Society for Endocrinology

Practical management of hyponatraemia

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Summer School 2002 9-12 July 2002
University of Reading, Whiteknights, Reading, UK

Introduction

Hyponatraemia is a common problem, occurring in up to 15% of non-selected emergency hospital admissions. Aetiology is diverse, and presentation can range from asymptomatic to one involving profound metabolic and neurological dysfunction. Management should reflect the severity of both the biochemical disturbance and clinical manifestations, the time over which these have developed, and the cause of the underlying problem in electrolyte balance.

Diagnosis and clinical manifestation

The morbidity and mortality of hyponatraemia are predominantly related to central nervous system (CNS) dysfunction. Values of serum sodium around 100 mmol/L are life threatening. However, patients can remain asymptomatic, if hyponatraemia develops slowly due to CNS adaptation: cellular oedema being limited by efflux of organic solutes.

Aetiology

Hyponatraemia can occur through one of three mechanisms.

- Reduction of renal free water clearance; increased total body water
- Water intake in excess of total body water loss; increased total body water.
- Sodium depletion: increased renal sodium loss in excess of free water loss

In many situations, hyponatraemia is multifactorial. Aetiologies to consider include:

1. Volume depletion.
2. Hypoadrenalism and hypothyroidism.
3. Inappropriate Vasopressin release (Syndrome of Inappropriate Antidiuresis – SIAD).
4. Drugs (including intravenous fluids).
5. Salt wasting syndromes.

The most frequent difficulty in practice is in distinguishing SIAD from chronic, mild hypovolaemia. Urine osmolality tends to be higher than plasma osmolality in both groups, and plasma Vasopressin concentrations will be detectable or elevated in both situations. Measurement of urinary sodium concentration is helpful.

Management

Chronic asymptomatic hyponatraemia, with plasma sodium concentrations greater than 125 mmol/L, may not require specific treatment. More severe degrees of hyponatraemia, particularly if symptomatic, require some form of intervention. However, there is no consensus on optimal treatment. Correction of the underlying causes, where identifiable, is appropriate: withdrawal of drugs, appropriate hormone replacement, avoidance of excess fluid intake, or correction of hypovolaemia. These measures should prevent worsening hyponatraemia, but may not address the deficit in plasma sodium. Any additional intervention should adhere to two key principles.

- Correction should not risk morbidity and mortality in excess of that associated with the initial degree of hyponatraemia.
- Correction should reverse life-threatening features of hyponatraemia as quickly as is feasible and safe.

Key in this approach are: the morbidity attributable to hyponatraemia; the time over which hyponatraemia has developed; and the target plasma sodium to be achieved. Hyponatraemia developing over several days is associated with adaptive responses in organic CNS osmolytes. Rapid correction in such circumstance risks changes in brain volume that can precipitate osmotic demyelination. Hyponatraemia developing over several hours is not associated with such adaptive responses, and rapid correction of sodium may be more appropriate in such circumstances if hyponatraemia is associated with severe symptoms.

Patients with mild to moderate symptoms of hyponatraemia, and who are not hypovolaemic, should be managed conservatively with fluid restriction, aiming to raise plasma sodium to 125-130 mmol/l at a rate not exceeding 8 mmols/L per 24 hours. Plasma sodium should be measured every 12 hours; if levels rise too quickly, fluid restriction should be relaxed. Chronic SIAD can be treated with demeclocycline (600 to 1200 mg/day). This may take several weeks to have a maximal effect. Synthetic, non-peptide Vasopressin receptor antagonists increase solute-free water excretion and are set to greatly improve the management of this condition.

If hyponatraemia is associated with severe symptoms, and especially if it has developed rapidly, intervention with hypertonic fluids may be indicated. The target plasma sodium should be one that reverses life-threatening complications, not normalisation. Plasma sodium concentration should rise no more than 1-2 mmol/L per hour, with a total increment of no more than 8 mmol/L per 24. It is imperative that the fluid regimen is reassessed at regular intervals, guided by careful clinical assessment and laboratory monitoring. The rapid correction of hyponatraemia should be stopped if the following targets are achieved.

- Reversal of life-threatening manifestations of hyponatraemia.
- Moderation of other non-life threatening manifestations of hyponatraemia.
- Achievement of a plasma sodium concentration of 125mmols/L.

Complications of treatment

Changes in brain volume in response to changes in osmolar gradient across the blood-brain barrier as serum sodium changes can trigger CNS demyelination: a rare but serious complication of hyponatraemia and its treatment. Neurological manifestations may include quadriplegia, opthalmoplegia, pseudo-bulbar palsy and coma. Osmotic demyelination can develop within 1-4 days of rapid (> 12 mmols per 24 hours) correction of plasma sodium, irrespective of the method employed to achieve it. It can occur even when sodium levels are corrected slowly. Other factors (hepatic failure, potassium depletion, acidosis, malnutrition) may play a role in susceptibility.

Summary

The management of hyponatraemia requires a practical understanding of fluid and electrolyte balance, the mechanisms through which these are maintained, and ultimately the pathophysiological processes that can influence them. It is key to place these within the clinical context of the patient, ensuring appropriate tailored therapy and optimal outcome.

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Go to top of page

Location: Home / Training
Revised: 04-Sep-2003
Contact the Society