University Hospitals of Derby and Burton NHS Foundation Trust

Electrolyte Disturbances - SDU - Full Clinical Guideline

Reference no.: CG-STEP/2019/003

Contents

Summary

Sodium

- *A rise or fall in plasma level of ≥ 20 mmol / 24 hours is considered significant.*
  - ❖ *Slow correction ≤ 8 mmol / 24 hours is the underlying management principle.*

Potassium

- *Hyperkalaemia is classified as mild (5.1-5.9 mmol/L), moderate (6.0.-7.0 mmol/L), or severe (≥ 7.0 mmol/L).*
  - ❖ *Moderate hyperkalaemia requires glucose / insulin infusion, whilst severe hyperkalaemia requires dialysis.*
- *Hypokalaemia is classified as mild (3.0-3.5 mmol/L), moderate (2.5-3.5 mmol/L), or severe (< 2.5 mmol/L).*
  - ❖ *Mild and moderate hypokalaemia should be managed by oral supplements, or by increased intravenous maintenance KCl as appropriate.*
  - ❖ *Severe hypokalaemia should be managed with an intravenous replacement regime. Hypomagnesaemia and hypophosphataemia may co-exist with hypokalaemia.*

Magnesium

- *Hypermagnesaemia is often iatrogenic.*
- *Hypomagnesaemia is often asymptomatic.*
  - ❖ *Asymptomatic patients do not require treatment if total serum level ≥ 0.5 mmol/L.*
  - ❖ *Asymptomatic patients require treatment if total serum level ≤ 0.4 mmol/L.*
  - ❖ *Symptomatic patients require intravenous treatment at any serum level.*

Calcium

- *Decisions to treat hypocalcaemia should be based on the presence of symptoms of hypocalaemia and serum ionized calcium level measured on a blood gas machine. Do not treat corrected total plasma calcium level in the absence of symptoms.*

Phosphate

- *Hyperphosphataemia may be iatrogenic and causes symptoms via associated hypocalcaemia which may itself be severe and require intravenous replacement.*
- *Hypophosphataemia is classified as mild (0.65 – 0.79 mmol/L), moderate (0.32 – 0.65 mmol/L), or severe (≤ 0.32 mmol/L).*
  - ❖ *Intravenous phosphate replacement should not be given until plasma levels are < 0.4 mmol/L, or unless re-feeding syndrome is considered to be a significant risk factor.*

Re-feeding Syndrome

Ask for an assessment from the Nutrition Team before re-starting feeding in any patient "nil by mouth" for 5 days or more.

Aim and Scope

This aim of this document is to guide the rational management of electrolyte disturbances that occur in postoperative patients on the Step Down Unit.

1. Introduction

Electrolyte management is often misunderstood and poor practice can be a cause of postoperative morbidity. Clinicians are often guilty of treating numbers rather than patients thereby attracting unnecessary complications.

The "*Prescribing Postoperative Fluids"* guidelines are designed to limit the likelihood of patients developing "hypo" or "hyper" serum electrolyte levels on SDU. The purpose of this document is to offer guidance for the situation where such a disturbance has occurred.

Serum electrolyte levels rarely reflect the status of total body stores, and short-term intravenous administration will not correct total body deficiency. Therefore it is better to manipulate serum electrolytes by continuous enteral administration where practicable, rather than by an intermittent intravenous regime. Discuss the availability of suitable preparations with the Critical Care pharmacist.

2. Sodium

2.1 Hypernatraemia

Definition: serum sodium >145 mmol/L.

Hypernatraemia causes lethargy, weakness and irritability, and may progress to twitching, seizures and coma when serum sodium > 158 mmol/L, or when there has been a rise > 20mmol/L in 24hrs. Brain tissue shrinkage can result in intracranial haemorrhage.

- Causes relevant to SDU:
    - Fluid loss without replacement (diarrhoea, excessive sweating, hyperventilation)
    - Diuretic therapy
    - Overzealous treatment with 0.9% saline solutions (antibiotic infusions, gelofusine and colloid challenges)
    - GI dysfunction and SBO
- Management:
    - The aim is to identify and correct any underlying cause.

- Stop water loss if relevant (antiemetics, antidiarrhoeals)
- Replace fluid losses with Hartmann's solution in addition to maintenance 4% glucose / 0.18% saline solution.
- Measure serum sodium 1 hour after first litre of replacement – if significant fall of > 8mmol alternate fluid requirements with 0.9% saline.
- Measure serum sodium at least twice daily – do not allow a fall of > 8mmol / 24 hrs. If this happens alternate fluid requirements with 0.9% saline.

2.2 Hyponatraemia Definition: serum sodium ≤ 130 mmol/L

- Mild – 125-135 mmol/L
- Moderate -120-125 mmol/L
- Severe – Below 120 mmol/L

Hyponatraemia causes anorexia, nausea, headache, cramps, altered mental status ranging from confusion and seizures to coma, and potentially death from raised intracranial pressure / cerebral herniation.

There is no correlation between serum level and symptoms. The rate of change is more important than the actual number ie a fall > 20mmol/L in 24hrs is likely to be symptomatic. Hyponatraemia is defined as acute when it has developed in ≤ 48 hours, and chronic thereafter as compensatory changes may have occurred and symptoms may be absent or mild. This classification determines the rate at which correction should be attempted. Rapid correction of any type of hyponatraemia may cause an osmotic demyelination syndrome. Discuss hyponatraemia with the consultant supervising SDU before commencing any management alterations.

Causes relevant to post-operative population:

- Iatrogenic excessive water gain with hypervolaemia (drugs infused in 5% glucose, inappropriate maintenance fluid infusion rates)
- TURP syndrome with hypervolaemia
- Excessive sodium and water loss secondary to;
  - Vomiting and diarrhoea
  - Diuretic therapy
  - Diabetic hyperosmolar states
  - GI dysfunction/SBO
- Excessive oral water intake and renin –angiotensin system activation

- Management:

The aim is to identify and correct any underlying cause.

- If acute hyponatraemia with GCS < 12 / seizures refer to ICU for ABCDE management and possible hypertonic saline infusion.

- If acute/chronic hyponatraemia with mild symptoms;

    - Exclude diabetic hyperosmolar state.
    - Stop water administration – ie maintenance 4% glucose / 0.18% saline solution, ask pharmacy to reformulate IV drugs, switch to 1 litre oral intake if feasible.
    - If hypervolaemic - restrict fluid to 1 litre oral intake if feasible or use Hartmann's solution for intravenous maintenance, give diuretics.
    - If hypovolaemic, replace losses with Hartmann's solution.
    - Measure serum sodium at least twice daily – do not allow a rise of > 8mmol / 24 hrs. If this happens discuss with the supervising consultant and alternate fluid requirements with 4% glucose / 0.18% saline solution.
    - Low dose diuretics 10/20mg BD IV furosemide for 2 days

3. Potassium

3.1 Hyperkalaemia

Definition: serum potassium > 5.3 mmol/L

- Mild – 5.5 – 5.9
- Moderate – 6.0 – 6.4 mmol/L
- Severe - >6.5 mmol/L or if K+ > 6 with ECG changes or symptoms

Symptoms include weakness, flaccid paralysis, paraesthesiae, depressed tendon reflexes and respiratory insufficiency. Most concern orientates around cardiac rhythm dysfunction, including peaked T waves, prolonged PR interval, QRS widening and fatal arrhythmias. Active management should be started when serum level exceeds 6.0 mmol/L. The serum potassium level is usually determined by the relationship between intake and urinary excretion.

- Causes relevant to SDU:

    - Pseudohyperkalaemia – repeat blood sample to exclude artefact caused by haemolysis
    - Drug Therapy (NSAIDs, "Prils", "Sartans", β blockers)
    - Excessive intake in patients with renal impairment
    - Acidosis (acute renal failure, sepsis, under-resuscitation)
    - Massive blood transfusion / haemolysis
    - Rhabdomyolosis / compartment syndrome / tumour lysis
    -

- Refer to Trust guidance for other medical causes and management

- Management

    - The aim is to identify and correct any underlying cause.

    - Mild – stop administration of potassium and promote a diuresis.

- Moderate – commence an infusion of 25g glucose and 10U insulin over 15-30 mins. Use hypertonic glucose only if central venous access is available - do not delay treatment by inserting such access. Administer ion exchange resins orally / PR as appropriate. Give 5mg salbutamol nebuliser repeated x 3.

25g glucose can be given as	*50mls x 50%*	*Hypertonic (central line)*
	125mls x 20%	*Hypertonic(large vein)*
	250mls x 10%	*Hypertonic(large vein)*
	500mls x 5%	*Isotonic(small vein)*

- Severe – this may be an indication for renal replacement therapy. Protect myocardium with 10ml calcium gluconate 10% iv over 2 mins - may be repeated. Caution digoxin (see Trust guidance). Commence an infusion of glucose and insulin as above - hypertonic solutions may be given peripherally in this instance. If the blood glucose is > 15 mmol/l, insulin alone can be given with careful BM monitoring. Give 5mg salbutamol nebuliser or intravenous salbutamol infusion. Refer immediately to a senior ICU clinician. Complete the Trust's EMERALD hyperkalaemia care bundle.

- If any evidence of ECG / rhythm disturbance, administer 10mls x 10% calcium gluconate solution intravenously.

3.2 Hypokalaemia Definition: serum potassium ≤ 3.5 mmol/L

- Mild – 3.0 – 3.4 mmol/L
- Moderate – 2.5 – 3.0 mmol/L
- Severe - < 2.5 mmol/L

Mild hypokalaemia usually causes no symptoms, while moderate hypokalaemia may result in confusion, disorientation, weakness and muscle cramps. Severe hypokalaemia may result in extreme weakness of the body and, on occasion, flaccid paralysis and respiratory insufficiency. The most significant result of severe hypokalaemia is development of cardiac arrhythmia leading to cardiac arrest. This is particularly likely in the presence of hypomagnesaemia which should be excluded. ECG findings in hypokalaemia include decreased T-wave amplitude, depression of the ST segment (≥ 0.5 mm), the appearance of U waves (amplitude >1 mm and >T wave in the same lead). U waves are often seen in the lateral precordial leads V4 to V6.

- Causes relevant to SDU:
  - Drug Therapy (diuretics, steroids, β_2 agonists, insulin)
  - Excessive loss (vomiting, diarrhoea, NG losses, GI ileus)
  - Inadequate maintenance administration

- Management:

- The aim is to identify and correct any underlying cause.
- Mild & Moderate – start oral / enteral potassium supplement if feasible

Drug	Dosage	Provides (mmol)
Sando K	*2 tabs TDS*	*72*
Slow K	*2 tabs TDS*	*48*
Kay-Cee-L	*20mls TDS*	*60*

- Mild & Moderate – if NBM, increase KCl to 40mmol/L in maintenance fluid regime.
- The situation in patients with renal failure is more complex. Hypokalaemia occurring immediately after haemodialysis may be transient and correct itself. Hypokalaemia in those with end-stage renal should not be treated without first discussing the case with the renal team.
- Severe – this is an indication for intravenous replacement therapy which must take place using ECG monitoring. Remember that the risks of iatrogenic hyperkalaemia are potentially more serious than those of hypokalaemia.
- Cardiac arrhythmias are another indication for intravenous replacement therapy. Remember to check the serum magenesium level and correct accordingly as hypomagnesaemia will make fatal arrhythmia more likely.

It is preferable to give low volume potassium infusions via a central venous catheter, but on balance of risks it is rarely justified to insert such a device in this circumstance. Frequent observation of the site of potassium infusion is necessary during peripheral administration.

- ✓ 20 mmol KCl will raise serum level ≈ 0.25 mmol/L.
- ✓ Potassium solutions must always be given by volumetric pump or syringe driver.
- ✓ The maximum usual amount to be infused for a severe deficit replacement is 40 - 80 mmol over 4 hours.
- ✓ The rate of administration should not exceed 10-20 mmol/hr.
- ✓ If malignant ventricular arrhythmia is present, 10 mmol may be given over 5 minutes after discussion with the supervising consultant.
- ✓ Only solutions made by pharmacy staff may be administered on SDU. The infusion should be made up by pharmacy and delivered according the ICU potassium monograph.
- ✓ Potassium infusions must not be given to oliguric / anuric patients.

4. Magnesium

Caution should be taken in interpreting and treating results from patients who have low serum magnesium and albumin levels, as they may have normal ionised magnesium concentrations (0.45 – 0.67 mmol/L) and be asymptomatic.

4.1 **Hypermagnesaemia** Definition: total serum magnesium > 1.2 mmol/L

Hypermagnesaemia is associated with muscular weakness, paralysis, ataxia, altered GCS, vasodilation, hypotension, respiratory insufficiency and cardiac arrhythmias. Symptoms begin at serum levels >2.1 mmol/L and serious arrhythmias at >5.0 mmol/L. Asystole occurs at >12.0 mmol/L.

- Causes relevant to SDU:
  - Iatrogenic administration
  - Treatment of pre-eclampsia
  - Renal failure
- Management
  - The aim is to identify and correct any underlying cause.
  - Remove source of administration.
  - If renal function normal, promote diuresis with saline infusion and furosemide (1mg/kg).
  - If arrhythmia present, give 10mls 10% calcium gluconate (2.26 mmol) intravenously over 3-5 minutes.
  - Refer symptomatic patient to senior ICU clinician as ABC management and renal replacement therapy is often required.

4.1 Hypomagnesaemia Definition: total serum magnesium < 0.7 mmol/L

Hypomagnesaemia is associated with muscular tremors and fasiculations, ocular nystagmus, tetany, ataxia, altered GCS and seizures. ECG changes can mimick hypokalaemia and progress to malignant cardiac arrhythmias such as Torsade de Pointes. Patients can develop Trousseau's and Chvostek's signs even in the presence of a normal ionised serum calcium concentration Hypomagnesaemia can induce hypokalaemia and hypocalcaemia which should be looked for in blood tests.

- Causes relevant to SDU:
  - Increased losses from GIT or kidneys
  - Decreased absorption from GIT or kidneys
  - Drug therapy (diuretics, aminoglycoside, amphotericin)
  - Alcoholism
  - Re-feeding syndrome
- Management:
  - The aim is to identify and correct any underlying cause.
  - Oral magnesium salts may cause diarrhoea and are usually avoided.
  - Asymptomatic patients do not require treatment if total serum level ≥ 0.5 mmol/L.
  - Asymptomatic patients require treatment if total serum level ≤ 0.5 mmol/L.
  - Symptomatic patients require intravenous treatment at any serum level.
  - Intravenous treatment is required when an arrhythmia is present.
    - ✓ Without arrhythmia give 10 mmol magnesium sulphate in 100mls 0.9% saline over 60 minutes.
    - ✓ If arrhythmia present give 10 mmol magnesium sulphate in 100mls 0.9% saline over 10 minutes.
  - Repeat serum level 2 hours after any intravenous intervention, and repeat dose as necessary.
  - Refer symptomatic patients to a senior ICU clinician as ABC management is often required

5. Calcium

Calcium is another vital cation. In plasma, calcium exists as a physiologically active ionized fraction (50%), a protein-bound component (40%), and attached to anions (10%). The normal ionized calcium concentration is 1.03 – 1.30 mmol/L, and can be measured by the ICU blood gas analyser. It is best to make treatment decisions based on the ionized serum level and the presence of symptoms.

5.1 Hypercalcaemia Definition Normal 2.20 – 2.60 mmol/L
Mild hypercalcaemia 2.61 – 3.00 mmol/L
Moderate hypercalcaemia 3.00 – 3.50 mmol/L
Severe hypercalcaemia > 3.5mmol/L

Hypercalcaemia is associated with a wide range of non-specific symptoms including; nausea and vomiting, alterations of mental status, abdominal pain, constipation, lethargy, depression, weakness and vague muscle/joint aches, polyuria, polydipsia, nocturia and headache.

- Causes relevant to SDU:
    - Iatrogenic administration.
    - Severe metastatic cancer with bony involvement.
- Management
    - The aim is to identify and correct any underlying cause.
    - Remove any source of administration.
    - If renal function normal, promote diuresis with saline infusion and furosemide (1mg/kg).
    - Refer symptomatic patient to senior ICU clinician as ABC management and renal replacement therapy is often required. An urgent endocrinology opinion should be sought.
    - Refer to Trust Guideline

5.1 Hypocalcaemia Definition: total serum calcium < 2.2 mmol/L or serum ionized calcium < 1.0 mmol/L

Symptoms occur when the ionized component becomes < 1 mmol L^{-1}, and is seen clinically as tetany and by prolongation of the QT_c interval and ST segment changes in a 12 lead ECG. The diagnosis can be confirmed by eliciting Trousseau's or Chvostek's signs. Cadiovascular collapse with refractory hypotension and dysrhythmias may occur in severe cases.

- Causes relevant to SDU:
    - Hypoalbuminaemia
    - Hypomagnesaemia
    - Hypophosphataemia
    - Massive transfusion
    - Parathyroid/thyroid surgery
    - Bilateral neck dissections
    - Pancreatitis

- Malabsorption following certain types of abdominal/pancreatic surgery

- Management:
  - The aim is to identify and correct any underlying cause.
  - Asymptomatic patients do not require treatment if ionized serum level ≥ 1.0 mmol/L.
  - Symptomatic patients require intravenous treatment at any serum total / ionized calcium level.
  - Intravenous treatment is only indicated for established tetany, abnormal QT_c interval, and as a protective measure in hyperkalaemia with ECG changes.
  - The intravenous dose is 10 mmol of calcium gluconate given in 100mls 0.9% saline over 5-10 mins,
  - If arrhythmia present, give 10-20mls 10% calcium gluconate (2.26mmol) intravenously over 3-5 minutes. with ECG monitoring
  - Followed by IV infusion of 22.5mmol calcium gluconate in 1000ml Sodium Chloride 0.9% or Glucose 5% at a rate 50-100 ml/hour.* Rate advised by endocrinology. Titrate according to levels. Give via a large vein and monitor for signs of phlebitis. **Urgent referral to Endocrinology**
  - Repeat serum ionized level 30 minutes after any intravenous intervention, and repeat dose as necessary.
  - Refer symptomatic patients to a senior ICU clinician as ABC management is often required.
  - Refer to Trust guideline

6. Phosphate

6.1 **Hyperphophataemia** Definition: serum phosphate > 1.5 mmol/L

Phosphate and calcium metabolism are interlinked. Hyperphosphataemia causes hypocalcaemia by precipitating calcium, decreasing vitamin D production, and interfering with parathyroid hormone-mediated bone resorption. Severe life-threatening hypocalcaemia may result from phosphate administration. Signs and symptoms of acute hyperphosphataemia are due to the effects of hypocalcaemia. Patients commonly complain of muscle cramping secondary to low calcium levels. This may progress to tetany, delirium, and seizures.

- Causes relevant to SDU:
  - Renal failure
  - Trauma
  - Tumour lysis
  - Iatrogenic administration

- Management:

- The aim is to identify and correct any underlying cause.
- Patients with symptomatic hypocalcaemia should have the ionised calcium restored to the normal range.
- Promote a diuresis using 0.9% saline and furosemide (1mg/kg).
- An urgent renal opinion should be obtained.
- Asymptomatic patients do not require intravenous calcium. Instead promote a diuresis and obtain a renal opinion.

6.2 Hypophophataemia Definition: Normal 0.8 – 1.45mmol/l
Mild hypophosphataemia 0.65 - 0.79mmol/l
Moderate hypophosphataemia 0.32 – 0.64mmol/l
Severe hypophosphataemia < 0.32mmol/l (or patient symptomatic)

A reduction in available phosphate may compromise any organ system, alone or in combination. The critical role phosphate plays in energy metabolism explains the systemic nature of symptoms caused by hypophosphataemia. Patients may present with; diplopia, dysarthria, dysphagia, weakness of trunk or extremity muscles, symptoms of respiratory insufficiency or myocardial depression, and a wide range of neurologic symptoms from paraesthesia to profound alterations in mental status. Hypophosphataemia may precipitate rhabdomylosis in alcoholics, diabetic ketoacidosis and re-feeding syndrome, and may co-exist with hypokalaemia.

Mild to moderate hypophosphataemia is usually asymptomatic. Major clinical sequelae usually occur only in severe hypophosphataemia. As a consequence of the problems of hyperphosphataemia outlined above, careful consideration must be given to the true need for phosphate replacement therapy.

Intravenous phosphate replacement should not be given until plasma levels are < 0.32 mmol/L, or unless re-feeding syndrome is considered to be a significant risk factor (see below). Otherwise recommence oral feeding and discuss oral phosphate replacement with the Critical Care pharmacist.

- Causes relevant to SDU:
  - Respiratory alkalosis – should be looked for
  - Increased losses from GIT or kidneys
  - Decreased absorption from GIT or kidneys
  - Diabetic ketoacidosis
  - Acute alcohol withdrawal
  - Re-feeding syndrome
- Management:
  - The aim is to identify and correct any underlying cause.

- Oral replacement as Phosphate-Sandoz tablets is suitable for most patients on SDU in the absence of “nil by mouth” orders or ileus. (Levels between 0.32-0.50mmol/L). Otherwise treat with IV phosphate.
- Intravenous replacement is required if serum level ≤ 0.32 mmol/L.
  - ✓ If asymptomatic give 10 - 20 mmol of Polyfusor Phosphate over 4 hours via central or peripheral cannula.
  - ✓ If symptomatic give 10-50 mmol of Polyfusor Phosphate via a central venous catheter over 60 minutes.

Serum phosphate (mmol/L) Dose band for body wt	40-60kg	61-80kg	>81kg
<0.32	25mmol	35mmol	50mmol
0.33-0.6 but oral route unavailable	10mmol	15mmol	20mmo

✓ Repeat serum level 2 hours after any intravenous intervention, and repeat dose as necessary.

- Refer symptomatic patients to a senior ICU clinician as ABC management is often required.

7. Re-feeding Syndrome

Refer to Hospital guideline

Re-feeding Syndrome may be defined as the consequences of severe fluid and electrolyte shifts that can occur in malnourished patients when they are re-fed via any route of administration. The biochemical derangements usually occur in the first 3-4 days of initiating nutritional support.

Following the stimulation of carbohydrate metabolism, insulin causes magnesium and potassium to enter cells and promotes glycogenesis and fat and protein synthesis. These anabolic processes require the phosphorylation of intermediate metabolites and results in a high intracellular demand for phosphate, which is taken into cells. Consequently the intracellular stores of these electrolytes become replenished at the expense of plasma concentration which can diminish rapidly.

Before recommencing feeding in any patient who has been “nil by mouth” for five days or more, ask for a review by the Nutrition Team who will score the patient’s risk of developing re-feeding problems.

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Documentation Controls

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Approved By:	June 2022 - Step Down Unit June 2022 - Surgical Division (review -no changes)
Review Date:	June 2025
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