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This Medicines Information Leaflet is produced locally to optimise the use of medicines by encouraging prescribing that is safe, clinically appropriate and cost-effective to the NHS.

Guidelines for the Management of Acute Severe Symptomatic Hyponatraemia in Adults

This Medicines Information Leaflet focuses on the management of acute symptomatic hyponatraemia. It is the most commonly seen electrolyte disturbance affecting both inpatients, affecting 30% of hospitalised patients, and also in the community. The majority of cases of hyponatraemia are mild and asymptomatic; however rapid changes to serum sodium levels can cause severe neurological disorders, which should be treated as a medical emergency.

Box 1: Medical Causes of Hyponatraemia

There are many causes of hyponatraemia some of these may include:

- Renal losses – Such as diuretics, hypoadrenalism and sodium-losing nephropathies
- Non-renal losses- diarrhoea, vomiting, burns, peritonitis, pancreatitis
- Syndrome of Inappropriate Antidiuretic Hormone (SIADH)
- Oedematous States causing the dilution of sodium within the body – Examples include severe renal impairment, congestive heart failure, cirrhosis
- Hypothyroidism
- Over-drinking – psychogenic polydipsia

The primary aim of treating acute symptomatic hyponatraemia is to increase serum sodium. However, any treatable or reversible conditions should be reviewed at the earliest opportunity. There are also a number of drugs which may cause a reduction in serum sodium (see box 2), these should be reviewed and when appropriate, discontinued at the earliest opportunity.

Diagnosis of Hyponatraemia

To fully assess the extent and cause of hyponatraemia a full clinical history and investigations should be undertaken. This should include an assessment of patient's volume status and serum osmolality (see Figure 1).

It is also important to exclude **pseudohyponatraemia**, this is when abnormally high concentrations of lipids or proteins in the blood interfere with the accurate measurement of sodium.

1. Symptoms of Severe Hyponatraemia

Severity	Symptom
Moderately severe	Nausea without vomiting Confusion Headache
Severe	Vomiting Cardiorespiratory distress Abnormal and deep somnolence Seizures Coma/ Altered GCS

2. Time of Development

Classification	Duration of hyponatraemia
Acute	Onset less than 48 hours
Chronic	Documented to exist for at least 48 hours (If the onset cannot be classified consider as chronic)

A review of the patient's medical history will reveal the time length that the patient may have been hyponatraemic. This may include polydipsia or recent medication which may cause a sudden decrease in serum sodium e.g. diuretics (See Box 2)

Management of acute symptomatic hyponatraemia

Acute symptomatic hyponatraemia (onset less than 48 hours and symptoms of severe hyponatraemia) is a medical emergency, and patients should be treated in a setting where the patient can be monitored and managed appropriately, consider advice from critical care. A senior endocrinologist (Consultant or Registrar) should be consulted as soon as possible.

Treatment involves the use of hypertonic saline to gradually correct the hyponatraemia, with the goal of ensuring that the sodium level does not rise by more than **6 mmol/L in the first 6 hours or 10 mmol/L in the first 24 hours**. Rapid over-correction leads to a risk of osmotic demyelination syndrome.

- Correct serum sodium with 150 mL of intravenous Sodium Chloride 2.7% via a central line over 15 minutes.
- If there is no clinical improvement, repeat the dose after 20 minutes.
- Check serum sodium at 6, 12, 24, and 48 hours to ensure that overcorrection (serum sodium rise of 10mmol/L or more in 24 hours or less) has not occurred.
- The serum sodium does not need to be normalised with hypertonic saline; an increase of 4–6 mmol/L often leads to major clinical improvements.
- Other electrolyte disturbances should be considered when treating hyponatraemia. The most common electrolyte of note would be potassium.

For the management of acute symptomatic hyponatraemia see algorithm below, Figure 1. For patients who are asymptomatic, refer to Figure 2.

Parenteral Hypertonic Sodium Chloride Preparations

The hypertonic solution available within the OUH, is;

- Sodium chloride 2.7% containing 225mmol of sodium in 500mL

Sodium chloride 2.7% is available from pharmacy and should be ordered as a **controlled drug.**

Administration (via volumetric infusion pump)

For the administration of 150mL over 15 minutes. set infusion rate of 600mL per hour and a total volume to be infused of 150mL.

Intravenous Access

Sodium Chloride 2.7% should be given via a **central line**.

However, if no central access is available it may be given via a **large peripheral vein.** Consultants must balance the need to treat against the high risk of local pain and phlebitis which can occur. Extravasation causes tissue necrosis.

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References:

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2. Spasovski et al. Clinical Practice Guideline on Diagnosis and Treatment of Hyponatraemia. Eur J Endocrinol March 1, 2014 170 G1-G47
3. Grant et al, The diagnosis and management of inpatient hyponatraemia and SIADH, (Not currently published)
4. Ramrakha, Moore and Sam, Oxford Handbook of Acute Medicine (3rd edition) Chapter 9, endocrine emergencies, 2010.

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Box 2: Common Drug Causes of Hyponatraemia

Anticancer agents: Vinca alkaloids (e.g. vincristine and vinblastine), Platinum compounds (cisplatin, carboplatin), Alkylating agents (Intravenous cyclophosphamide, melphalan, ifosfamide).

Antidepressants: Most classes of antidepressants may cause low serum sodium.

Anti-epileptics: Carbamazepine, Oxcarbazepine and Sodium Valproate. **Antiepileptics should not be discontinued without considering clinical impact of discontinuation, contact neurology for advice.**

Anti-hypertensives: Angiotensin-converting enzymes (ACE) inhibitors, Amlodipine.

Diuretics: Thiazides, Indapamide, Amiloride, Loop Diuretics.

Proton Pump Inhibitors

Figure 1: Management of Acute Severe Symptomatic Hyponatraemia

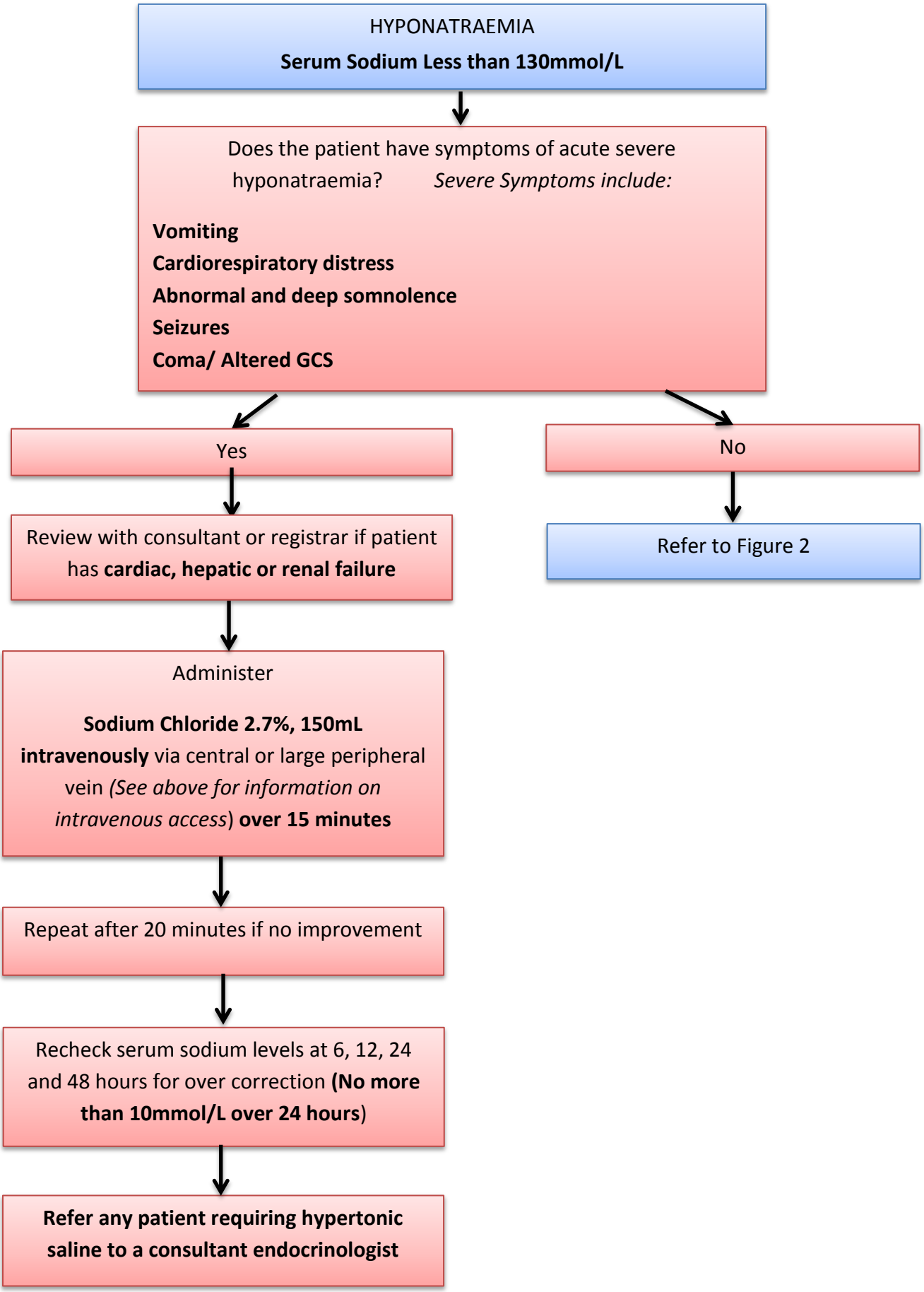

Figure 2: Management of Acute Severe Hyponatraemia without Symptoms