofloxacin, hence she was started on IV ciprofloxacin in view of long standing ulcer with copious discharge, inflamed and indurated surrounding skin.

On twelfth day she developed nausea, vomiting (4 episodes, non-projectile, non-bile stained containing undigested food particles) and weakness. Injection ondansetron 4mg and 75mg injection ranitidine were administered. Her pulse rate was 98 beats/minute regular in rhythm, BP - 96/60 mm Hg. Vomiting stopped but nausea and weakness persisted, hence she was started on IV fluids with 0.9% saline and Ringer lactate (1500 ml IV fluids) and 800 ml of oral fluids. Next morning her serum electrolytes, KFT and LFT were obtained, her serum sodium level was decreased to 122meq/l (Table 1). She was treated with isotonic fluids (2500ml of IV fluids and 1000ml oral intake) and oral sodium chloride in the form of table salt. Along with persistent nausea and weakness she developed headache and dizziness, serum electrolytes showed her serum sodium level had dropped further to 114meq/l along with decrease in serum potassium level to 3.2meq/l. ECG and chest x-ray was normal.

Since there were no signs of dehydration or fluid overload a diagnosis of euvoaemic hyponatraemia was made. The commonest cause of euvoaemic hyponatraemia is SIADH, hence blood and urine analysis were carried out for the same which revealed serum hypoosmolality (240m osmn ol/kg), urine hyperosmolality (510m osnn

ol/kg), increased urine sodium excretion (46meq/L) and decreased blood urea nitrogen 8mg/dl (Table 2). Based on her clinical findings and investigation reports she was diagnosed to have SIADH as per Bartter and Schwartz criteria⁴ (Table 3). She was treated accordingly with fluid restriction 1000ml/day, 3% saline infusion - 2 ml/kg body weight per hour, (0.5mmol/l/hour), increased oral salt intake and intravenous 40mg injection furosemide. Repeat serum electrolytes were done after 12 hours, her serum sodium level had improved to 120meq/l and potassium was 4.1meq/l and her symptoms improved. Further on day 16 her serum sodium increased to 131meq/l and potassium was 4.8meq/l (Table 1) and symptoms disappeared.

Discussion:

Hyponatraemia is frequently found in both acute and chronic SCI. The aetiology of hyponatraemia in SCI patients is multifactorial and includes not only general factors such as the use of diuretics and the intravenous infusion of hypotonic fluids, but also certain mechanisms which operate in the spinal cord injured: decreased renal water excretion due to both intrarenal and arginine vasopressin dependent mechanisms (resetting of the osmostat), coupled with habitually increased fluid intake, and the ingestion of a low salt diet⁵. Frisbie⁶ reported that higher levels of SCI correlate with reduced sodium conservation, hypotension, and hyponatraemia. While Peruzzi *et al*⁷ suggested that the most significant predictor of hyponatraemia is Frankel class A (loss of sensory and motor function below the level of injury) to E (no motor or sensory deficits associated with spine fracture), defined by the neurological classification of SCI, rather than the level of SCI.

Table 1: Blood Investigation Values on Daily Basis

Baseline	Day - 13	Day - 14	Day - 15	Day - 16
Hb: 10.8g/dl	T. BILI: 0.2	T. BILI: 0.2	T. BILI: 0.2	T. BILI: 0.2
TLC: 6500	S. ALP: 302	S. ALP: 288	S. ALP: 288	S. ALP: 270
DC: P66, L30, M2, E2	SGPT: 14	SGPT: 14	SGPT: 14	SGPT: 16
ESR: 25mm/hour	SGOT: 45	SGOT: 42	SGOT: 42	SGOT: 48
Creatinine: 0.3mg/dl	Creatinine: 0.6 mg/dl	Creatinine: 0.5 mg/dl	Creatinine: 0.5 mg/dl	Creatinine: 0.4 mg/dl
Blood urea: 12mg/dl	Blood urea: 12 mg/dl	Blood urea: 14 mg/dl	Blood urea: 16 mg/dl	Blood urea: 14 mg/dl
Sodium: 141 meq/l	Sodium: 122 meq/l	Sodium: 114 meq/l	Sodium: 120 meq/l	Sodium: 131 meq/l
Potassium: 4.9meq/l	Potassium: 3.8 meq/l	Potassium: 3.2 meq/l	Potassium: 4.1meq/l	Potassium: 4.8 meq/l
RBS: 121mg/dl	RBS: 108mg/dl	RBS: 102mg/dl	RBS: 114mg/dl	RBS: 120mg/dl
Total protein: 5.9g/dl			Total protein: 6.1g/dl	
Albumin: 3.2g/dl			Albumin: 3.2g/dl	
Globulin: 2.7g/dl			Globulin: 2.9g/dl	
CRP - 2 mg/l	CRP - 6 mg/l			

Table 2 - Blood and Urine Parameters of Patient on 14th Day

Lab test	Blood / Urine parameter	
	PATIENT	SIADH
Serum sodium	114meq/l	<135meq/l
Serum osmolality	240mosmol/kg	<275mosmol/kg
Urine sodium	46meq	> 20meq/l
Urine osmolality	510mosmol/kg	>100 mosmol/kg
BUN	8mg/dl	<10mg/dl

BUN - Blood urea nitrogen

Hyponatraemia is classified into three types - hypovolaemic hyponatraemia, hypervolaemic hyponatraemia and euvolaemic hyponatraemia. Our patient had euvolaemic hyponatraemia. Commonest cause of euvolaemic hyponatraemia being SIADH and other causes are excessive drinking, renal failure, hypothyroidism, extreme exercising during which over hydration has occurred, low solute intake⁸.

The syndrome of inappropriate antidiuresis (SIADH) is one of the most common causes of hyponatraemia. It is a disorder of sodium and water balance characterised by urinary dilution impairment and hyponatraemia, in the absence of renal disease or any identifiable non-osmotic stimulus capable of inducing antidiuretic hormone (ADH) release. According to its definition, it is diagnosed through an exclusion algorithm. The pathophysiological basis of SIADH is an absolute increase in body water. This increase depends on an excessive water intake that overwhelms the restricted renal ability of diluting urine and mounting compensatory diuresis due to ADH dysregulation⁹. In our case we excluded other causes of euvolaemic hyponatraemia and narrowed on to SIADH as per Bartter and Schwartz criteria and managed appropriately, we attribute multiple factors (antibiotics, pressure ulcers, stress, long term immobilisation and hospitalisation) interacted in initiation of SIADH and increased fluid administration as possible cause of worsening of hyponatraemia. Patient's sodium levels were improved after tapering infusion volume and water intake restriction in this case. Treatment of the case is the same as that for other causes of SIADH, which have suggested that the reduced extracellular volume is important to improve the hyponatraemia after traumatic SCI.

Conclusions:

Hyponatraemia is common in traumatic SCI, especially in cervical injuries. As physiatrists, we deal with SCI patients in our daily practice this situation can be encountered and one should be vigilant in presence of symptoms described above. SIADH is a frequent cause of hyponatraemia; this condition can be misdiagnosed

Table 3- Bartter and Schwartz criteria for SIADH:

1.	Decreased plasma osmolality (<275 mosmol/kg)
2.	Inappropriately concentrated urine (>100 mosmol/kg)
3.	Euvolaemic
4.	Elevated urine Na (>20 meq/l)
5.	Euthyroid, eucortisolemic and no diuretic use.
6.	Chest x-ray and in selected cases, computed tomography (CT) scan of head may be appropriate to reveal an underlying cause.

Supplemental features

Uric acid < 4 mg/dl
BUN < 10 mg/dl
Failure to correct hyponatraemia after NS infusion
Correction of hyponatraemia after fluid restriction

and mismanaged. Early diagnosis and prompt management of SIADH is of paramount importance as it can be fatal.

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