Int J Pharm Bio Sci 2016 April; 7(2): (B) 434 - 440

Original Research Article Biochemistry

International Journal of Pharma and Bio Sciences

ISSN 0975-6299

STUDY OF SYMPTOMATIC HYPONATREMIA IN ELDERLY PATIENTS

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ABSTRACT

Hyponatremia is one of the commonest electrolyte disturbances encountered in medical wards and contributes to substantial morbidity and mortality. Therefore, this study was taken up to explore the clinical profile of hyponatremia. This cross sectional study was conducted in a tertiary care hospital with 100 patients. History and clinical examinations were recorded in all patients at admission. History specially included compulsive water drinking and intake of diuretics. Complete hemogram, measurement of fasting blood glucose, urea, and creatinine, lipid profile, {thyroid function tests morning serum cortisol (optional)}, and serum electrolytes were done in every patient. This was followed by a plasma and urinary osmolality determination as well as urinary sodium estimation. In our study total incidence of hyponatremia was 14.3%incidence of symptomatic hyponatremia were 5%,out of 100 patients 24 patients had SIADH(24%) which was the commonest cause of hyponatremia. SIADH and euvolumic hyponatremia formed the largest subgroup in the study. Drugs, especially diuretics such as furosemide and hydochlorothiazide, have also contributed fairly for the cause of hyponatremia these results correlated significantly with other studies

KEY WORDS: Hyponatremia, Diuretics, hypovolemia,SIADH-syndrome of inappropriate antidiuretic hormone

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INTRODUCTION

Hyponatremia is one of the treatable causes of electrolyte disorder with neuropsychatric manifestations. Early diagnosis and prompt treatment will save the patient from fatal complications. Hyponatremia is defined as a serum sodium concentration (Na^+) less than 135mEq/L . Disorders of serum sodium concentration are caused by abnormalities in water homeostasis that lead to changes in the relative ratio of sodium to body water. Serum sodium levels and serum osmolality are maintained under precise control by homeostatic mechanisms involving thirst, anti - diuretic hormone and the renal handling of filtered sodium. The clinical features are developed due to swelling of the brain tissue as hyponatremia induces hypotonicity in the circulation. The majority of the clinical manifestations of hyponatremia are neuropsychiatric, including lethargy, psychosis, and seizures, designated as hyponatremic encephalopathy. This study was done in a tertiary care hospital to find out the etiology and incidence of hyponatremia in elderly patients.

AIM

1.To study the incidence of symptomatic hyponatremia in elderly hospitalized in medical wards and ICU.
2.To determine the etiology of randomly selected 100 symptomatic hyponatremic elderly patients .
3. To explore the clinical profile of the patients with hyponatremia
This study was conducted in a tertiary care centre during the period of July 2014 to July 2015. Total number of people included in this study was 100.This study is a cross-sectional study.

INCLUSION CRITERIA

Patients above age 65 years.
Symptomatic patients with serum sodium level less than 135.

EXCLUSION CRITERIA

Asymptomatic patients.
Age below 65 years.
Hypercholesterolemia
Patients treated with mannitol.

RESULTS

The number of patients with hyponatremia less than 135 mmol /L were about 100 patients
The number of patients admitted with symptomatic hyponatremia was about 35 patients.
The number of patients with severe hyponatremia with serum sodium less than 120 mmol/L were 25 patients .

Figure 1
35% were symptomatic with varied manifestations of hyponatremia.
65% of admitted patients with hyponatremia were men and 35 % were women

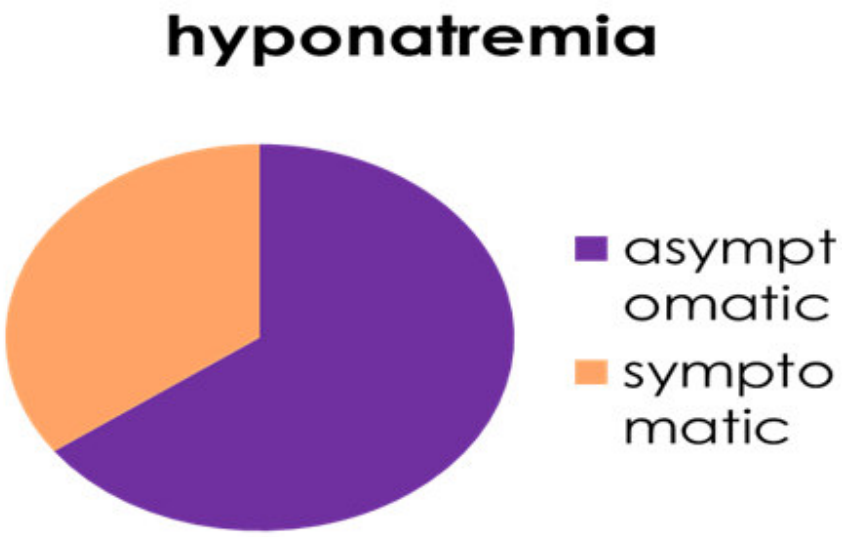
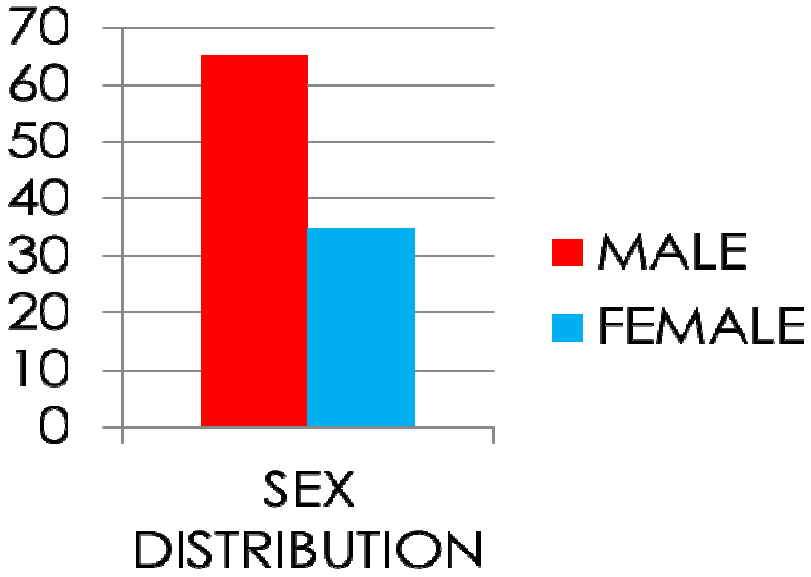


Figure 2
Most of the patients were between 65 to 80 yr of age.



24 patients were between 65-69 yr of age
24 patients were between 70-74 yr of age
24 patients were between 75-79 yr of age
14 were between 80-84 yr of age
11 were between 85-89 yr of age
2 were between 90-95 yr of age

Figure 3
Patients with hyponatremia were admitted due to a variety of causes.The common causes were SIADH and prolonged thiazide diuretic intake

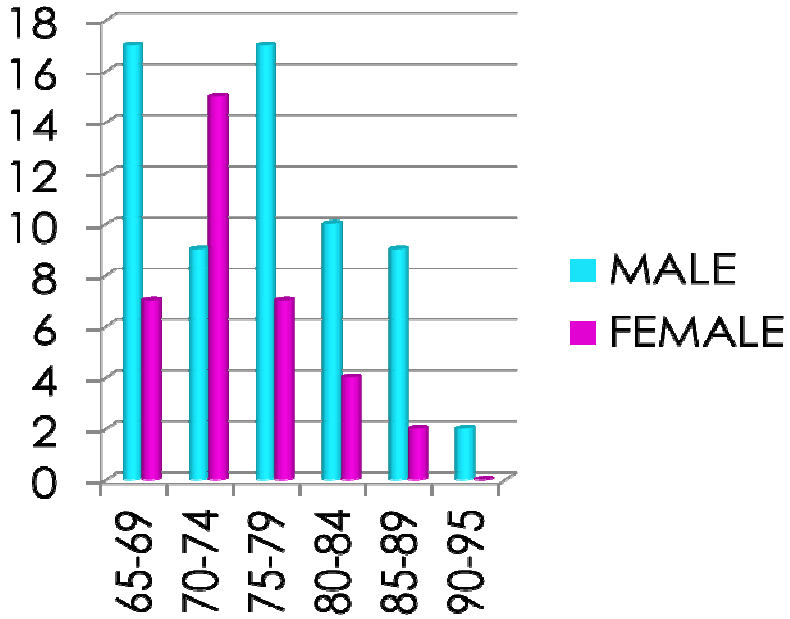


Table 1
ETIOLOGICAL DISTRIBUTION OF HYPONATREMIC PATIENTS

CAUSES	NUMBER OF PATIENTS
SIADH	24
DRUG INDUCED (DIURETICS)	16
VOLUME OVERLOAD	14
HYPOTHYROIDISM	13
VOLUME LOSS	12
MULTIFACTORIAL	10
ADRENAL INSUFFICENCY	6
RENAL FAILURE	5

Table 2
ASSOCIATED CHRONIC DISEASES

HEART FAILURE	- 20
DIABETES MELLITUS	- 8
HYPOTHYROIDISM	- 15
HYPERTENSION	- 15
DCLD	- 7
HYPOADRENALISM	- 6
CKD	- 3

Figure 4
Of the patients with hyponatremia,48% were euvolemic,21 % were hypervolemic and 31% were hypovolemic.

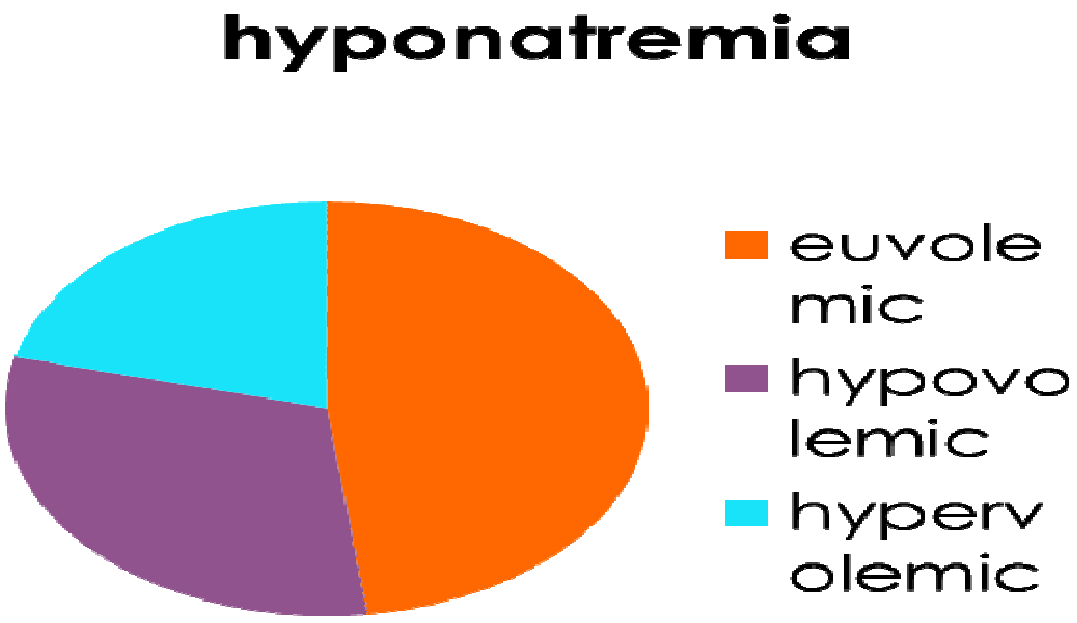


Table 3
OUTCOME OF ADMITTED PATIENTS WITH HYPONATREMIA

OUTCOME	NO.OF PATEINTS
IMPROVED	93
DIED	4
AMA DISCHARGE	3

Of the patients who died one had lung carcinoma and SIADH, one had CKD and SIADH ,one had sepsis and another DCMP with multifactorial etiology Incidence of hyponatremia around 22% of overall hospitalized patients. Incidence of hyponatremia is higher during the peak southwest monsoon season ,it varies with season to season. Humidity and temperature may have important role in the manifestation of hyponatremia.[1].There were 65 males and 35 females in our study .Other studies such as Vergese et al (male 56% , female 44%) , Chatterjee et al (male 62.69 , female 37.31) have shown male predominance in the incidence of the disease. In the present study, SIADH was the most common cause of hyponatremia -24% of cases. Infections especially pneumonia was found to be the commonest disease associated with SIADH in our study.We used Barter and Scwartz criteria mentioned earlier for the diagnosis of SIADHIn other studies by Hochman[2], SIADH represented 28.3% of cases, 34% in the study by Anderson[3],32.84% in the study by Chatterjee and 34.8% in the study by Vurgese. This correlates with other studies on hyponatremia.

Table 4
COMPARATIVE STUDIES

HYPONATREMIA	RAO STUDY	CHATTERJEE STUDY	OUR STUDY
EUVOLEMIA	61	50.74	48
HYPERVOLEMIA	23	26.86	21
HYPOVOLEMIA	16	22.4	31

DISCUSSION

PSEUDOHYPONATREMIA

It refers to a laboratory phenomenon by which a high content of plasma proteins and lipid proteins expands the non - aqueous portion of the plasma sample, leading to an errant report of low ECF sodium level. This can be rectified by sodium sensitive electrodes and also by normal serum osmolality.[4,5]

HYPEROSMOLAR HYPONATREMIA

It refers to the circumstances in which an osmotically active solute other than sodium accumulates in the ECF,drawing water into the ECF and diluting the Na+content. Some refer this as translocation hyponatremia. It occurs with glucose, mannitol 50, sorbitol, glycerol and radio - contrast agents. Poor glycemic control accounts for hyponatremia in 10% to 20% of the hospitalized patients This is most common among hyperglycemic individuals, fall in the plasma sodium of 1.6 - 2.4 mEq/L for every 100 mg/dl rise in plasma glucose.

HYPOVOLEMIC HYPONATREMIA

It may result from any cause of net Na+loss such as in thiazide use and cerebral salt wasting. Loss of both sodium and water either through the kidneys, gastrointestinal tract or skin can lead to ECF volume depletion.

Diuretics can cause hyponatremia by more than one mechanism

(1) volume depletion, which results in impaired water excretion by both enhanced AVP release and decreased fluid delivery to the diluting segment
(2) a direct effect of diuretics on the diluting segment; and
(3) K+ depletion causing a decrease in the water permeability of the collecting duct as well as an increase in water intake. K+depletion leads to hyponatremia independent of the Na+depletion that frequently accompanies diuretic use. Cerebral salt wasting syndrome is characterized by hyponatremia, hypovolumia, natriuresis and diuresis. The mechanism by which intracranial disease leads to cerebral salt wasting syndrome is not well understood. The postulated mechanism include disruption of neural input into the kidney or the central elaboration of a circulating natriuretic factor or both,[6,7] resolves spontaneously within two or three weeks of cerebral insult and responds well to prompt treatment with intravenous saline.[8]

HYPERVOLEMIC HYPONATREMIA

It occurs in edematous states such as congestive heart failure, hepatic cirrhosis and nephrotic syndrome. Despite of the expanded interstitial space, effective circulating volume is reduced. Increase in total body water results from increased vasopressin concentration, causing up - regulation at aquaporin2 water channels and further anti – diuresis

EUVOLUMIC HYPONATREMIA

The body water content is increased with a normal to mild decrease in total body sodium. In most cases of euvolumic hyponatremia, the intravascular volume is normal. Euvolumic hyponatremia can be due to translocation hyponatremia, SIADH, hypothyroidism, adreno - corticotropin deficiency or primary polydipsia.

DRUG INDUCED HYPONATREMIA

Diuretics have potential to cause hyponatremia. Other drugs that can cause hyponatremia are amiodarone, lisinopril, digoxin, chlorpropamide, tolbutamide, desmopressin, cl ofibrate, clozapine, theophiline, carbamazepine, opiates, oxytocin, prostaglandin synthesis inhibitors, nicotine, phenothiazines, selective serotonin re - uptake inhibitors, antipsychotics, tricyclic antidepressants, trazodone, vincristine, cyclophophomide and NSAID's are known to cause hyponatremia.

SIADH

Excessive secretion or function of AVP results in the production of decreased volumes of more highly concentrated urine. The hyponatremia in SIADH secretion results from either increased circulating vasopressin as seen in lung malignancies or from abnormal osmoregulation from a 'reset osmostat ' for vasopressin secretion, such that vasopressin is released at an abnormally low threshold of plasma osmolality.[9,10] Another important factor that leads to hyponatremia in patients with SIADH is the abnormal increase in thirst.[11]

SIADH is best defined by the classic criteria introduced by Bartter and Schwartz in 1967 which is as follows,

1. Hyponatremia with corresponding hypoosmolality
2.Continued renal excretion of Na^+
3. Urine less than maximally dilute
4. Absence of clinical evidence of volume depletion -Normal skin turgor, blood pressure within the reference range
5. Absence of other causes of hyponatremia - Adrenal insufficiency (mineralocorticoid deficiency, glucocorticoid deficiency), hypothyroidism, cardiac failure, pituitary insufficiency, renal disease with salt wastage, hepatic disease, drugs that impair renal water excretion
6. Correction of hyponatremia by fluid restriction [12]

Recent treatment recommendations and guidelines framed by the European Society of Intensive Care Medicine, European Society of Endocrinology and the European Renal Association – European Dialysis and Transplant Association

1.For serious symptomatic hyponatremia,the first line of treatment is intravenous infusion of hypertonic saline,with a target increase of 6mmol/L during every 24 hours thereafter until the patient's serum sodium concentration reaches 130 mmol/L.
2. First line of treatment for patients with SIADH and moderate or profound hyponatremia should be fluid restriction; Second line of treatments include increasing solute intake with 0.25 - 0.50 g/kg per day of urea or combined treatment with low dose loop diuretics and oral sodium chloride.
3.Lithium, demeclocycline and vaptans are not recommended for patients with moderate or profound hyponatremia.

CLINICAL FEATURES OF HYPONATREMIA

The severity of symptoms and signs are generally more prominent in elderly patients due to their failure of homeostatic mechanisms. It can also be explained by the increase in use of sodium lowering drugs in the elderly such as selective serotonin reuptake inhibitors for depression, or thaizide diuretics for hypertension and secondary prevention of stroke.The signs and symptoms depend on the degree of hyponatremia and speed of reduction of sodium.
Clinical features may be headache, lassitude, musclecramps, confusion , obtundation , eventually seizures, coma and respiratory arrest in acute hyponatremia. Evaluation of other disease associated with the hyponatremia is vital as they are involved in the pathogenesis of the disease. Hence the physician should have suspicion of hyponatremia in patients pneumonia , pulmonary tuberculosis, pulmonary abscess, bronchiectasis, malignancies and chronic obstructive pulmonary disease.[13,14] In chronic hyponaremia cerebral adaptation helps the patient to be asymptomatic though the plasma sodium is as low as 115 - 120mEq/L.

CONCLUSION

1. Symptomatic hyp
onatremia is common among the hospitalized patients.
2. Male population outnumbered female population in the incidence of hyponatremia.
3. SIADH and euvolumic hyponatremia formed the largest subgroup in the study.
4. Drugs, especially diuretics such as furosemide and hydochlorothiazide, are a common cause of hyponatremia.
5. Significant number of patients had endocrine abnormalities (hypothyroidism,hypoadrenalismand panpituitirism).

LIMITATIONS OF THIS STUDY

Small sample size

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