Journal of Acute Disease

journal homepage: www.jadweb.org

doi: 10.4103/2221-6189.236823

Hyponatremia in the acute phase of spinal cord trauma: Review

Luis Rafael Moscote Salazar[1,✉], Amit Agrawal[2], Guru Dutta Satyarthee[3], Huber S. Padilla-Zambrano[4], Boris Vladimir Cabrera-Nanclares[4], Romario Mendoza-Flórez[4], Samer Hoz[5], Alexis Narvaez-Rojas[6]

[1]*Universidad de Cartagena, Cartagena de Indias, Colombia*

[2]*Department of Neurosurgery, Narayna Medical College Hospital, Chinthareddypalem, Nellore-524003, Andhra Pradesh, India*

[3]*All India Institute of Medical Sciences, New Delhi, India*

[4]*School of Medicine. Centro de Investigaciones Biomédicas (CIB). Cartagena Neurotrauma Research Group research line. Faculty of Medicine – University of Cartagena, Cartagena, Colombia*

[5]*Neurosurgery Teaching Hospital, Baghdad, Iraq*

[6]*Universidad Nacional Autonoma de Managua, Nicaragua*

ARTICLE INFO

Article history:
Received 10 April 2018
Revision 20 April 2018
Accepted 25 April 2018
Available 1 May 2018

Keywords:
Hyponatremia
Serum sodium
Spinal cord trauma
Neurotrauma
Injury

ABSTRACT

Hyponatremia is a common electrolyte disturbance usually observed in neurosurgical patients undergoing surgical management of traumatic, as well as, nontraumatic intracranial pathology. The spinal cord trauma is also associated with occasional development of such hyponatremia; it usually occurs within the first two-weeks of the injury. Hyponatremia can lead to alterations of consciousness, convulsions, coma, cardiac arrhythmias and on rare occasions, death. Authors present a practical oriented review of the literature.

1. Introduction

Spinal cord injury represents a series of injuries leading to a compromise of the spine and/or spinal cord. It constitutes one of the main factors of morbidity and mortality, especially in young people all over the world[1]. It is estimated that the incidence is 2.3 per 100 000 events annually in the population, despite of regional variations[2]. It has a bimodal age distribution, with first peak being observed in the age between 15 and 30 years, usually caused by traffic accidents, falls from heights, sports injuries and violence. The other peak is noted in the population of over 65 years old. As there is a higher life expectancy in developed countries, it's been frequently noticed that falls from low heights are the most common causes of vertebral and spinal cord injuries[3,4].

✉First and corresponding author: Luis Rafael Moscote Salazar, Universidad de Cartagena, Cartagena de Indias, Colombia.
E-mail: mineurocirujano@aol.com

How to cite this article: Salazar LRM, Agrawal A, Satyarthee GD, Padilla-Zambrano HS, Cabrera-Nanclares BV, Mendoza-Flórez R, et al. Hyponatremia in the acute phase of spinal cord trauma: Review. J Acute Dis 2018; 7(3): 103-107.

2. Pathophysiology of spinal trauma

The pathophysiological events in the human body following traumatic injury involve the participation of chiefly two mechanisms: the primary injury and secondary injury[2]. The primary injury lesions correspond to the initial traumatic events, and the secondary injury is vascular, ionic, biochemical and inflammatory; these cellular mechanisms occur subsequently after the initial trauma[1,2]. Changes occurring in the body following an injury can be roughly divided into: alteration in the energy metabolism and changes in the substrates, water and electrolytes metabolism, and alteration in local wound metabolism. However, in related multiple injuries, surgeries can cause metabolic, endocrinal and hemodynamic responses[3]. These responses collectively cause alteration characterized by changes in the protein metabolism with negative nitrogen balance, hyperglycemia, sodium retention, and increased lipolysis[2,3].

3. Definition of hyponatremia

Sodium is the most abundant electrolyte in the vascular space with a range of normal sodium level from 135 mmol/L to 145 mmol/L. Hyponatremia is described as a reduction in sodium levels in blood below 136 mmol/L[4]. Acute hyponatremia elevates the chances of cerebral edema leading to an increased intracranial pressure and, in the most severe cases, can cause massive cerebral edema and associated cerebral herniation and seldom if ever, death. In chronic hyponatremia, the brain uses adaptive mechanisms that prevent development of cerebral edema. However, over enthusiastic correction of hyponatremia aimed to reach normal sodium values within a few hours can result in osmotic demyelination syndrome[2,4]. While elevated serum sodium levels are associated with hypertension, Low sodium levels can be related to decreased, normal or elevated tone. Therefore the effective osmolality, is denoted by solutes such as sodium and glucose, which do not passively pass through the cell membranes, promoting certain changes in the transcellular fluid[5].

4. Types of hyponatremia

Hyponatremia is described as the imbalance between body water and the concentration of sodium in the body[4]. The extracellular volume reflects the amount of body sodium, therefore, hyponatremia must be related to extracellular volume [5]. Accordingly, hyponatremia can be classified as hypovolemic, euvolemic, and hypervolemic[4,6]. Sometimes a treating physician encounters a low serum sodium level following administration of some substances such as lipid or Glucose bloodstream in the blood also called pseudohyponatremia or translocational hyponatremia, a non-typical water-sodium imbalance[4,7].

5. Euvolemic hyponatremia (Dilutional)

It is characterized as normal or nearly normal extracellular fluid volume, and total sodium reserve with partial increase of total water as Primary polydipsia has the potential to cause a decrease in sodium levels only when the balance of ingested and eliminated liquids is positive[4]. This decrease can also be related to the abundant intake of liquids in Addison's disease. The postoperative states are related to the increase of antidiuretic hormone and administration of hypotonic liquids after the operation[4,5].

Dilutional hyponatremia is the most usual form of presentation of the entity associated with accumulation of liquids[2]. if the renal excretion is overcome by the intake of liquids, the excess water intake causes dilution of solutes leading to low osmolality and low tonicity[4]. Hypotonia, can predispose to cerebral edema development, a a probable mortal event[6]. The Hypotonic and the hyponatremia may occur even in presence of normal or increased serum osmolality if a considerable amount of solutes has accumulated, it can produce permeability at the membrane level[5]. people with high or even normal blood osmoticity but suffering from hypotonic hyponatremia are predisposed to risks of hypotonicity[7].

Hypovolemic hyponatremia is characterized by total low sodium body content as well as total low body water volume, although, proportionally increased loss in sodium than water causes hypovolemia associated with decrease in both serum osmolality

and blood volume decrease[5]. Common causes are diarrhea, vomiting, peritonitis, diuretics and osmotic diuretic therapy ie glucose, urea, mannitol other causes are cirrhosis of liver and congestive cardiac failure[5,7].

High blood glucose levels has been described as one of the most common etiology for translocational hyponatremia. An increase of 100 mg/dL serum glucose concentration (5.6 mmol per liter) in blood can cause a decrease in serum sodium of approximately 1.7 mmol/L[4]. This results in an increase in serum osmolality of about 2.0 mOsm /Kg of water[6]. The accumulation of mannitol, its related with which renal failure, does the same.

In both contexts the increase in tonicity is worsened by the osmotic diaeresis; whose moderation may result in frank increase of blood sodium, since urinary concentrations are lower than those of[6,7].

Both in hypernatremia and hypothetical hyponatremia is associated with central nervous system dysfunction, however, hypotonic hyponatremia becomes more noticeable when there is a broad and rapid decline in sodium levels[7].

Hyponatremia can clinically present with nausea and vomiting, headache, muscle cramps, lethargy, restlessness and disorientation [1,4]. While most of the patients with a blood sodium level above 125 mmol/l are asymptomatic, however those with lesser values become symptomatic, especially if the sodium levels falls rapidly [6-8].

Complications of severe low sodium levels include convulsions, coma, irreversible brain damage, respiratory failure, brain stem herniation and fatal outcome[4,6,9,10]. It typically occurs with accumulation liquid in essentially euvolemic patients in the postoperative or primary polydipsia); menstruating women seem to be particularly at risk[11].

In pediatric patients with acute hyponatremia, manifestation includes irritability, convulsion, drowsiness, coma and respiratory arrest[4]. Therefore, before a seizure, it is not clearly explainable; a complete electrolytic study is necessary[6]. Rapid correction of serum sodium decreases cerebral water and restores brain function. In practice, it's not easy to distinguish between an acute or chronic hyponatremia, so most clinical studies differ in symptomatic hyponatremia. Comparable to the acute and asymptomatic, and comparable to chronic[6,11].

6. Hyponatremia in spinal cord trauma

Various electrolyte abnormalities can be observed in the patients with spinal cord trauma[9]. Hyponatremia is typically observed within the first two weeks (usually 2-7 d) in patients with post-traumatic cervical lesions[8]. They described six potential causes of hyponatremia in spinal cord trauma: (1) Stimulating action of orthostatic hypotension on the secretion of antidiuretic hormone, which can be altered by the inhibitory effect of hypotonicity [7,8,11]. (2) High uptake of fluids may intervene in production of antidiuretic hormone inducing hyponatremia during the early stages of the spinal cord injury[7,9,11]. (3) Action of a mechanism independent of water maintenance renal antidiuretic hormone in quadriplegic patients, which can be combined with a defect intrarenal excretion of water[7,12]. (4) marked renal elimination of sodium in patients with spinal cord trauma associated with a traumatic brain or carrier of diseases prior to brain injury may develop brain salt loser syndrome[6,8]. (5) The eventual appearance of pseudohyponatremia may occur secondarily to an apparent reduction of sodium levels when the plasma is rich in lipid and protein loss[5,7]. It has been hypothesized that impaired function of the supraspinal pathways that are responsible for sympathetic innervation of the kidney or renal blood flow result in the impaired renal sodium conservation mechanisms in patients with traumatic spinal cord injury[8,9].

It has been observed that hyponatremia is common in the early stage of spinal cord trauma (85%). In the work of Peruzzi *et al* occurred in 29% of persons with spinal cord traumatic injury. It is likely that discrepancies between studies defining the threshold for defining hyponatremia represents some variations[13].

7. Treatment

The presence of symptoms and symptom severity is an important factor, which largely determines the pace of correction of hyponatremia[3]. The presence of hyponatremia in patients with spinal cord trauma is based on physiological events and is no different from management of hyponatremia in patients without severed spinal cords[14,15].

Patients with symptomatic hyponatremia with concentrated urine (osmolality, 200 mOsm per kg of water) and increased or normal clinical volume require the infusion of hypertonic saline solution, seeking to correct serum hyponatremia in a rapid and controlled manner[3,14,15]. Usually, the saline solution is mixed with furosemide in order to avoid the expansion of the extracellular volume that causes the treatment[15].

The treatment of hyponatremia must fulfill four primary objectives: maintainace of proper blood volume, rapid rise in serum sodium if there is acute symptoms, removing excess water if this is the cause and finally, continuously maintaining near normal serum sodium level[14,15].

Patients with low urinary osmolality (<200 mOsm) and hypernatremia with symptoms but with less severe symptoms can be managed with careful monitoring and restricted water intake[13-15]. While if associated with symptoms like convulsions or comas, it requires perfusion with hypertonic saline. An adequate treatment for the management of patients with symptomatic hyponatremia has not yet been determined[8-10]. However, the management of reverting the manifestations of hypotonicity without causing the risk of osmotic demyelination of the development[7,8].

In cases where the is edema or the syndrome of inadequate secretion of antidiuretic hormone, there is defect is water excretion. [6,7]. These asymptomatic cases in the long term can be managed by restriciting the water intake to less than 800 mL per day to obtain a negative water balance[3,11,15].

In severe heart failure, hemodynamic optimization and the use of ACE inhibitor use can increase the excretion of electrolytes and water, causing moderate hyponatremia[7-9]. Thiazide diuretics cannot reduce the concentration of the urine but increases the excretion of electrolyte-free water, allowing attenuating fluid restriction[3,15].

In cases of non-hypotonic hyponatremia, the treatment is mainly aimed at latent disease. Insulin is used to control diabetes, simultaneous the attention should be paid to optimize water, sodium and potassium levels[6,7,15]. Furosemide increases the recovery of patients who absorbed irrigation solutions; in cases of impaired renal function, hemodialysis is used[14]. In cases of syndrome of antidiuretic hormone the use of hypotonic saline is inadequate for correction, because it is not sufficient to raise the serum sodium and will further cause a water retention and exacerbation of hyponatremia[3,14,15].

8. Conclusion

Hyponatremia is a common disorder in patients with spinal cord traumatic injury. Various pathophysiological mechanisms lead to the development of hyponatremia. Strict management of electrolyte imbalance is crucial for the recovery of spine injured patients.

Conflict of interest statement

The authors report no conflict of interest.

References

[1] Bracken MB, Freeman DH Jr, Hellenbrand K. Incidence of acute traumatic spinal cord injury in the United States, 1970-1977. *Am J Epidemiol* 1981; **113**(6): 615-622.

[2] Tator C. *Pathophysiology and pathology of spinal cord injury*. In: Wilkins RH, Rengachary SS, (eds). Neurosurgery. Balt Williams Wilkins; 1996, p. 2847-2859.

[3] Benzel E. *Management of acute spinal cord injury*. In: Wilkins RH, Rengachary SS, (eds). Neurosurgery. Philadelphia: Williams & Wilkins; 1996, p. 2861-2866.

[4] Sica D, Midha M, Zawada E, Stacy WHR. Hyponatremia in spinal cord injury. *J Am Paraplegia Soc* 1990; **13**(4): 78-83.

[5] Sica DA, Culpepper RM. Severe hyponatremia in spinal cord injury. *Am J Med Sci* 1989; **298**(5): 331-333.

[6] Sibley P. Hyponatremia in spinal cord injured persons. *Rehabil Nurs* 1989; **14**(1): 29-30.

[7] Sherlock M, O'Sullivan E, Agha A, Behan L, Owens D, Finucane F, et al. Incidence and pathophysiology of severe hyponatraemia in neurosurgical patients. *Postgr Med J* 2009; **85**(1002): 171-175.

[8] Furlan JC, Fehlings MG. Hyponatremia in the acute stage after traumatic cervical spinal cord injury: clinical and neuroanatomic evidence for autonomic dysfunction. *Spine (Phila Pa 1976)* 2009; **34**(5):

501-511.

[9]Biyani A, Inman CG, el Masry WS. Hyponatraemia after acute spinal injury. *Injury* 1993; **24**(10): 671-673.

[10]Kageyama K, Suda T. A case of hyponatremia after cervical spinal cord injury. *Endocr J* 2011; **58**(5): 369-372.

[11]Mesko T, Garcia O, Yee L, Villar M CH. The syndrome of inappropriate secretion of antidiuretic hormone (SIADH) as a consequence of neck dissection. *J Laryngol Otol* 1997; **111**(5): 449-453.

[12]Soni BM, Vaidyanthan S, Watt JW, Krishnan KR. A retrospective study of hyponatremia in tetraplegic/paraplegic patients with a review of the literature. *Paraplegia* 1994; **32**(9): 597-607.

[13]Peruzzi WT, Shapiro BA, Meyer PR Jr, Krumlovsky F, Seo BW. Hyponatremia in acute spinal cord injury. *Crit Care Med* 1994; **22**(2): 252-825.

[14]Kriz J, Schuck O, Horackova M. Hyponatremia in spinal cord injury patients: new insight into differentiating between the dilution and depletion forms. *Spinal Cord* 2015; **53**(4): 291-296.

[15]Wang H, Hu Y. Analysis of mechanisms and treatment of hyponatremia in acute spinal cord injuries. *Zhongguo Gu Shang* 2012; **25**(4): 306-309.