Management of Hyponatraemia

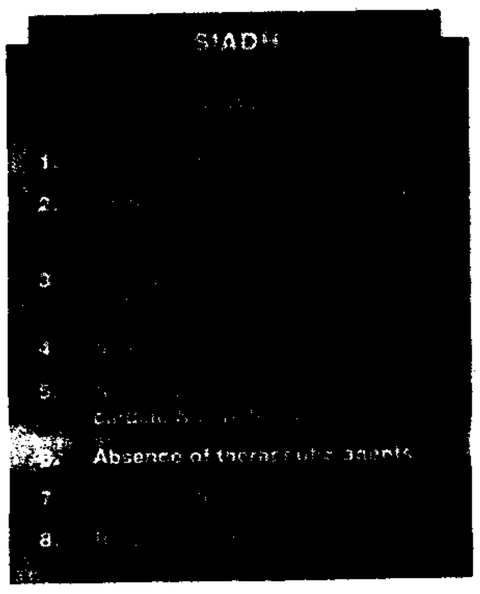
Dr Peter C Sharpe MD, MRCP, MRCPath,
Consultant Chemical Pathologist
Craigavon Area Hospital

INTRODUCTION

Hyponatraemia is the most common electrolyte disturbance in hospitalised patients. It is also commonly encountered in General Practice, particularly as a consequence of diuretics. Identifying hyponatraemia and applying the appropriate measures to elucidate the underlying aetiology and instigate correct treatment is of paramount importance for two reasons:

1. Severe hyponatraemia (Na <120 mmol/L), although less common, is associated with substantial morbidity and mortality, irrespective of the underlying disease.

2. Mild hyponatraemia can progress rapidly to a more dangerous degree with devastating results.
Hyponatraemia is often improperly managed because of a lack of understanding of the causes and failure to differentiate between them. There is also poor uptake of important laboratory investigations such as urine Na & osmolality. Last, but by no means least, there is gross overdiagnosis of the Syndrome of Inappropriate ADH secretion (SIADH).



From clinical point of view, it is helpful to categorise hyponatraemic patients into 3 categories:

1. Water excess states such as acute water intoxication and chronic water overload. Hyponatraemia should be regarded as primary water disturbance in these situations. A combination of increased fluid intake and reduced renal free water clearance can result in acute water overload. A normal fluid intake coupled with decreased renal free water clearance can lead to chronic water overload such as in SIADH. Patients are usually euvolaemic. This is by far the commonest category in which patients with hyponatraemia present.

2. Salt depletion states such as Addison's disease, diuretics, salt losing nephropathy and GI losses. In such cases hyponatraemia is a sodium disturbance primarily. Patients have volume contraction and in severe cases exhibit hypovolaemic shock.

3. A combination of water excess and salt depletion states. The patient may be euvolaemic or mildly hypovolaemic.

SYMPTOMS AND SEQUELAE OF HYPONATRAEMIA

The symptoms and sequelae of hyponatraemia depend very much on the category in which the patient presents. For example, in acute water intoxication the serum sodium level falls rapidly without time for brain adaptive mechanisms to develop. This leads to more pronounced CNS signs and symptoms, necessitating urgent management to restore normal plasma tonicity. On the other hand, patients with chronic hyponatraemia (water overload) are usually asymptomatic, due to development of brain adaptive mechanisms against hypotonicity. Therefore, less urgent management is appropriate.
The signs and symptoms of hyponatraemia are diverse and non-specific and are usually evident when serum sodium has fallen below 125 mmol/L. The most common symptoms are confusion, nausea, vomiting and headaches followed by seizures, coma and respiratory arrest. These result from ECF hypotonicity leading to movement of water inside the cells (cellular swelling). Brain cells are particularly vulnerable in this regard as they are confined within the bony skull; this is why neurological symptoms frequently predominate the clinical picture.

CLINICAL GUIDELINES FOR THE EVALUATION OF HYPONATRAEMIC PATIENTS

1. Document all relevant clinical details including fluid losses (vomiting, diarrhoea, NG aspirate, fistula drainage), drug history (diuretics, anticpileptics, antidepressants, antipsychotics, sulphonylurea), history of fluid intake (both type and volume). You should be able to suspect the type of disturbance from this.

2. Clinically assess the volume status of the patient (hypovolaemic, euvolaemic or oedematous). Document pulse rate and BP (standing and lying).

3. Measure serum urea, creatinine, electrolytes and osmolality to confirm hypo-osmolal hyponatraemia. Exclude spurious (pseudo) hyponatraemia (associated with hyperproteinaemia and hyperchylomicronaemia) and hypertonic hyponatraemia (associated with hyperglycaemia). Hyponatraemia due to salt depletion is associated with raised urea and to a lesser degree creatinine as in Addisonian crisis. Urea is usually low in SIADH. Potassium is often low (with an alkalosis) in patients taking diuretics and is often high (with an acidosis) in Addisonian crisis.

4. Measure random urine sodium and osmolality. A urine osmolality >200 mOsm/kg is inappropriately high in the presence of hyponatraemia. A random urine sodium > 20 mmol/L is inappropriate and can be due to SIADH or renal salt wasting states such as diuretics, Addison's disease or salt losing nephropathy.

5. Request other tests to help identify a specific cause for the hyponatraemia e.g. thyroid function tests (hypothyroidism), cortisol (adrenocortical insufficiency), synacthen test. Chest X-ray may uncover pulmonary pathology. Intracranial pathology should also be considered.

6. Having arrived at a diagnosis, apply appropriate therapy and clearly document it in the clinical notes, together with accurate fluid balance charts. Serum sodium measurement must be performed at frequent intervals to assess and guide response to therapy.

WHAT IS THE APPROPRIATE THERAPY FOR INDIVIDUAL PATIENTS?

1. Acute symptomatic hyponatraemia (Acute water intoxication)

This is rare, but is a Medical Emergency and ideally the patient should be managed in ICU.

A rapid fall of serum sodium in these patients results in hypotonicity with cerebral oedema, raised intracranial pressure, tentorial herniation, depression of the respiratory centre and death. Therefore rapid correction of plasma hypotonicity (serum sodium rise by 1-2 mmol/h) should be undertaken with infusion of hypertonic saline. The amount needed may be calculated as follows:

Amount of sodium (mmol) to be given per hour = 0.6 x Body weight (kg) x correction rate

For a 70 kg man and a correction rate of 1 mmol/h, it will be:

0.6 x 70 x 1 mmol/h= 42 mmol/h

To achieve this rate of correction, you must give 135 ml/h if double strength normal saline (1.8% = 310 mmol/L) is used.

You should give 81 ml/h if triple strength 2.7% NaCl (517 mmol/L) is used.

The target serum sodium for correction should be 125 mmol/L, so if initial sodium is 105 mmol/L, the hypertonic saline infusion should continue for 20 hours if 1 mmol/h infusion rate is used. Fluid restriction to 750 – 1000 ml/day should be employed alongside.
In elderly patients, use a slower infusion rate and add frusemide to enhance renal free water clearance. Check serum sodium 4 hourly during treatment to identify and avoid too rapid correction.

2. Chronic asymptomatic hyponatraemia (Chronic water overload)

As in SIADH, patients are mildly symptomatic and the brain adaptive mechanisms are fully developed. Rapid correction is therefore dangerous and can precipitate osmotic demyelination syndromes such as central pontine myelinolysis (CPM). Fluid restriction to 1 litre per day is recommended and in refractory cases demeclocycline or lithium can be added.

3. Hypovolaemic hyponatraemia (Salt depletion states)

The main problem in this group of patients is hypovolaemia. Rapid infusion of normal saline will correct both hyponatraemia and hypovolaemia. An important diagnosis not to miss in this category is Addisonian crisis, were hydrocortisone must also be given, 100 mg IV every 6 hours.

4. Hypervolaemic hyponatraemia (Oedematous states)

This is encountered in patients with heart failure, nephrotic syndrome and liver cirrhosis. There is excess of total body salt and water, but with water being proportionately more than sodium leading to hyponatraemia. Specific therapy of the underlying condition, alongside water restriction and judicious use of diuretics is required. □

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