Original Research Article

A study of etio-pathogenesis of moderate to severe hyponatremia in patients admitted to MICU in a tertiary care hospital, Nandyal

Apsara P.S[1*], B Sarath Kumar Reddy[2], G Vijaya kumar[3]

[1]III Year Post-Graduate, [2]Assistant Professor, [3]Professor and HOD, Department of General Medicine, Santhiram Medical College and General Hospital, Nandyal.
Email:apsara.aps73@gmail.com

Abstract

Background: Hyponatremia is the most common electrolyte disorder in critically ill patients and is a leading cause of morbidity and mortality. Sodium is the principal extracellular cation and the main salt of osmolality. Despite the awareness of hyponatremia for a long time, this common disorder remains an enigma. Because it is associated with a plethora of underlying disease states and multiple etiologies with different pathophysiological mechanisms. **Aim:** To find etiopathogenesis of moderate to severe hyponatremia in patients admitted to MICU. To assess the predominant system involvement of the study population. To assess the outcome of patients among the study group. **Materials and Methods:** A Hospital-based Prospective study was conducted in the Department of Medicine, Santhiram medical college and general hospital for six months period. Patients with >18 years of age with moderate to severe hyponatremia(<125meq/l) in MICU, with informed and written consent taken from the patient. **Results:** The male to female ratio was 1.43:1, with 58% of the patients being males. The mean age was 58.94 ± 16.10 years. Nearly half of the study group had altered sensorium (48%). The commonest system to be involved was the central nervous system (43%).50% of patients were euvolemic. 54% of the patients had severe hyponatremia, with confusion being significantly high in such patients ($p<0.001$). The commonest cause of hyponatremia was SIADH (46%) with infections (Tuberculosis, found in 57.7%) being the predominant cause. The majority (94%) of the patients in the study improved. **Conclusion:** Clinicians need to be aware of the frequent occurrence of hyponatremia, its early identification, and its association with a large variety of diseases. Patients with hyponatremia should be meticulously screened for the presence or absence of tuberculosis. A thorough understanding of the pathophysiological process of hyponatremia and its associated risk factor is of great importance in prompt and effective treatment.
Key Word: hyponatremia.

***Address for Correspondence:**
Dr.Apsara PS,III Year Post-Graduate, Department of General Medicine, Santhiram Medical College and General Hospital, Nandyal.
Email:apsara.aps73@gmail.com
Received Date: 09/11/2019 Revised Date: 25/12/2019 Accepted Date: 13/01/2020
DOI: https://doi.org/10.26611/102113112

Access this article online	
Quick Response Code:	Website: www.medpulse.in
	Accessed Date: 20 January 2020

INTRODUCTION

Sodium is the principal extracellular cation and the main salt of osmolality[1]. The majority of the body's sodium is found in blood plasma and other extracellular fluids, 40% in bone, and the remaining 2%-5% in different cells and organs. This asymmetric distribution of sodium is essential for life and aids in nerve conduction, the passage of various nutrients into the cell, and maintenance of blood pressure. Sodium related disorders (both hyponatremia and hypernatremia) are associated with considerable morbidity and mortality[2]. Hyponatremia is the commonest electrolyte disorder. The occurrence and consequences of hyponatremia increase with age. Hyponatremia that is moderate to severe (plasma sodium 125 and below), and particularly of rapid onset, is often associated with substantial morbidity and mortality[3]. Hyponatremia, which is defined as plasma sodium concentration of less than 135meq/L, occurs primarily

How to cite this article: Apsara PS, B Sarath Kumar Reddy, G Vijaya kumar. A study of etio-pathogenesis of moderate to severe hyponatremia in patients admitted to MICU in a tertiary care hospital, Nandyal. *MedPulse International Journal of Medicine.* January 2020; 13(1): 49-52. https://www.medpulse.in/Medicine/

due to imbalance in water homeostasis, antidiuretic hormone (ADH) regulation, and renal handling of filtered sodium. The two most common causes are effective circulating volume depletion causing the non-osmotic release of ADH and the syndrome of inappropriate ADH secretion, disorders in which ADH secretion is not suppressed despite a decrease in plasma osmolality[4]. Alterations in water and sodium balance can affect the central nervous system. Some of these patients with clinically significant hyponatremia may present with non-specific or neurological symptoms due to cerebral edema[5]. Patients with serum sodium concentration greater than 125 mEq/l are usually asymptomatic. In contrast, those in whom these values are lower may have symptoms that include headache, nausea, vomiting, muscle cramps, lethargy, restlessness, disorientation, and depressed reflexes. Severe and rapidly evolving hyponatremia may present with seizures, coma, permanent brain damage, respiratory arrest, brainstem herniation, and death[6]. The Emergency Department physician should have an increased index of suspicion of hyponatremia in patients with pneumonia, active tuberculosis, pulmonary abscess; neoplasm; asthma, or in patients with CNS infection, trauma, or neoplasm. The patient's medication list should be examined for drugs known to cause hyponatremia.

AIM AND OBJECTIVES

- To find etiopathogenesis of moderate to severe hyponatremia in patients admitted to MICU.
- To assess the predominant system involvement of the study population.
- To assess the outcome of patients among the study group.

MATERIAL AND METHODS

A Hospital-based Prospective study was conducted in the Department of General Medicine, Santhiram Medical College, and General Hospital for three months after taking approval from the Hospital Ethics and Research Committee. Sampling Technique and Sample Size: Universal Sampling Technique was used for the selection of study subjects. All the patients coming to the medicine department during the study period and fulfilling the inclusion criteria were taken for study after taking prior informed consent. The patients included in the study were from MICU. The final sample size came to be 50 subjects of moderate to severe hyponatremia (<125meq/l).

Inclusion Criteria

- Patients aged >18 years.
- Patients with moderate to severe hyponatremia (serum sodium < 125mmol/L) admitted to MICU.
- Patients with informed written consent taken.

Exclusion criteria

- Patients with hyperglycemia, hyperlipidemia, and paraproteinemias.
- Patients receiving mannitol.
- Patients without informed consent

Data Analysis

Association between qualitative variables was done with the help of the Chisquare
test. P-value <0.05 was taken as significant. Quantitative data was represented
using mean ± SD and median and IQR (Interquartile range).

RESULTS

In our study of 50 patients admitted with moderate to severe hyponatremia, slight male preponderance was noted, with 58% of patients being males. The male to female ratio was 1.38:1. In this study, most of the patients were aged 61 to 70 years (15%). The mean age of the population under study was 58.94 ± 16.10 years. In the present study, out of 50 adult patients, 52% of the patients were conscious, and 48% of the patients presented with altered sensorium.

Table 1: Distribution of patients as per Serum Na^+ Level (meq/L)

Serum sodium	Distribution (n=50)	
	No.	%
< 120	27	54.00
120 to 125	23	46.00
Total	**50**	**100.00**

In this study serum sodium levels were < 120 meq/L (severe hyponatremia) in 54%of the patients, and 46% of the patients had serum sodium levels between 120 to 125 meq/L (moderate hyponatremia).

Table 2. Presenting complaints of the study population

In the present study, the commonest presenting complaint was vomiting (28%), followed by confusion (26%), seizure (10%), and coma (4%). Among comorbid conditions, a history of hypertension and diabetes mellitus was noted in 49% and 29%, respectively. 32% individuals were asymptomatic in the present study.

Table 2: Presenting complaints of the study population

Chief complaints	Distribution(n=50) No	%
Vomiting	14	28.00
Confusion	13	26.00
Seizure	5	10.00
Coma	2	4.00
Asymptomatic	16	32%

Table 3: Distribution of patients according to Type of hyponatremia

Type of hyponatremia	Distribution (n=50) No.	%
Euvolemic hypoosmolar	25	50.00
Hypervolemic hypoosmolar	15	30.00
Hypovolemic hypoosmolar	9	18.00
Hypervolemic iso-osmolar	1	2.00
Total	**50**	**100.00**

In the present study, 50% of the patients had euvolemic hypoosmolar hyponatremia. In the remaining, 30% had hypervolemic hypoosmolar, 18% had hypovolemic hypoosmolar, and 2% had hypervolemic iso-osmolar hyponatremia.

Table 4: System involvement of the study population

Cause	Distribution (n=50) No	%
CNS	22	44.00
Abdominal	11	22.00
Renal	7	14.00
CVS	6	12.00
Respiratory	4	8.00

In this study of moderate to severe hyponatremia, CNS symptoms were present among 44% of the patients.

Table 5: Distribution of patients based on the cause of hyponatremia

Cause	Distribution (n=50) No	%
SIADH	23	46.00
Renal	6	12.00
GI loss	6	12.00
Cirrhosis	5	10.00
Drugs	5	10.00
Cardiac	5	10.00
Total	**50**	**100.00**

In the present study, SIADH was the commonest cause of hyponatremia noted in nearly half of the study population (46%). The other causes were renal loss (12%), gastrointestinal loss (12%), while cirrhosis of the liver, drug induced, and cardiac causes constitute 10% each. Similarly, in the present study, infections 13 (56.52%) were the predominant cause of SIADH followed by disorders of CNS 6 (26.08%). These disorders of CNS included cerebrovascular accidents and Guillain-Barre syndrome. There was 1 patient (4.35%) with Neoplasm as the cause of SIADH, while in 3 patients(13.04%), no cause could be found and were thus classified as idiopathic.8 (61.54%) of patients having an infectious etiology as the cause of SIADH had tuberculosis, 4(50%) of them had tubercular meningitis,2(25%) had pulmonary tuberculosis, while 1(12.5%) had miliary tuberculosis and tuberculomas each. Other infections included viral meningoencephalitis, bacterial meningitis, neurocysticercosis, cerebral malaria, and pneumonia. In the present study majority (94%) of the patients improved, and mortality was noted in 6% of the patients, this finding is consistent with another study by Joseph and Paniker where mortality was observed in 7% of patients. There was a positive association among SIADH and euvolemic hypoosmolar hyponatremia ($p<0.001$) and high urine sodium ($\geq$ 25mM) ($p<0.001$). Also, confusion was noted in a significantly higher number of patients with severe hyponatremia ($p<0.001$). No significant association of severity was found with gender ($p=0.642$), age ($p=0.098$), causes of hyponatremia ($p=0.328$), and type of hyponatremia ($p=0.325$) in thisstudy.

DISCUSSION

The clinical presentations of hyponatremia can range from mild non-specific symptoms such as nausea, headache, and lethargy, to severe symptoms like seizures and coma. In a study by Manish Patni *et al*[7]. from Nagpur, Maharashtra, similar findings were reported with drowsiness as the commonest symptom present in 51% of the cases, 14% of patients had vomiting while 6% patients had seizures. Recently, a descriptive study by Rao MY *et al*[8]. from Bangalore, Karnataka, reported 76% of patients with CNS symptoms, which was similar to our study. In Rao MY *et al*. 61% were euvolemic, 23% were overloaded, and 16% dehydrated, and the commonest type of hyponatremia noted was euvolemic hypo-osmolar hyponatremia. Chung and Hubbard[9] reported that 11% of patients with active TB (pulmonary or non-pulmonary) were affected with hyponatremia, and it is apparent that the main cause of serum sodium depletion in these patients is SIADH. Vorherr *et al*[10]. have reported a case with Pulmonary TB and hyponatremia and found antidiuretic agents in tuberculous lung tissues. Cockcroft *et al*[11]. reported a 74-year-old woman with miliary tuberculosis, which was complicated by severe hyponatremia due to SIADH. In one of the first reports, Weiss *et al*[12]. reported hyponatremia resulting from SIADH in patients with Pulmonary TB. Then it was declared that an increased ADH level in the presence of hyponatremia in Pulmonary TB cases is an indicator for ectopic ADH production. Few studies demonstrated that the ADH level was not detectable following full anti-TB therapy[13,14]. There are many reports of SIADH associated with pulmonary, miliary, and central nervous system-related TB. More than 60% of the patients with tubercular meningitis may present with hyponatremia or SIADH as the first presentation[15].

CONCLUSION

Based on the results of this study, it may be concluded that hyponatremia can present with many clinical

manifestations. The majority of patients report CNS symptoms, and these patients are likely to have euvolemic hypoosmolar hyponatremia with SIADH as the predominant cause. In our country, where tuberculosis is common and remains latent can often present as symptomatic hyponatremia. Tuberculosis can cause hyponatremia in several ways, like a local invasion of adrenal glands or pituitary gland, tubercular meningitis, and SIADH (via pulmonary infection). Therefore, all patients with hyponatremia should be compulsorily screened for the presence or absence of tuberculosis. Thus, to conclude, a thorough understanding of the pathophysiological process of hyponatremia and its associated risk factors.

REFERENCES

1. LF, Palevsky PMLevin ER, Gardner DG, Samson WK. Natriuretic peptides. N Engl J Med 1998;339(5):321–8.
2. Fried . Hyponatremia and Hypernatremia. Medical Clinics of North America 1997;81:585-609.
3. Anderson RJ. Hospital-acquired Hyponatremia. Kidney International 1986; 29:1237-47.
4. Fall PJ. Hyponatraemia and hypernatraemia: A systematic approach to causes and their correction. Postgrad Med 2000;107(5):75-82.
5. Nurko S, Freda BJ. Hyponatraemia and hypernatraemia. Available from: URL: http://www.clevelandclinicmeded.com/medicalpubs/diseasemanagement/nephrology/hyponatremia-and hypernatremia/Default.htm Access Date: 18.06.2015.
6. Craig S. Hyponatraemia. Available from: URL: http:/www.emedicine com/emerg/topic275.htm. Access date: 28.06.2015.
7. Patni M, Humaney NR, Jeswani J, Deoke SA. Clinico-Etiological Profile of Hyponatremia in Patients Admitted in Intensive Care Unit of Tertiary Health Care Rural Center. JMSCR 2015;03(08):6975-82.
8. Rao MY, Sudhir U, Anil Kumar T, Saravanan S, Mahesh E, Punith K. Hospital-Based Descriptive Study of Symptomatic Hyponatremia in Elderly Patients. JAPI 2010;58:667-9.
9. Chung DK, Hubbard WW. Hyponatremia in untreated active pulmonary tuberculosis. Am Rev Respir Dis 1969;99(4)(4):595-7.
10. Vorherr H, Massry SG, Fallet R, Kaplan L, Kleeman CR. Antidiuretic principle in tuberculous lung tissue of a patient with pulmonary tuberculosis and hyponatremia. Ann Intern Med 1970;72(3):383-7.
11. Cockcroft DW, Donevan RE, Copland GM, Ibbott JW. Miliary tuberculosis presenting with hyponatremia and thrombocytopenia. Can Med Assoc J 1976;115(9):871-3.
12. Weiss H, Katz S. Hyponatremia resulting from apparently inappropriate secretion of antidiuretic hormone in patients with pulmonary tuberculosis. Am Rev Respir Dis 1965;92(4):609-16.
13. Lee P, Ho KK. Hyponatremia in pulmonary TB: evidence of ectopic antidiuretic hormone production. Chest 2010;137(1):207-8.
14. Hill AR, Uribarri J, Mann J, Berl T. Altered water metabolism in tuberculosis: role of vasopressin. Am J Med 1990;88(4):357-64.
15. Singh BS, Patwari AK, Deb M. Serum sodium and osmolal changes in tuberculous meningitis. Indian Pediatr 1994;31(11):1345-50.

Source of Support: None Declared
Conflict of Interest: None Declared