

Hospital Practice

Serious hypernatraemia in a hospital population

D. Bhatnagar and C. Weinkove

University of Manchester Department of Clinical Biochemistry, Hope Hospital, Eccles Old Road, Salford, M6 8HD, UK.

Summary: Severe hypernatraemia in a hospital population should be an avoidable problem. We have looked at its causes and incidence over one year and have shown that serious hypernatraemia (serum sodium >160 mmol/l) as a manifestation of severe dehydration is associated with a high morbidity and mortality. Failure to maintain adequate fluid intake, intentional or unintentional, was the most frequent cause. Nursing and medical staff must be made more aware of this problem and encouraged to initiate early treatment of dehydration.

Introduction

Serum sodium is frequently estimated in patients admitted to hospital. The commonest abnormality seen is hyponatraemia and hypernatraemia is much less frequent.^{1,2,3} Serious hypernatraemia has a mortality approaching 60% in adults and almost certainly contributes to the morbidity and mortality of the underlying disease.⁴

We were interested in investigating the factors which contribute to serious hypernatraemia and what measures can be taken for prevention.

Methods

All serum sodium requests made over a period of one year were identified by running a search on the Department of Chemical Pathology computer. A serum sodium of 160 mmol/l was chosen as the cut-off point for severe hypernatraemia since this is indisputably abnormal and associated with a high mortality.⁴

Results

Twenty-seven inpatients with a serum sodium more than 160 mmol/l were discovered and cumulative

reports were produced on all their biochemical results. Case notes from 24 patients were scrutinized for the reason for admission, the course of the illness and the treatment carried out. Records on the remaining 3 patients could not be traced.

The patients ranged from 19 years to 99 years old with a mean age of 71.4 years. In 10 patients the serum sodium was >160 mmol/l on admission. In the other 14 patients serious hypernatraemia developed while in hospital. In 7 of these patients action regarding the hypernatraemia was taken within 48 hours of results being available. In the other 7 patients it took between 3 to 12 days before treatment was initiated.

Fluid balance charts were maintained in only 13 of the 24 patients even after hypernatraemia was noted.

In 8 patients the hypernatraemia resulted from withholding all treatment including intravenous fluids because of the advanced age or illness of the patient. All but one of these patients had biochemical estimations done after the decision to stop treatment was taken.

The mechanisms and causes resulting in hypernatraemia are shown in Table I. Five patients had established renal failure. In the remainder of the 19 patients there was biochemical evidence of pre-renal uraemia with serum urea levels elevated relative to serum creatinine.

The 16 patients treated were given 5% dextrose or half normal saline or in some cases normal

Correspondence: D. Bhatnagar, M.B., B.S., University Department of Medicine, Manchester Royal Infirmary, Oxford Road, Manchester M13 9WL, UK.

Accepted: 13 January, 1988

Table I Mechanisms and causes of serious hypernatraemia (>160 mmol/l) in 24 inpatients. (Note: most patients had more than one mechanism leading to hypernatraemia)

Mechanism	Cause	Number of patients	
A. Poor intake	Confusion	4	
	Coma	4	
	Depression	1	
B. Water loss due to			
	(a) Increased insensible losses	Fever	5
		Tachypnoea	8
	(b) Diuresis	Diuretic overuse	2
		Non-ketotic hyperglycaemia	5
		(c) Loss from gastrointestinal tract	Diarrhoea
	Rectovaginal fistula	1	
C. Essential hypernatraemia	Head injury	1	
D. Excessive salt intake	Nil	Nil	

saline. All showed clinical and biochemical improvement within 3 days of treatment, but 11 patients within this group died. In all 19 of the 24 patients died, including the 8 untreated patients.

Discussion

The majority of our patients were hypernatraemic due to a water deficit. More than one mechanism was responsible for this, but poor fluid intake due to an altered sensorium and increased insensible water losses were most common. These are potentially remediable causes of hypernatraemia and should have been anticipated, especially in the confused and unconscious patients.

The other major mechanisms of hypernatraemia were fluid loss from the gastrointestinal tract and osmotic diuresis associated with non-ketotic hyperglycaemia.

The delay in diagnosis leading to severe hypernatraemia in 14 patients occurred despite apparently constant nursing and medical care (as judged by documentation in the case notes) and the ready availability of biochemical results. This strongly suggests a lack of awareness of the problem of water balance. Further evidence of this was seen in diabetic patients where diabetic charts were filled in diligently, but fluid intake/output charts were not maintained or were incomplete.

Hypernatraemic dehydration in an institutionalized patient was considered to be an indicator of neglect in a study from California.⁵ Twenty-nine of 56 hypernatraemic patients admitted to 2 public hospitals over a 26 month period came from 2 nursing homes which later had their licences withdrawn. The seriousness of underlying disease may sometimes distract from attention to water balance. In 2 of our patients, one with complete heart block and the other with myocardial infarction, gross hypernatraemia and confusion resulted after small rises in plasma urea and sodium were ignored. Both these patients responded well on being given fluids.

Other factors could have led to severe hypernatraemia in our patients. The majority of our patients were elderly and as a group they are prone to dehydration following mild stress⁶ and have reduced sensation of thirst.⁷ In the confused patient the desire to drink may not be apparent. Another, often ignored, cause of dehydration in the elderly is water loss due to an increase in ventilation following a chest infection or other disease leading to rapid respiration.⁸

The clinical features of dehydration are difficult to detect especially in the elderly.⁹ The more reliable signs, such as dry mucous membranes, may not appear until substantial water loss has occurred and serum sodium levels only begin to rise when about 3 litres of water have been lost.¹⁰

The best means of avoiding dehydration is increased awareness of the potential causes of water depletion such as infections, fever, diarrhoea. Simple measures such as fluid balance charts and careful observation may warn of impending dehydration. Nursing staff frequently bring to the attention of medical staff the changes in temperature, blood pressure, general condition or social circumstances. We feel they can help in the detection of dehydration. This is also suggested by Turner *et al.*¹¹ who reviewed current practices in maintaining hydration in a hospital population. They suggested the use of spot urine and serum osmolalities to assess early dehydration, although this may not be useful in every patient.

In the 8 untreated patients the question arises as to why the serum sodium measurements were made. There is debate amongst physicians whether terminal dehydration is painful.^{12,13} In surveys in the United States it would seem that most physicians prefer to give intravenous fluids to a comatose dying patient¹⁴ to try and maintain normal blood biochemistry.¹⁵ However, it has been suggested that this may act as a barrier between patients and relatives and may divert attention from patient and family care.¹⁶ We feel that doing tests in such circumstances is unnecessary unless some action is planned on the results obtained.

Conclusions

Our findings highlight the need for increased awareness and prompt treatment of conditions leading to serious hypernatraemia, especially in the elderly. Elevated plasma sodium is a late marker of dehydration and rising values of plasma urea and sodium and of the haematocrit should be watched

closely along with urine and serum osmolalities. Nursing staff should draw to the doctor's attention any changes in the patient's sensorium and in the fluid intake output charts. Patients with terminal disease should not have their blood biochemistry measured without strong reason to act on the results.

References

1. Chung, H.M., Anderson, R.J., Kluge, R. & Schrier, R.W. Hyponatraemia: A prospective study of frequency, cause and outcome. *Kidney Int* 1984, **25**: 160.
2. Zierler, K.L. Hyperosmolarity in adults: A critical review. *J Chron Dis* 1958, **7**: 1-23.
3. Ross, E.J. & Christie, S.B.M. Hypernatraemia. *Medicine* 1969, **48**: 441-473.
4. Arieff, A.I. Central nervous system manifestations of disordered sodium metabolism. *Clin Endocrinol Metab* 1984, **13**: 269-294.
5. Himmelstein, D.U., Jones, A.A. & Woolhandler, S. Hypernatremic dehydration in nursing home patients: An indicator of neglect. *J Am Geriatr Soc* 1983, **31**: 466-471.
6. Leaf, A. Dehydration in the elderly. *N Engl J Med* 1984, **311**: 791-792.
7. Phillips, P.A., Rolls, B.J., Ledingham, J.G.G. *et al.* Reduced thirst after water deprivation in healthy elderly men. *N Engl J Med* 1984, **311**: 753-759.
8. Wynn, V. Disorders of water and electrolyte metabolism and of acid-base balance in man. In: Scott, Sir Ronald Bodley (ed) *Price's Textbook of the Practice of Medicine*. Oxford University Press, Oxford, 1978, p. 396.
9. Lye, M. Electrolyte disorders in the elderly. *Clin Endocrinol Metab* 1984, **13**: 383.
10. Willatts, S.M. Fluid and electrolyte disorders: Water. *Br J Hosp Med* 1984, July: 8-14.
11. Turner, J., Brown, A., Russell, P., Scott, P. & Browne, M. 'Pushing fluids' - can current practices of maintaining hydration in hospital patients be improved? *J R Coll Physicians Lond* 1987, **21**: 196-198.
12. Baines, M.J. Control of other symptoms. In: Saunders, C. (ed) *The Management of Terminal Malignant Disease*. Edward Arnold, London, 1984, p 101.
13. Billings, J.A. Comfort measures for the terminally ill: Is dehydration painful? *J Am Geriatr Soc* 1985, **33**: 808-810.
14. Micetich, K.C., Stienecker, P.H. & Thomasana, D.C. Are intravenous fluids morally required for dying patients? *Arch Intern Med* 1983, **143**: 975-978.
15. Simpson, M.A. The facts of death. Prentice-Hall, Englewood Cliffs, NJ, 1979.
16. Editorial. Terminal dehydration. *Lancet* 1986, **i**: 306.