

SOCIETY FOR ENDOCRINOLOGY ENDOCRINE EMERGENCY GUIDANCE

Emergency management of severe symptomatic hyponatraemia in adult patients

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Introduction

Hyponatraemia (serum sodium <135 mmol/L) is common. Presentation can cover a broad spectrum of symptoms and signs. Severe hyponatraemia can be life threatening requiring emergency assessment and treatment. This guidance covers emergency management of severe symptomatic hyponatraemia.

Recognition of the patient presenting with severe and moderately severe, symptomatic hyponatraemia

Biochemical assessment

The degree of biochemical hyponatraemia is classified in three groups:

- Mild: 130–135 mmol/L
- Moderate: 125–129 mmol/L
- Profound: <125 mmol/L

Clinical assessment

Severity of clinical presentation may not match the degree of hyponatraemia: profound hyponatraemia may be symptom-free, while some patients with moderate

biochemistry may have significant neurological symptoms and signs. For the purposes of this guidance, symptoms have been classified as follows:

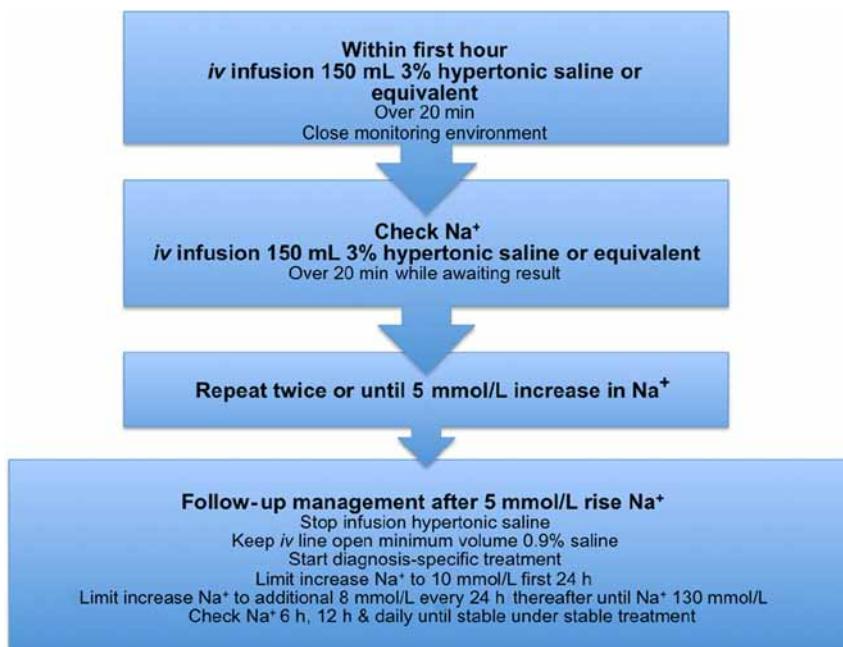
- Severe symptoms: vomiting, cardiorespiratory arrest; seizures; reduced consciousness/ coma (Glasgow Coma Scale ≤8)
- Moderately severe symptoms: nausea without vomiting; confusion; headache
- Mild or absent symptoms

The clinical status of the patient reflects the balance of a number of factors:

- Biochemical degree of hyponatraemia
- Rate of development of hyponatraemia
- The intrinsic ability of the central nervous system to adapt to changing osmolar stress
- The range and degree of co-morbidities

Severe symptoms are unlikely with serum sodium >130 mmol/L and alternative causes of neurological dysfunction should be considered in this context.

Management decisions should be made on the basis of presenting clinical symptoms and signs rather than the degree of hyponatraemia (1, 2).



Treatment of the patient presenting with severe or moderately severe symptomatic hyponatraemia

See Fig. 1 for the recommended approach. Patients with severe symptoms require immediate treatment with hypertonic saline, irrespective of the cause of the hyponatraemia.

The decision to treat with hypertonic fluid and the supervision of treatment with hypertonic fluid should be the responsibility of a senior clinician with appropriate training and experience. The aim is to achieve a 5 mmol/L rise in serum Na^+ within the first hour, reducing immediate danger from cerebral oedema

while minimising the risk of over-rapid correction and osmotic demyelination.

If the clinical status of the patient does not improve after a 5 mmol/L rise in serum Na^+ in the first hour, we recommend taking additional steps as outlined in Fig. 2.

Managing over-correction of serum Na^+

Over-correction of serum Na^+ risks precipitating osmotic demyelination. The condition underlying the patient's presentation with hyponatraemia may well change during the first 24 h with cause-specific intervention; the situation is dynamic. If the limit of 10 mmol/L in the first 24 h or 18 mmol/L in the first 48 h of treatment

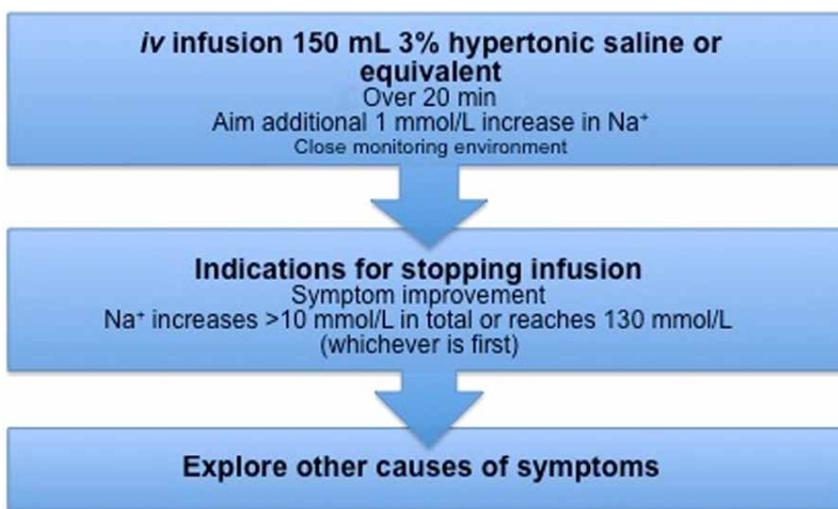
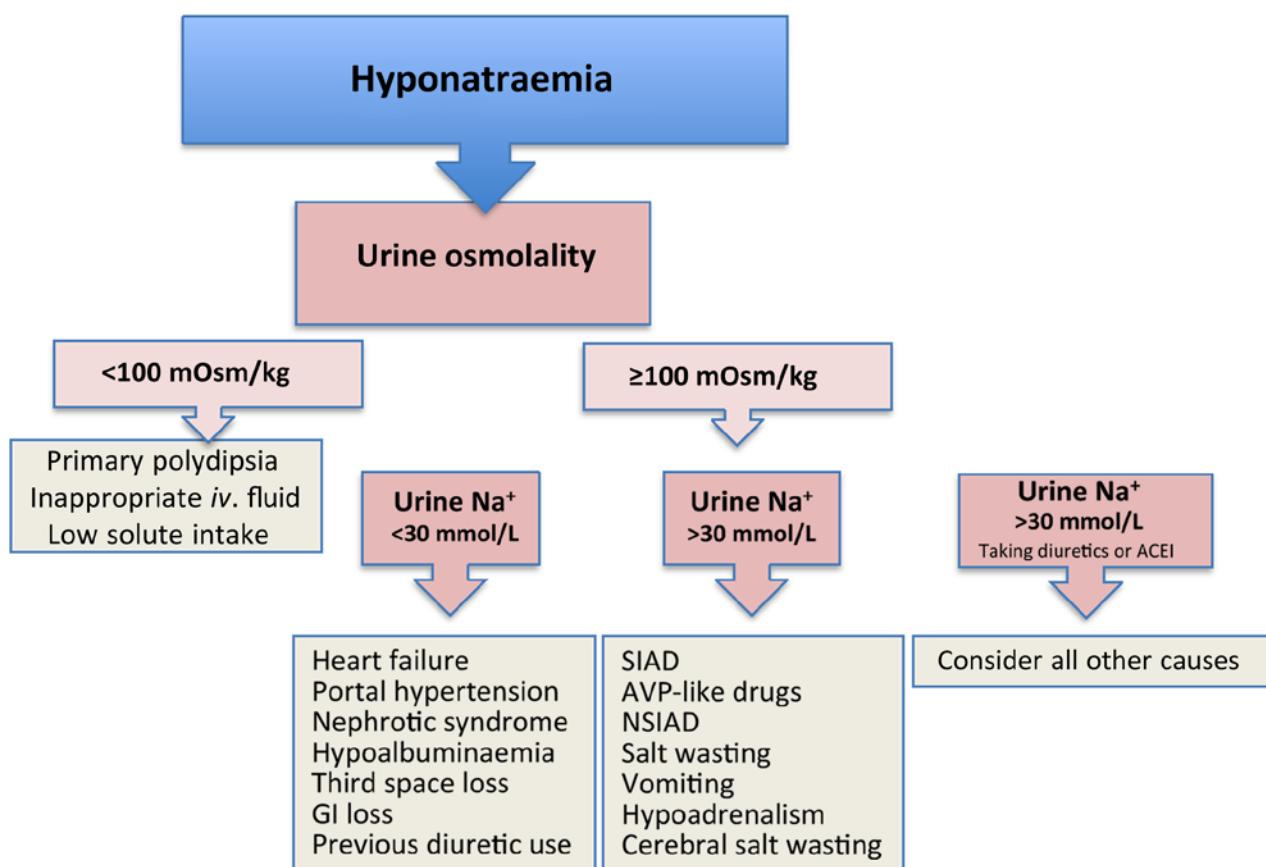


Figure 2

Patients with hyponatraemia treated with hypertonic saline. Recommended approach if no improvement following 5 mmol/L rise in Na^+ in the first hour.

**Figure 3**

Diagnostic algorithm for patients presenting with hyponatraemia. For use following emergency treatment.

is exceeded, hypertonic fluid should be stopped. We recommend consulting a clinician with experience in managing over-correction who may wish to consider introducing hypotonic fluid, with or without concurrent anti-diuresis (3).

The doctors concerned must make the management plan for an individual patient.

Differential diagnosis of hyponatraemia following emergency treatment

Measurement of urine osmolality and urine Na^+ concentration are central to defining the aetiology of hyponatraemia. An algorithmic approach to establishing the cause of hyponatraemia, in-line with other recent guidance, is outlined in Fig. 3 (1, 4).

References

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