Clinical Guideline

Hyponatraemia Prevention and Management

1. Introduction

Hyponatraemia is defined as a serum sodium concentration below 135mmol/L. Hyponatraemia may develop due to either an excess of water (dilution) or depletion in sodium. This may occur due to excess administration of fluid (especially glucose 5%), retention of fluid or an abnormal loss of sodium.

Symptoms of hyponatraemia:

Mild: lethargy, anorexia and nausea

Severe: agitation, disorientation, depressed reflexes, focal neurological deficits, Cheyne-Stokes respiration, seizures, cerebral oedema and coma.

All causes of hyponatraemia require prompt recognition and treatment.

Risk factors for developing hyponatraemia include:

- Postoperative
- Alcohol excess
- Malnutrition
- Psychiatric conditions
- Elderly
- Hypovolaemia
- Burns
- Medication

CAUTION

Over rapid correction of hyponatraemia may lead to central pontine myelinolysis (CPM), especially when serum sodium concentration is less than 120mmol/L. Symptoms are usually irreversible and include: dysarthria, dysphagia, hallucinations, tremor, paraparesis or quadriparesis, behavioural disturbances, lethargy, confusion, disorientation, coma or seizures. In severe cases patients may appear ‘locked in’, they are awake but unable to move or communicate.

The NPSA issued a signal alert in February 2012 following a series of national incidents. The incidents related to errors in the strength, volume or rate of administration of sodium chloride infusion. The alert requires that the risk of CPM is highlighted to health care professionals, that serum sodium concentrations are monitored every 2 to 4 hours when correcting sodium levels, that a maximum rate of change of serum sodium concentration is stated and that patients are closely monitored (clinical state, central nervous system observations and fluid balance).

2. Prevention of Hyponatraemia

i. Use enteral route where possible
ii. Review intravenous fluid prescriptions daily and discontinue as soon as possible.
iii. Hypotonic fluids (such as glucose 5%, sodium chloride 0.18% with glucose 4% and sodium chloride 0.45%) should be used with care.
iv. Daily measurement of urea and electrolytes for all patients receiving parenteral fluids.
v. Accurate fluid balance charts.

3. Assessment

Determine whether the hyponatraemia is due to depletion (gastro intestinal losses, renal or adrenal causes) or dilution (excess administration of fluids or syndrome of inappropriate antidiuretic hormone (SIADH) which may be due to infection, cardiovascular, respiratory, central nervous system or drug therapy).

The distinction between depletional hyponatraemia and dilutional may be made by reviewing biochemical results; serum potassium, urea and albumin likely to be low in dilutional hyponatraemia. Blood pressure is likely to be normal or high in dilutional hyponatraemia and is often low in depletional hyponatraemia.

4. Investigations

- Urea and electrolytes
- Plasma and urine osmolality
- Glucose
- Thyroid function tests
- Urinary sodium concentration
- Consider synacthen test to exclude Addison's disease

5. Treatment

Treatment will depend on the cause, severity and duration. It is essential to have a comprehensive history, examination and laboratory tests in order to determine the cause.

5.1 Depletional hyponatraemia

- Correct the cause of hyponatraemia
- Reduce gastro intestinal losses by appropriate treatment of diarrhoea and vomiting.
- Allow correction of hyponatraemia by oral intake.
- If necessary correct with sodium chloride 0.9%.
- Treat Addison's disease with a stat dose of hydrocortisone 50mg IV or IM and then hydrocortisone, 20mg mane and 10mg teatime orally. Some patients may require a prolonged course of intravenous hydrocortisone, an endocrinology review is recommended. Fludrocortisone may also be required.
- Check serum sodium concentration at least every 2 to 4 hours
- Raising sodium levels too rapidly may cause harm.
- Severe hyponatraemia (usually only if serum sodium concentration is less than 120mmol/L) associated with seizures or loss of consciousness must be reviewed by a consultant.
- Consider a critical care review.
- Hypertonic sodium chloride is very rarely needed and is potentially hazardous. **It can only be given under the direct supervision of a consultant**. A suggested dose is 100ml sodium chloride 1.8% over 1 hour. Note that sodium chloride 1.8% is only available as a 500ml polyfusor. The infusion must be monitored to ensure that no more than 100ml of the polyfusor is given.
- Aim to increase sodium levels by 0.5 to 1.0 mmol/L per hour to a maximum of 12mmol/L in a 24 hour period. In high risk patients a slower correction may be necessary.

5.2 Dilutional hyponatraemia

- Restrict fluid, potentially to less than 1Litre per day depending on severity
- Stop causative agents e.g. drugs and hypotonic fluids.

- Administration of hypertonic sodium chloride will only be required in exceptional circumstances and the decision should be made by a consultant, see above for dose.
- Aim to increase sodium levels by 0.5 to 1.0 mmol/L per hour to a maximum of 12mmol/L in a 24 hour period. In high risk patients a slower correction may be necessary.
- Check serum sodium concentration at least every 2 to 4 hours.
- Raising sodium levels too rapidly may cause harm.

5.2.1 SIADH

In SIADH patients may have hyponatraemia due to respiratory infection, cardiovascular disease, central nervous system pathology or other causes. Serum osmolality will be low e.g. less than 275mOsmol/Kg and urine osmolality will be concentrated e.g. above 300mOsmol/Kg.

- Treatment is fluid restriction to less than 1 litre per day
- Address the underlying cause
    - Treatment of infection
    - Cessation of offending drugs.

Drugs commonly causing hyponatraemia include:

- **Diuretics**
- **Antidepressants especially SSRIs**
- ACE inhibitors: enalapril and captopril.
- Anticonvulsants: carbamazepine, sodium valproate.
- Antidiuretic hormone analogues: desmopressin, vasopressin, terlipressin.
- Antipsychotic drugs: haloperidol, clozapine
- COX-2 inhibitors: celecoxib
- Proton pump inhibitors: omeprazole, pantoprazole.

Please note this is not an exhaustive list; please contact pharmacy for advice if hyponatraemia is thought to be drug related.

In patients with persistent SIADH prolonged therapy with **demeclocycline** may be required. This should be consultant led.

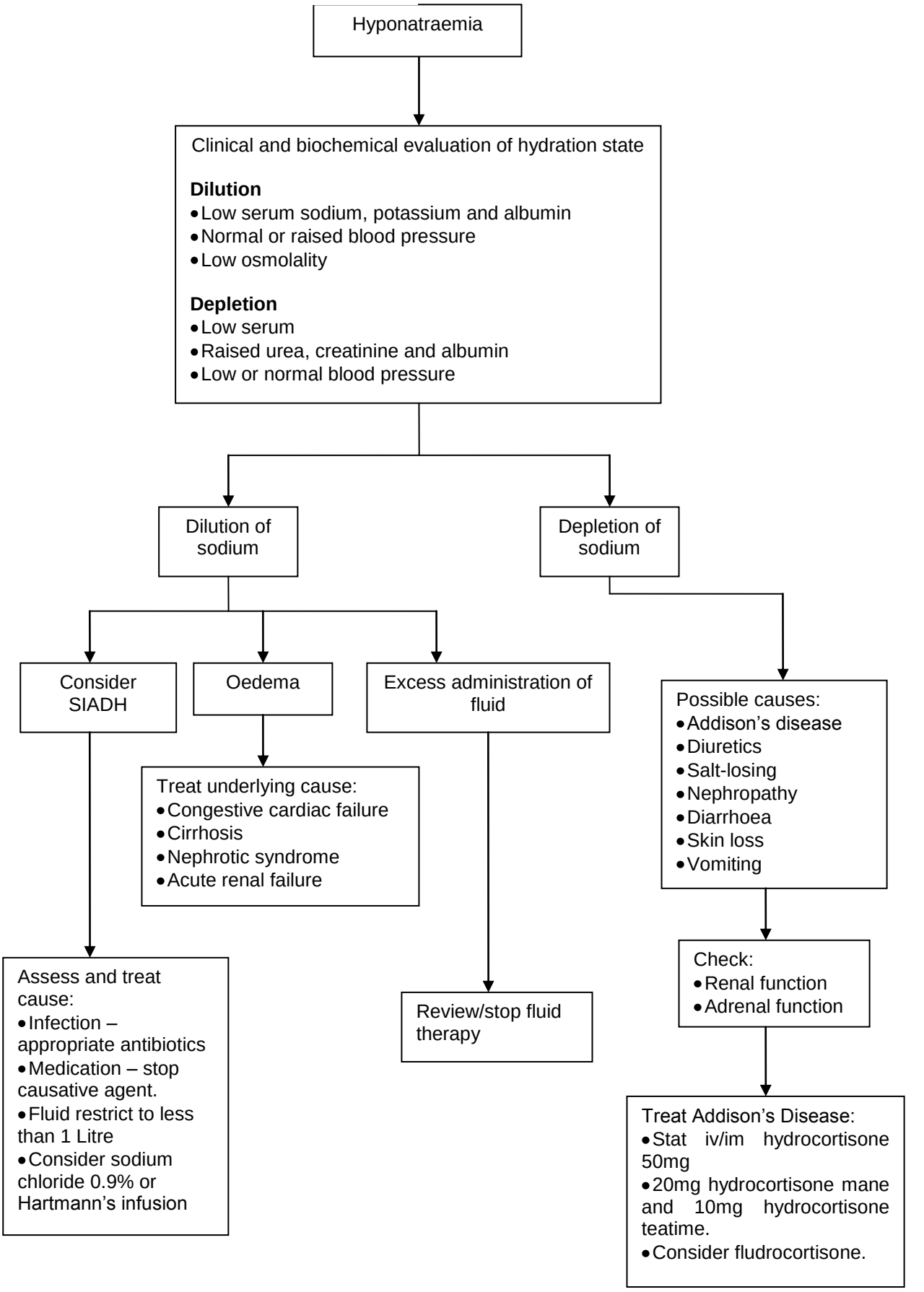
6. Monitoring

- Clinical state including: pulse, blood pressure, nausea, lethargy, apathy.
- Central nervous system observations
- Fluid balance (daily review by an experienced member of medical staff)
- In symptomatic patients or following correction of hyponatraemia serum sodium should be measured every 2 to 4 hours.
- A consultant should be involved in the treatment of severe hyponatraemia.

4. References

1. The Guidelines and Audit Implementation Network (GAIN) (Northern Ireland) guidance on Hyponatraemia in adults (on or after 16th birthday) February 2010. Available at: http://www.gain-ni.org/Publications/Guidelines/Hyponatraemia_guideline.pdf
2. The National Patient Safety Agency. Signal alert. Risk of harm from CPM syndrome following rapid correction of sodium. February 2012.
3. BNF edition 63 March 2012
4. Sterns R.H et al. UpToDate Treatment of hyponatraemia: Syndrome of inappropriate antidiuretic hormone secretion (SIADH) and reset osmostat. Feb 2012.
5. Fourlanos S et al. Managing drug induced hyponatraemia in adults. Australian Prescriber. Volume 26 No 5. 2003. 114-117.

Flow chart for management of hyponatraemia



Maximum rate of change of serum sodium concentration is 12mmol/L in 24 hours