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HOSPITAL BASED DESCRIPTIVE STUDY OF HYPONATREMIA IN CRITICALLY ILL PATIENTS

Medicine

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ABSTRACT

Hyponatremia is the most commonly encountered electrolyte abnormality in a hospital setting. Recent studies on hyponatremia have been limited to the patients with severe hyponatremia and there has been no consensus on optimal management of hyponatremia. High mortality among the patients of hyponatremia is secondary to the underlying medical condition, rather than the degree of hyponatremia. This study was conducted at Medical Intensive Care Unit at D Y Patil University School of Medicine Hospital, Mumbai of 100 patients. Successive patients of hyponatremia who were admitted to the Intensive Care Unit in the hospital were included in the study. These patients were evaluated for the underlying cause of hyponatremia, which included detailed history and physical examination followed by appropriate laboratory investigations based on the serum osmolality. Patients were followed up till the hyponatremia was corrected or patients were discharged from the hospital.

KEYWORDS:

Hyponatremia, Critically ill, Sodium, Electrolytes

INTRODUCTION

Hyponatremia is defined as a plasma [Na+] <135 meq/L.

Disorders of sodium and water metabolism are common in hospitalized patients and are occasionally encountered in outpatients. Both hyponatremia and hypernatremia can cause substantial morbidity and mortality, and ironically, incorrect treatment can add to the problem[1] . Serum sodium concentration and serum osmolarity normally are maintained under precise control by homeostatic mechanisms involving thirst, antidiuretic hormone (ADH), and renal handling of filtered sodium. Clinically significant hyponatremia is relatively uncommon in its presentation[2] .About 40% of the body's sodium is contained in bone. Approximately 2-5% occurs within organs and cells and the remaining 55% is in blood plasma and other extra cellular fluids. The amount of sodium in blood plasma is typically 140 mmol/L, a much higher amount than is found in intracellular sodium (about 5 mmol/L). This asymmetric distribution of sodium ions is essential for human life. It makes possible proper nerve conduction, the passage of various nutrients into cells, and the maintenance of blood pressure[3].

Management of abnormalities in water homeostasis is frequently challenging. Because age-related changes and chronic diseases are often associated with impairment of water metabolism in elderly patients, it is absolutely essential to be aware of the pathophysiology of hyponatremia. The sensation of thirst, renal function, concentrating abilities and hormonal modulators of salt and water balance are often impaired in critically ill patients, which makes such patients highly susceptible to morbid and iatrogenic events involving salt and water.

A systematic approach in evaluating water and sodium problems, utilizing a comprehensive history and physical examination, and a few directed laboratory tests to make the clinical diagnosis. Furthermore, clinicians should have a clear appreciation of the roles that iatrogenic interventions and lapses in nutrition and nursing care frequently play in upsetting the homeostatic balance in the critically ill patients.

AIMS & OBJECTIVES

1. To assess the incidence of hyponatremia in patients in Intensive Care Unit
2. To describe the etiological factors responsible for hyponatremia
3. To describe clinical manifestations of hyponatremia
4. To describe treatment modalities for hyponatremia in ICU patients

MATERIALS & METHODS

The present study was conducted in patients admitted in the Intensive Care Unit of D. Y. Patil School of Medicine over a period of one year.

1. DIAGNOSIS OF HYPONATREMIA :

- Hyponatremia : <130 mEq/L
- Severe hyponatremia : <125 mEq/L

2. INCLUSION CRITERIA :

- Patients admitted to the ICU with serum sodium levels less than or equal to 130 mEq/L
- Age of the patients > 18 yrs

3. EXCLUSION CRITERIA :

- Age of the patients < 18 yrs
- Patients admitted to the ICU with serum sodium levels more than 130 mEq/L
- Post operative patients

4. SAMPLE SIZE : 100 cases

RESULTS & ANALYSIS

Among the 100 subjects studied 59% were males and 41% were females (Table 5 and

Figure 2). The male to female ratio was 1.43:1

TABLE 1

Gender Distribution	Count	Percent
Female	41	41
Male	59	59

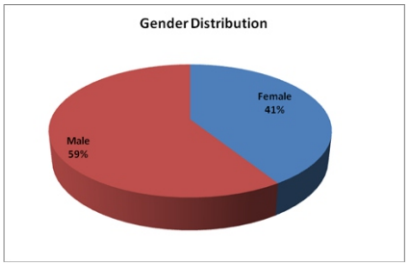

TABLE 2

Age Group	No.
20-30	6
31-40	8
41-50	17
51-60	22
61-70	20
71-80	21
>80	6
Total	100

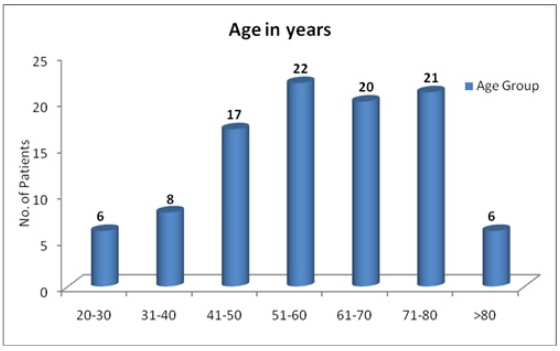

TABLE 3

Age in years	Male		Female		Total	
	Count	Percent	Count	Percent	Count	Percent
20-30	4	6.8	2	4.9	6	6
31-40	6	10.2	2	4.9	8	8
41-50	12	20.3	5	12.2	17	17
51-60	12	20.3	10	24.4	22	22
61-70	14	23.7	6	14.6	20	20
71-80	11	18.6	10	24.4	21	21
>80	0	0.0	6	14.6	6	6
Total	59	100	41	100	100	100
Mean ±S.D	56.58±15.45		62.19±16.83		58.88±16.27	

TABLE 4

Sodium	Frequency	Percent
<=120	72	72
121-125	22	22
>=126	6	6
Total	100	100

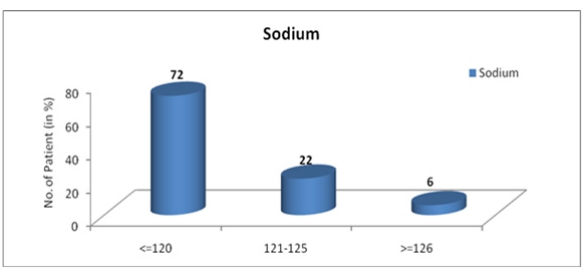

TABLE 5

Symptoms	Absent	Present	Total
NAUSEA	66	34	100
VOMITING	50	50	100
HEADACHE	83	17	100
FATIGUE	37	63	100
IRRITABILITY	41	59	100
SEIZURES	77	23	100
CRAMPS	96	4	100
DROWSY	43	57	100
DIARRHOEA	90	10	100

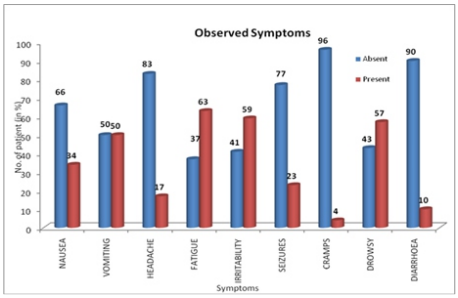

TABLE 6

Sensorium	Frequency	Percent
Conscious	34	34
Disoriented	13	13
Drowsy	34	34
Restless	2	2
Unconscious	17	17
Grand Total	100	100

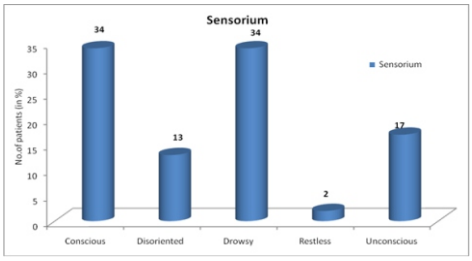

TABLE 7

Hydration	Frequency	Percent
Euvolemia/Isovolemia	65	65
Hypervolemia	21	21
Hypovolemia	14	14
Total	100	100

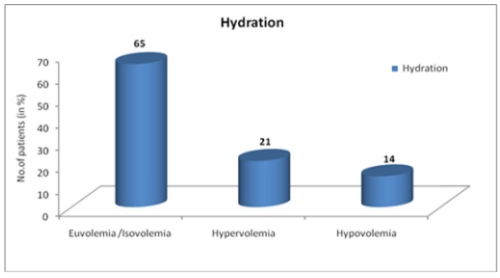

TABLE 8

Outcome	Frequency	Percent
DEATH	19	19
DISCHARGE	81	81
Total	100	100

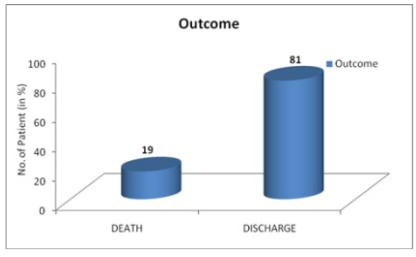

TABLE 9

Hydration		Death	Ratio	Discharge	Ratio
Euvolumia	65	12	65:12	53	65:53
Hypervolemia	21	5	21:05	16	21:16
Hypovolemia	14	2	7:01	12	7:06

TABLE 10

DIAGNOSIS	Frequency	Percent
ALD	7	7
CEREBRAL MALARIA	15	15
CONGESTIVE CARDIAC FAILURE	8	8
CHRONIC RENAL FAILURE	7	7
DIURETICS	9	9
HYPOTHYROIDISM	12	12
PNEUMONIA	14	14
SEPSIS	13	13
SYNDROME OF INAPPROPRIATE ADH SECRETION	10	10
VOMITING	5	5
Total	100	100

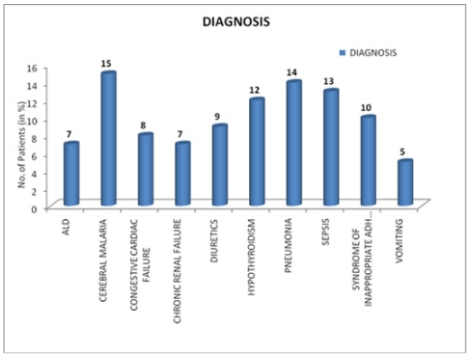

TABLE 11

Sodium	Number	DEATH	Ratio	DISCHARGE	Ratio
<=120	72	12	06:01	60	06:05
>=126	22	1	22:01	5	22:05
121-125	6	6	01:01	16	03:08
Total	100	19		81	

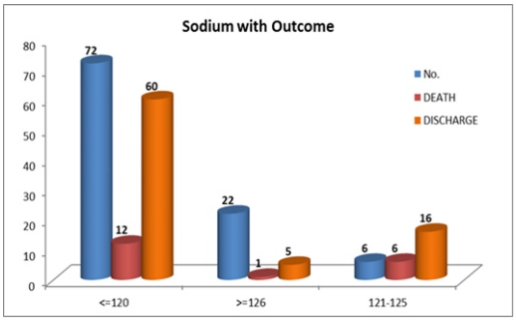

TABLE 12

Row Labels	Number	Below normal (U. Na)	Ratio	Normal (U. Na.)	Ratio
<=120	72	24	3	48	03:02
>=126	22	3	22:03	3	22:03
121-125	6	11	06:11	11	06:11
Total	100	38		62	

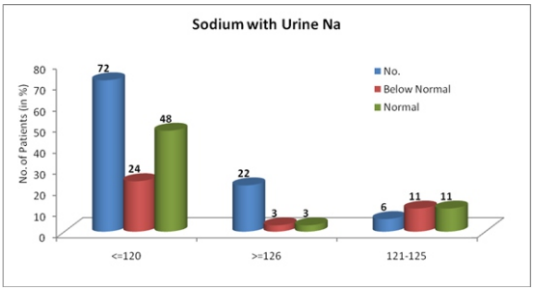

TABLE 13

Parameters	Na			Total
	Mild (>=126)	Moderate (121-125)	Severe (<=120)	
	n=6	n=22	n=72	n=100
S. OSM	266±14.44	256.77±10.69	246.63±41.57	250.02±36.17
U. OSM	318.67±120.33	283.77±148.77	319.44±131.34	311.55±134.23
U. NA	56.33±36.74	71.23±51.52	72.52±39.28	71.26±41.86

Hyponatremia is the most common electrolyte disturbance seen in hospital practice. It is more common in the elderly patients with multiple medical comorbidities[4]. Hyponatremia has been associated with considerable morbidity and mortality in many chronic diseases, most notably in patients with congestive heart failure[5] and cirrhosis of liver. Hyponatremia also leads to increased health care cost and the majority of these costs are attributable to the incremental resource utilization for patients who were not admitted specifically for hyponatremia, but whose hospitalization was prolonged due to hyponatremia.

This study had included 100 critically ill patients admitted to ICU having hyponatremia. In previous studies incidence of hyponatremia in hospitalized patients was found to be about 1% to 6%. Incidence of hyponatremia has been shown to have direct correlation with age[4]. In our study 63% of the patients were 50 to 80 years old. 5-year retrospective study of 2,188 patients, Bennani et al found the incidence of hyponatremia to be 14% at the time of admission to the intensive care unit while DeVita et al found the incidence of hyponatremia in ICU to be 29.6%. Multiple comorbidities like hypertension and Diabetes Mellitus are present in this age group, treatment of which predisposes a patient to hyponatremia. Use of diuretics is also more common among the elderly patients, which has been a major cause of hyponatremia in hospitalized patients. Hawkins et al noted that increasing age, after adjusting for sex, was independently associated with both hyponatremia at presentation and hospital-acquired hyponatremia[4].In the present study prevalence of hyponatremia was more in male patients with male: female ratio of 1.4:1 (59 males and 41 females).

There was difficulty in assessing the exact duration of hyponatremia as the data of serum sodium prior to the detection of hyponatremia was not available for most patients in this study. In this study 66% of the patients had manifestations of hyponatremia at presentation. A major proportion (34%) of the patients in the study did not have evident clinical manifestations of hyponatremia. This can be possibly due to the reason that acute hyponatremia (hyponatremia of <48 hr duration) is less frequent than chronic hyponatremia (>48 hr duration) in which symptoms are ameliorated by the phenomenon of cerebral adaptation to hyponatremic state.

In study of severe hyponatremia in Queen's Medical Centre, UK, by Clayton et al, 36.2% patients had neurological symptoms attributable to the hyponatremia at presentation.[6] In another study by Nzerue et al on outcome of hyponatremia in hospitalized patients 52.9% patients had neurological manifestations[7]. Major clinical manifestations of hyponatremia were fatigue (63%) and irritability (59%). In our study, 66% patients had neurological manifestations. These include seizures, reduced consciousness level, confusion, unsteadiness and falls. The major pre-existing illnesses present among the patients in our study were hypertension, diabetes mellitus, chronic kidney disease, heart failure and chronic liver diseases and hypothyroidism. In our study hypertension was a major risk factor for hyponatremia due to diuretic use in elderly patients. In our study, cerebral malaria was the most common cause of malaria. 15 (15%) patients of the 100 patients had cerebral malaria. Holst et al noticed in their study that serum sodium levels were low in 13 of 17 patients with malaria upon admission and returned to normal levels during antiparasitic therapy[8].

In our study 7 (7%) of the patients had hyponatremia due to renal disorders out of which 6 patients had pre-existing renal disease, one

patient had acute renal failure. Total 7 (7%) patients had liver disorder and another 8 (8%) patients had hyponatremia due to heart failure. These patients were admitted to the hospital due to non-compliance with treatment and inappropriate fluid intake leading to volume expansion and dilutional hyponatremia. In majority of these patients loop diuretics and fluid restriction was sufficient to correct hyponatremia. In study by Saeed et al, 37 % of the patients had hyponatremia due to these disorders (renal disorders 21%, liver disorders 7% and CHF 9%)[9].

12 per cent patients in our study had pre-existing hypothyroidism. In study by Clayton et al hypothyroidism induced hyponatremia occurred in 3.7%.[6] Diuretic use were 9 percent of the cause in our study, present in 9 of the total patients. Thiazide diuretics are a common cause of severe hyponatraemia[4]. Up to a third of elderly patients taking a thiazide at hospital admission are hyponatremic and 14% of patients prescribed a thiazide diuretic in primary care have a sodium below the normal range. In a study by Huda et al, 14 out of 22 (63.6%) patients of hyponatremia on diuretics were taking thiazide diuretics[10].

Our hyponatremic patients who were taking a loop diuretic had at least one other cause for the hyponatremia in the form of liver disease, heart failure, vomiting. Loop diuretics are frequently used to treat conditions such as congestive cardiac failure and cirrhosis in which hyponatremia occurs due to hypervolemia, while thiazide diuretics are being prescribed routinely for management of primary hypertension especially in the elderly age group.

Vomiting is one of the strongest known stimuli for ADH release[11]. 5 % of the total patients had vomiting. Vomiting in our patients was associated with infective illnesses (gastroenteritis, enteric fever, viral fever), systemic illnesses (acute and chronic liver diseases, renal disorder) gastrointestinal obstruction , drug induced and increased ICP . It was also associated with poor intake and diarrhea. Poor nutritional intake secondary to various other co morbidities was a major risk factor in this study. Total 49 (49%) patients had history of poor intake which was associated with various other causes. In the available literature none of the studies have considered vomiting and poor intake as an etiology or risk factor for hyponatremia. Thus our study highlights the importance of eliciting history of poor nutritional intake and vomiting in patients of hyponatremia.

Ten (10%) patients in present study fulfilled the diagnostic criteria for SIADH. The incidence is comparable to the available literature on hyponatremia in hospitalized patients. In study by Saeed et al incidence of SIADH among hyponatremia patients was 14.03% while in study by Huda et al it was 19.8%[9,10]. Other causes were pneumonia (14%), sepsis (13%).

Treatment of hyponatremia in our study was decided by the severity of hyponatremia, presence of symptoms and the underlying disorders. Patients with clinical evidence of dehydration as in patients with vomiting, diarrhea and febrile illnesses and no neurological symptoms of hyponatremia were treated with normal saline infusion. Patients with dilutional hyponatremia were treated with fluid restriction (intake equal to the urine output in previous 24 h) and/or loop diuretics for promoting excretion of free water. Patients with SIADH were treated with fluid restriction and loop diuretics along with oral sodium chloride supplementation for free water excretion. All other patients with moderate or asymptomatic hyponatremia were treated with oral sodium chloride supplementation.

In our study 14% of the patients received normal saline, 21% of the patients were on fluid restriction, 65% patients were given oral sodium chloride supplementations. There are considerable differences in the treatment strategies for hyponatremia in recent studies on hyponatremia in hospitalized patients. In study by Hoorn et al on severe hyponatremia in hospitalized patients, 29% patients were given normal saline, 9% patients were advised fluid restriction, 10% patients received oral sodium chloride supplementation and 19% patients received no therapy for hyponatremia whereas in study by Nzerue et al 82% of the patients received normal saline, 9% patient were given fluid restriction while 6% patients were treated with other treatment modalities such as withdrawal of drug causing hyponatremia[71].

In our study hypertonic (3%) saline was used for the treatment of severe symptomatic hyponatremia. Twelve patients (12%) were given hypertonic saline infusion. The aim was gradual correction of hyponatremia with increase of serum sodium by 8 meq/L in 24 hours. The level of correction was usually within the recommended level in most patients. In study by Nzerue et al 3% patients received hypertonic saline while in study by Hoorn et al 5% of the patients received hypertonic saline[12]. Therefore the use of hypertonic saline in the present study has been limited to 3% saline and it commensurates with the available literature.

The incidence of osmotic demyelination syndrome (ODS) following treatment of hyponatremia has been very rare. There was no such complication in our study. The mortality in patients with severe hyponatremia has been found to be between 20 to 27% in literature[10]. The overall mortality among patients of hyponatremia in our study was 19%. Mortality was not directly related to hyponatremia but to the severity of the underlying medical condition in the patients. In 2005, Huda et al in their study found that there was 27% mortality among patients of severe hyponatremia[10]; however mortality among these patients was not directly related to hyponatremia but to other concomitant severe comorbidity. Papadakis et al had found that hyponatremia is an independent risk factor for mortality in patients with cirrhosis[13].

CONCLUSION

Hyponatremia is a common electrolyte abnormality found in hospitalized patients in general medical and surgical wards. It is more common in elderly patients and critically ill patients admitted to the ICU. Hypertension and Diabetes Mellitus as pre existing comorbidity was present in majority of patients and it predisposed the patients to hyponatremia. Thiazide diuretics were the single most important etiology of hyponatremia. Vomiting and poor intake were also significant cause of hyponatremia in this study. Other major causes of hyponatremia were renal disorders, SIADH, CHF, and chronic liver disease. Hyponatremia was found to be related to multiple etiological factors in a large number of patients.

Treatment of hyponatremia with hypertonic saline should be restricted to the patients with severe hyponatremia and those with neurological symptoms of hyponatremia. Treatment with hypertonic saline is safe provided gradual correction of hyponatremia is followed. Osmotic demyelination syndrome is a rare complication related to the treatment of hyponatremia and should be suspected in a case of hyponatremia who develop fresh neurological deficits while on treatment or after treatment with hypertonic saline.

Severe hyponatremia is associated with considerable mortality in patients with underlying medical diseases as advanced cirrhosis.
A systematic approach to the diagnosis of hyponatremia with the application of simple diagnostic algorithms, using history, clinical examination and laboratory findings to establish mechanism of hyponatremia can significantly improve the assessment and management of hyponatremia.

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