

Review of cases of hyponatraemia in the Port Moresby General Hospital between August 1993 and June 1995

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SUMMARY

Hyponatraemia (serum sodium level below 130 mmol/l) is a common electrolyte abnormality in a hospital population. It can be associated with dehydration, overhydration or normal hydration. Clinically, it is important to recognize the common diseases associated with hyponatraemia since correct treatment in terms of fluid replacement is essential in preventing complications of low serum sodium. We have reviewed results of serum sodium tested from patients admitted to the Port Moresby General Hospital between 1993 and 1995. This was aimed at identifying the most common features associated with low sodium. Clinical information and diagnosis were obtained by looking through a series of request forms. Of the approximately 30,000 blood samples taken over 23 months, the percentage of samples with hyponatraemia was about 1%. Hyponatraemia was more common in medical (38%) and paediatric (35%) cases and at the extremes of ages, ie, under the age of 6 years and above 40 years. Over a quarter of the hyponatraemic patients had severe hyponatraemia (serum sodium below 120 mmol/l). Clinical conditions commonly associated with hyponatraemia, in descending order of importance, were diarrhoea and vomiting, renal failure, central nervous system infections and trauma, pulmonary infections, oedematous states (eg, nephrotic syndrome) and diabetes mellitus.

Introduction

Hyponatraemia is defined as a blood sodium level below 130 mmol/l. It is the most common electrolyte abnormality seen in a general hospital population (1). The incidence and prevalence are about 1.0% and 2.5% respectively (2). Patients with hyponatraemia compared to those with normal sodium concentration have poor treatment outcomes in any medical disorder (1,2). Besides, hyponatraemia alone can cause various central nervous system (CNS) symptoms including demyelination of the pons in severe cases (3).

Since sodium is the major determinant of the serum osmolality, a change in its concentration will be followed by a corresponding change in serum osmolality. Thus, the clinical features reflect the combined

effects of cellular overhydration – the movement of hypo-osmolar extracellular fluid (ECF) into the intracellular fluid (ICF) compartment – and acute water intoxication or depletion.

Hyponatraemia is almost always the result of dilution. However, every state of hydration status, ie dehydration, overhydration and normal hydration, can be associated with hyponatraemia. These may follow diarrhoea or vomiting, over-infusion of intravenous fluids and the syndrome of inappropriate secretion of antidiuretic hormone (SIADH) caused by various diseases.

In many rural hospitals in Papua New Guinea where measurements of electrolytes and osmolality may not be immediately available, it is important to recognize the

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common clinical conditions associated with hyponatraemia so that fluid balance can be closely monitored to avoid poor response to treatment.

We performed a retrospective analysis of low blood sodium levels analyzed and recorded in the Department of Biochemistry in the Port Moresby General Hospital to describe associated features as determined from the laboratory request forms.

Methods

From the sample sent to the Department of Biochemistry serum sodium was measured on Technicon RA-XT and RA-1000 electrolyte analyzers. The results were usually recorded at the back of the request forms and kept for at least 5 years before being destroyed. The original laboratory request forms of hyponatraemic patients were collected for the period between August 1993 and June 1995. Information including name, age, sex, date of specimen collection, ward of admission, clinical diagnosis and hand-written results of laboratory tests were collected from the laboratory request forms. Sufficient clinical information was gathered by looking through a series of request forms for each patient to extract the relevant features. Where multiple results of hyponatraemia were recorded in a single patient, the lowest sodium level was taken so that no multiple results of one individual were included in the calculations.

Results

Approximately 30,000 patients' sera were tested for sodium concentration between August 1993 and June 1995. Of these, 312 samples were found to have a plasma sodium concentration below 130 mmol/l (hyponatraemia). Therefore, the rate of hyponatraemia in the Port Moresby General Hospital laboratory was approximately 1%.

Over one-third (38%) of the samples with hyponatraemia were from the medical wards while paediatric and surgical samples accounted for another 35% and 11% respectively (Table 1). 15% of samples with hyponatraemia came from the Acute and Emergency Department. There were much fewer hyponatraemic samples from the obstetrics and gynaecological wards.

More than 85% of the hyponatraemic patients had a serum sodium concentration of less than 126 mmol/l with a mean of 121 mmol/l. Approximately 27% of the patients with low sodium had severe hyponatraemia. There were more males with hyponatraemia (63%) than females (37%) (Table 2). Furthermore, the number of patients with hyponatraemia was greater at the extremes of age than in the adolescent and middle-age groups (Table 3). However, the degrees of hyponatraemia between sexes and age groups were similar. 35% of the patients were adults who did not know their exact age. The major

TABLE 1

DEGREES OF HYPONATRAEMIA ACCORDING TO THE WARD OF ADMISSION

Ward	Hyponatraemia			Total	
	Mild 126-130 mmol/l	Moderate 120-125 mmol/l	Severe <120 mmol/l	N	%
Surgical	3	25	6	34	11
Medical	15	69	33	117	38
Paediatrics	15	54	41	110	35
Acute and Emergency	11	34	3	48*	15
Obstetrics and Gynaecology	0	1	2	3	1
Total	44	183	85	312	100

* All except 3 were medical patients

TABLE 2

INCIDENCE OF HYPONATRAEMIA ACCORDING TO SEX

Sex	Hyponatraemia			Total	
	Mild 126-130 mmol/l	Moderate 120-125 mmol/l	Severe <120 mmol/l	N	%
Male	27	117	54	198	63
Female	17	66	31	114	37
Total	44	183	85	312	100

TABLE 3

INCIDENCE OF HYPONATRAEMIA ACCORDING TO AGE

Age (Years)	Hyponatraemia			Total	
	Mild 126-130 mmol/l	Moderate 120-125 mmol/l	Severe <120 mmol/l	N	%
<1	5	31	19	55	18
1-5	10	27	18	55	18
6-10	0	7	4	11	4
11-20	4	12	3	19	6
21-30	4	8	1	13	4
31-40	1	7	5	13	4
>40	4	20	14	38	12
Adults	16	71	21	108	35
Total	44	183	85	312	100

causes of hyponatraemia (Table 4) were gastrointestinal losses (29%), renal failure including neonatal sepsis with azotaemia (16%), CNS infections and trauma (9%), pulmonary infections (9%), oedema states (6%) and diabetes mellitus (4%).

Discussion

1% of blood samples for sodium measurement tested in the Port Moresby General Hospital laboratory showed hyponatraemia. It is suggested that severe hyponatraemia is associated with CNS symptoms due to brain damage. These hyponatraemic effects are more serious in the presence of other severe medical illnesses (1,2). In our review over one-quarter (27%) of the patients with low sodium had severe

hyponatraemia. It would be of great interest to evaluate the progress and prognosis of such patients.

Most illnesses associated with hyponatraemia require admission to medical or paediatric wards. Consequently, a high proportion of hyponatraemic samples originated from these wards (medical 38% and paediatric 35%). The mechanisms of development of hyponatraemia are multifactorial but are always due to dilution (4-6). Even in the case of predominant sodium loss from the body, there is relatively more water than sodium in the body. Thus, this will still result in dilutional hyponatraemia. Surgical patients accounted for only 11% of the total of hyponatraemia patients. Interestingly the majority (92%) of these

TABLE 4

DISORDERS THAT WERE ASSOCIATED WITH HYPONATRAEMIA

Disorders	Hyponatraemia			Total	
	Mild 126-130 mmol/l	Moderate 120-125 mmol/l	Severe <120 mmol/l	N	%
Diarrhoea and vomiting	11	59	19	89	29
Bowel obstruction	-	10	2	12	4
Marasmus and failure to thrive	-	4	8	12	4
Cancer of upper and lower GIT	-	5	-	5	2
Meningitis (TB and acute bacterial)	2	11	3	16	5
Cerebral malaria	3	5	-	8	3
Hydrocephalus and convulsions	1	2	2	5	2
Head injury, CVA and SOL	-	2	3	5	2
Acute and chronic renal failure	3	22	12	37	12
Bacterial (neonatal) sepsis*	-	7	7	14	4
Pneumonia and pulmonary TB	4	17	8	29	9
COAD, bronchiectasis and asthma	-	3	4	7	2
Pleural effusion, cancer of the lung	-	2	3	5	2
Assisted respiratory ventilation	-	1	-	1	0
Nephrotic syndrome	-	2	2	4	1
Cirrhosis and ascites	1	-	1	2	1
Congestive cardiac failure	1	1	2	4	1
Hepatitis	1	6	1	8	3
Congenital adrenal hyperplasia	-	-	1	1	0
Haemorrhage and skin burns	1	2	-	3	1
Malignancies (breast cancer, pancreas and lymphoma)	-	3	1	4	1
Diabetes mellitus**	3	6	2	11	4
Others (including IV drip site/Indocid intoxication)	13	13	4	30	10
Total	44	183	85	312	100

* All had associated azotaemia

** Without evidence of renal impairment

GIT = gastrointestinal tract

TB = tuberculosis

CVA = cerebrovascular accident

SOL = space occupying lesion

COAD = chronic obstructive airway disease

developed hyponatraemia while in the ward whereas only 8% presented at the initial examination in the Acute and Emergency Department of the hospital.

Reports in other countries show hyponatraemia developing in more than 4% of all surgical patients (7,8). These reports do suggest that excessive administration of hypotonic fluid is the major (85%) cause of hyponatraemia in surgical patients. In the Port Moresby General Hospital we do not know the incidence and prevalence of hyponatraemia in surgical patients. However, it is obvious from this study that more surgical patients are developing hyponatraemia whilst in the wards than on arrival at the Acute and Emergency Department. Thus, an additional investigation is needed to determine the percentages of ward-induced hyponatraemia such as that due to excessive administration of hypotonic fluids postoperatively.

The low number of cases of hyponatraemia seen in obstetrics and gynaecological wards is expected since apart from hyponatraemia induced by post-surgical infusion of fluids, most diseases associated with hyponatraemia are unlikely to be admitted to these wards. Degrees of hyponatraemia between sexes were similar. However, a definite male preponderance in the number of cases of hyponatraemia was seen. The reason for this difference could not be deduced.

Our review shows that hyponatraemia is more common at the extremes of age (<6 years old and >40 years of age) than in those between the ages of 6 and 40 years. This is related to the relatively high prevalence of illnesses causing hyponatraemia such as diarrhoea and respiratory diseases in these age groups. In general, young adults and adolescents are more efficient at correcting changes in electrolytes and body fluids than those at the extremes of age for the same illness.

It has been reported that SIADH from a wide variety of diseases including drugs is the commonest cause of hyponatraemia (4-6). In this preliminary survey in the Port Moresby General Hospital, body fluid loss seems to be more commonly associated with hyponatraemia than SIADH (35% versus

23%). However, this is based on the limited clinical information we obtained. A more definitive pathophysiology and diagnosis of each disorder causing hyponatraemia can only be established by reviewing complete patient admission notes in addition to the levels of serum sodium, and measuring urine osmolality, urinary sodium and plasma ADH levels. A thorough check of a series of request slips with review of other laboratory test results was performed for each hyponatraemic patient to support the associated clinical diagnosis. Furthermore, the high percentage of hyponatraemia associated with fluid loss is to be expected in the Port Moresby General Hospital with the high prevalence of diarrhoeal diseases, a leading cause of admission and morbidity in children.

Hyponatraemia in diabetes without laboratory signs of severe renal impairment (creatinine <0.3 mmol/l) was 4%. These patients could have had hypertonic hyponatraemia due to the high glucose level. A measure of plasma osmolality would have been helpful to confirm or exclude this. On the other hand, the effect of oral hypoglycaemic drugs that are known to cause SIADH cannot be excluded.

Conclusion

Although limited by the nature of retrospective analysis, low sodium was detected in about 1% of the blood samples sent to the biochemistry laboratory in the Port Moresby General Hospital over the 23-month period of the study. More than 85% of the patients with low serum sodium had moderate to severe hyponatraemia. Low sodium levels were more common in children (below 6 years) and in adults over 40 years of age. The most common clinical features (as indicated on request forms) associated with low serum sodium were diarrhoea and/or vomiting. Other features indicated included renal failure, tuberculosis, bacterial pneumonia, diabetes mellitus, neonatal sepsis and bowel obstruction. A further study involving a review of patients' ward notes and measurement of serum and urinary sodium and osmolality plus serum ADH is required to determine the definitive causes of hyponatraemia at the Port Moresby General

Hospital. This may also involve an assessment of the prognosis of such patients in the hospital.

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