

CLINICAL PROFILE OF SEVERE HYPONATRAEMIA

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ABSTRACT

BACKGROUND

Hyponatraemia is a common electrolyte abnormality that can be seen in isolation or as most often as a complication of other medical illness. Clinically, hyponatraemia is often unrecognised when it develops slowly, whereas severe hyponatraemia particularly of rapid onset is associated with substantial morbidity and can be life-threatening. Hyponatraemia is one of the treatable causes of electrolyte disorder with neuropsychiatric manifestations.

MATERIALS AND METHODS

Patients detected to have severe/symptomatic hyponatraemia admitted in the Department of General Medicine and Surgery was included in the present study. All were evaluated by a detailed history, clinical examination and relevant blood investigations.

RESULTS

Among the study subjects, 51% were males, 84% of subjects were beyond 60 years of age with mean age of 69.94 years. Majority of them had acute onset. Alteration in the sensorium was the most common presentation (98%), followed by weakness.

CONCLUSION

Severe/symptomatic hyponatraemia is more common among the elderly population. Lower the serum sodium baseline level, higher the probability of severe grades of altered sensorium (coma, stupor) and convulsions.

KEYWORDS

Hyponatraemia, Osmolality, Volume Status, SIADH.

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BACKGROUND

Hyponatraemia is a common electrolyte abnormality that can be seen in isolation or most often as a complication of other medical illness. Clinically, hyponatraemia is often unrecognised when it develops slowly, whereas severe hyponatraemia particularly of rapid onset is associated with substantial morbidity and can be life-threatening.

The normal serum sodium level is 136-145 mEq/L.¹ When hyponatraemia is defined as serum sodium level <136 mEq/L² incidence as high as 15-30% have been observed in the studies of both acutely^{3,4} and chronically^{5,6} hospitalised patients. However, incidence decreases to the range of 1-4% when only serum sodium <130 mEq/L are included,^{7,8,9} which represents a more reliable level to define the occurrence of clinically significant cases of this disorder. Perhaps most importantly, reports of all studies till date have noted a high proportion of iatrogenic or hospital-acquired hyponatraemia, which has accounted for as much as 40-75% of all patients studied.^{9,10} Although, hyponatraemia and

hypo-osmolality are relatively common. Most cases are relatively mild and manifest during the course of hospitalisation. The present study was conducted to study the clinical profile and patient characteristics in individuals admitted with severe/symptomatic hyponatraemia in the Department of General Medicine and Surgery in T.D. Medical College Hospital, Alappuzha, from September 2007 to August 2008.

MATERIALS AND METHODS

A descriptive study was conducted for a period of one year in T.D. Medical College Hospital, Alappuzha. 100 patients detected to have severe/symptomatic hyponatraemia admitted in the Department of General Medicine and Surgery was included in the present study. All were evaluated by a detailed history including prior drug therapy if any clinical examination. Blood samples were taken for assessment of complete haemogram, random blood sugar, renal function tests (blood urea, serum creatinine), liver function tests, lipid profile, serum electrolytes and serum uric acid. Urine sample was taken for evaluation of microscopic deposits, albumin, sugar, spot sodium mmol/L and urine osmolality (mOsm/kg of water). They were also subjected to an x-ray chest and electrocardiogram as a part of routine evaluation. Creatinine clearance is assessed as per the Cockcroft-Gault equation.^{1,2} If indicated as per the history, clinical examination and baseline laboratory evaluation, patients were also subjected to imaging studies like USG abdomen, CT, lumbar puncture,

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echocardiography and hormonal assays - serum cortisol, thyroid function tests. Volume status is assessed as per history, clinical examination and investigation results.¹Patients were categorised as per the duration from the onset of symptoms to clinical presentation into acute (≤ 48 hours) and chronic (>48 hours) and based on the volume status clinically into hypervolaemic, hypovolaemic and as euvolaemic.¹

RESULTS

In the present study, we included 100 patients, of which 49% females and 51% males.

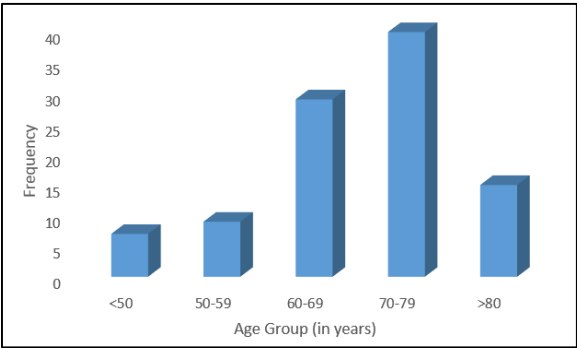


Figure 1. Distribution of Study Subjects Based on their Age

84% of subjects were beyond 60 years of age with mean age of 69.94 years (S.D. - 11.722). Mean age in males was 67.25 years (S.D. - 12.15) and in females was 72.7 years (S.D. - 10.68).

Based on the mode of presentation, 77% had acute onset and 23% had chronic onset.

Symptoms	No.	%	95% Confidence Interval
Headache	35	35	0.26-0.44
Anorexia	71	71	0.69-0.85
Nausea	77	77	0.62-0.80
Vomiting	70	70	0.61-0.79
Convulsions	37	37	0.26-0.47
Weakness and/or cramps	92	92	0.86-0.97
Alteration in sensorium	98	98	0.95-1.00

Table 1. Distribution of Subjects Based on their Presentation

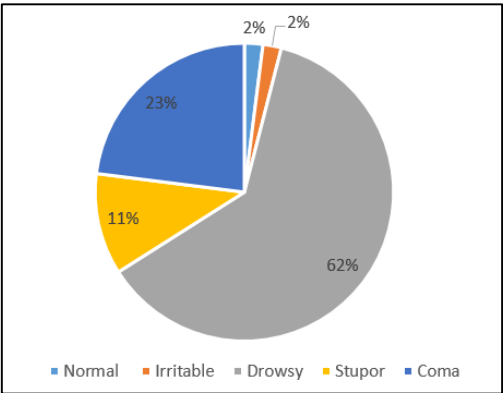


Figure 2. Distribution of Subjects Based on Alternation in Sensorium

Volume	Percentage
Euvolaemia	26%
Hypervolaemia	27%
Hypovolaemia	47%

Table 2. Distribution Based on Volume Category

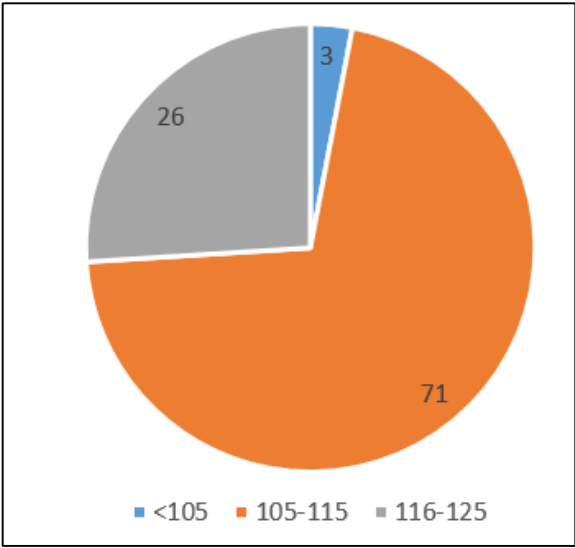


Figure 3. Distribution of Serum Sodium Level (mmol/L)

Mean baseline serum sodium level was 112.79 (S.D. - 4.006). Around 4% had urine spot sodium <10 mmol/L, 16% had 10-20 mmol/L and 80% of them had >20 .

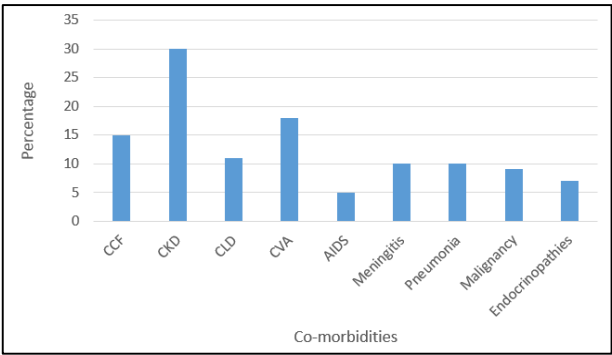


Figure 4. Distribution Based on the Comorbidities

DISCUSSION

Hyponatraemia is defined as a serum sodium concentration less than 136 mmol/L, whereas hypernatraemia always denotes hypertonicity and hyponatraemia can be associated with low, normal or high tonicity.

Dilutional hyponatraemia, the most common form of the disorder is caused by water retention. If water intake exceeds the capacity of the kidney to excrete water, dilution of body solutes ensues with hypo-osmolality and hypotonicity. Hypotonicity, in turn, can lead to cerebral oedema, a potentially life-threatening complication. Hypotonic hyponatraemia can be associated with normal or even high serum osmolality, if sufficient amounts of solutes that permeate cell membranes (e.g., urea and ethanol) have been retained. Importantly, patients who have hypotonic hyponatraemia, but normal or high serum osmolality are

subjected to the risk of hypotonicity as the patients with hyposmolar hyponatraemia.

The non-hypotonic hyponatraemias include hypertonic (or translocational) hyponatraemia, isotonic hyponatraemia, and pseudohyponatraemia. Translocational hyponatraemia results from a shift of water from within cells to the extracellular fluid that is driven by solutes confined in the extracellular compartment (occurs with hyperglycaemia in uncontrolled DM or retention of hypertonic mannitol); serum osmolality is increased, as is tonicity, the latter causing dehydration of cells. Hyperglycaemia is the most common cause of translocational hyponatraemia. Retention of large volumes of isotonic fluids in the extracellular space that do not contain sodium (e.g. isotonic mannitol solution) generates iso-osmolar and isotonic hyponatraemia, but no transcellular shifts of water. Massive absorption of isotonic irrigates that are sodium free (e.g. those used during transurethral prostatectomy or for control of uterine bleeding) can cause severe hyponatraemia.

Pseudohyponatraemia is a spurious form of iso-osmolar and isotonic hyponatraemia that represents a laboratory artefact. Marked elevations of either lipids or proteins in plasma can cause artificial decreases in serum sodium, because the relatively larger proportion of plasma volume that is occupied by the excess lipids or proteins. Direct measurement of serum sodium with the ion-specific electrode in undiluted sample eliminates this artefact.

In our study, we have included 100 cases of severe/symptomatic hyponatraemia admitted to the Department of General Medicine and General Surgery, T.D. Medical College, Alappuzha, between September 2007 and August 2008. We have analysed the clinical profile of severe/symptomatic hyponatraemia in our hospital. We have attempted to categorise them according to the duration of presenting symptoms and volume status clinically as well as to analyse the probable determinants of outcome of these subjects given the standard recommended treatment for hyponatraemia and underlying illnesses (if any).

In the study population, 40% of individuals were in the 70-79 years age group with the mean age of the total population being 69.94 years (S.D. - 11.722). This observation was consistent with the established data, which suggested that severe/symptomatic hyponatraemia was primarily a geriatric disease.¹¹ Of them were 51% males and 49% females. Mean age in males and females were 67.25 years (S.D. - 12.15) and 72.7 years (S.D. - 10.68), respectively. The most common symptom in the study population was alteration in sensorium 98% with 62% drowsy, 23% comatose, 11% stuporous and 2% irritable. Statistically significant correlations were noted between grades of alteration in sensorium and serum sodium level, development of convulsions and mode of presentation. Higher grades of altered sensorium like coma, stupor, etc. was associated with lower level of serum sodium and higher chance of developing convulsions in acute mode of presentation. This is consistent with established data and is probably due to higher degrees of cerebral oedema

associated with severe hyponatraemia as well as the lack of compensatory mechanisms on account of acute onset.¹³

77 of the 100 patients presented acutely (95%, C.I. - 0.69-0.85), while 23 presented as chronic hyponatraemia (95%, C.I. - 0.19-0.31). Mean baseline serum sodium (mmol/L) in acute hyponatraemics was 112.08 (S.D. - 3.76) when compared to 115.17 (S.D. - 3.97) in chronic hyponatraemics. A statistically significant correlation was noted between serum sodium level and mode of presentation with more severe hyponatraemia presenting acutely. This is probably due to the lesser chances of compensatory mechanisms to take effect acutely as the serum sodium level reduces, thereby increasing the chances for presentation of severe/symptomatic hyponatraemia. In our study population, 47% had hypovolaemia, 27% had hypervolaemia and 26% had euvolaemia. This was not consistent with the established data, where euvolaemia and hypervolaemia were associated more with severe hyponatraemia.¹¹ This discrepancy might be due to the small sample size of our study population and utilisation of clinical assessment criteria for volume status as against the invasive haemodynamic assessment utilising (CVP) with a sensitivity and specificity of the former being only 50-60%, when compared to the latter on account of lack of ample availability of invasive assessment facilities in our institution. Mean baseline serum sodium level in our study population was 112.79 (S.D. - 4.006) with values in males and females being 112.12 (S.D. - 3.91) and 113.49 (S.D. - 4.03), respectively. Mean sodium value in euvolaemics was 112.60 (S.D. - 3.83), hypervolaemics was 113.74 (S.D. - 3.70) and hypovolaemics was 112.36 (S.D. - 4.26). A total of 8 subjects were in the postoperative state.

In our study population, 58% had hypertension, 29% had diabetes mellitus and 39% were using diuretics. 64% of those using thiazide diuretics developed acute hyponatraemia, whereas 71.4% of those using loop diuretics developed chronic hyponatraemia. This was probably due to the indications for which diuretics were prescribed, (i.e.) loop diuretics being prescribed more often for hypervolaemic states who are more prone to develop chronic hyponatraemia as per our study (p value-0.000) and also due to the predilection of thiazide diuretics to produce more symptomatic hyponatraemia than equivalent doses of loop diuretics on account of their effect on the free water clearance.⁵

80% of the study population had urine spot sodium (mmol/L) of more than 20 mmol/L with a mean value of 65.7 mmol/L. A statistically significant correlation was noted between age of the patients and urine spot sodium level. Hence, the older age of the patient, higher the probability of a salt losing state, which could be due to a defect in salt conserving machinery at the level of renal tubules and also increased risk of symptomatic hyponatraemia on account of low basal sodium stores.¹¹

CONCLUSION

Severe/symptomatic hyponatraemia is more common among the elderly population. Mode of presentation of

majority of the subjects was acute (77%). Females were more commonly affected in the age group of above 70 years. Lower the baseline serum sodium level, higher the probability of more severe grades of altered sensorium (coma, stupor) and convulsions. Severe/symptomatic hyponatraemia patients are more likely to have an adverse outcome, if they have any of the following concomitant disease states or comorbid illnesses states like Congestive Cardiac Failure (CCF), Chronic Kidney Disease (CKD), chronic liver disease, diabetes mellitus, etc. Serum uric acid is a useful marker of volume status.

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