GUIDELINES FOR THE MANAGEMENT OF POTASSIUM IMBALANCE IN ADULTS

Developed by -

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These are clinical guidelines only. The interpretation and application of clinical guidelines will remain the responsibility of the individual clinician.

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Introduction

Potassium imbalance is commonly encountered in the critical care setting and can lead to significant morbidity & mortality if not corrected appropriately.

Normal range for potassium: 3.5-5.5mmol/l (May vary slightly between different laboratories)

HYPERKALAEMIA

Hyperkalaemia is classified as follows according to European Resuscitation Council Guideline definition.

	Potassium level (mmol/l)
Mild	5.5-5.9
Moderate	6.0-6.4
Severe	≥ 6.5 Or ECG changes + } with a serum Or symptoms + (can be nonspecific) } potassium <6.5

Hyperkalaemia is a common condition in intensive care units,emergency treatment units and renal units. Severe hyperkalaemia is a medical emergency; unless prompt attention is given the result is cardiac arrhythmias and cardiac arrest.

Always consider a precipitant for hyperkalaemia and correct it.

CAUSES OF HYPERKALAEMIA

- Renal failure
- Drugs (e.g. ACE inhibitors, angiotensin II receptor blockers, spironolactone, and potassium sparing diuretics, potassium supplements, digitalis glycosides, non-selective beta blockers).
- Addison's disease

- Metabolic acidosis (includinding diabetic ketoacidosis)
- High platelet count and high WBC artificially raise the potassium level
- Cardio-pulmonary bypass
- Severe tissue trauma causing rhabdomyolysis
- Cardiopulmonary bypass
- Rhabdomyolysis
- Hyperkalaemic Periodic Paralysis
- Malignant Hyperthermia
- Haemolysed blood sample (repeat sample) - one should never waste time on waiting for results if the background information is highly suggestive of hyperkalaemia)

Clinical Assessment

Urgent assessment of clinical status should be carried out by a medical officer using the ABCDE approach (Appendix 1), bearing in mind that a malignant arrhythmia may be the first presentation.

The patient should undergo a comprehensive medical and drug history along with clinical examination to determine the cause of hyperkalaemia.

Hyperkalaemia is rarely an isolated problem.

As symptoms such as fatigue, weakness, palpitations paralysis etc are non specific as well as unreliable in critically ill patients, they are not very useful in diagnosis.

An urgent 12 lead ECG is recommended in patients with a potassium>6.0 mmol/l, though severe hyperkalaemia can still be present in patients without obvious ECG changes.

Serum potassium	Typical ECG appearance	Possible ECG abnormalities
Mild (5.5-6.5 mEq/L)		Peaked T waves Prolonged PR segment
Moderate (6.5-8.0 mEq/L)		Loss of P wave Prolonged QRS complex ST-segment elevation Ectopic beats and escape rhythms
Severe (>8.0 mEq/L)		Progressive widening of QRS complex Sine wave Ventricular fibrillation Asystole Axis deviations Bundle branch blocks Fascicular blocks

MANAGEMENT

SEVERE HYPERKALAEMIA

If serum potassium is ≥6.5mmol/l or hyperkalaemia is accompanied by ECG changes or above symptoms then it is considered a medical emergency. Urgent treatment is necessary as outlined below:

- **Stop further potassium rise**

  Stop all potentially offending drugs or infusions immediately.

- **Monitor ECG**

  A 12-lead ECG and cardiac monitoring is mandatory in patients with severe hyperkalaemia.

  The ECG may be normal even in the presence of severe hyperkalaemia, and that should not delay the treatment. However, the presence of ECG findings should be a strong impetus for urgent action.

  The typical ECG findings may or may not be there. It is often difficult to judge if T-waves are truly peaked and this finding on its own should not be an automatic indication for urgent therapy.

- **Protect the myocardium (cardiac membrane)**

  Give - **10ml of calcium chloride (Ca^{2+} 6.8 mmol/ml)**

  or

  30ml of 10% calcium gluconate (Ca^{2+} 2.26 mmol/ml)

  intravenously over 5-10 minutes via a large peripheral vein or a central venous catheter.

  Continuous cardiac monitoring is essential during the infusion& do a 12 lead ECG after the infusion is over.

  If ECG changes were present, there should be improvement seen within 1 to 3 minutes.

  Dose can be repeated after 5-10 minutes if hyperkalaemic changes persist.

  The effect of calcium lasts approximately 30 minutes therefore it is essential to continue other treatment methods to bring the serum potassium down.

  Calcium chloride is the more irritant of the two & extravasation of calcium salts can cause tissue necrosis. Therefore ensure access is patent and watch for signs of irritation.

  Do not administer sodium bicarbonate simultaneously via same access.

  Historically, caution had been advised with administration of calcium in the presence of digoxin toxicity but recent evidence has shown no increased risk of arrhythmias or mortality in this group of patients.[(1)]

- **Shift potassium from the blood into the cell**

1. Do a random blood sugar using a glucometer.

   10 units of soluble insulin (Actrapid or Humalin S) can be added **to 50ml of 50% glucose**and administer intravenously, to a large vein via a syringe driver over 15 – 30 minutes.

   Instead add 10units of soluble insulin (i.e. Actrapid or Humalin S) to 250ml of 10% glucose and give peripherally, ideally into a large vein over a minimum of 30 minutes.

   Dose may be repeated if necessary.

   Effect will be seen within 15 – 30 minutes and will last 4-6 hours.

   Blood glucose monitoring is essential for at least 6 hours post administration.

   Hyperosmolar glucose should not be used in hyperkalaemia seen with diabetic ketoacidosis.

2. Give 10mg (10 ml of salbutamol solution) of nebulised salbutamol. This lowers the potassium by 0.5-1.0mmol/L and its effects are seen within 15-30mins, lasting up to 4 hours.

   As 40% of patients will not respond to salbutamol, it should not be used as monotherapy.

   Caution should be exercised with ischaemic heart disease & open angle glaucoma.

A combination of insulin-glucose infusion and salbutamol has additive effects in lowering the potassium, so should be used together for best results.

- **Further treatment options:**

1. **IV Sodium bicarbonate**-

   Not recommended for routine use for the acute treatment of hyperkalaemia,unless the patient is acidotic: i.e. diabetic ketoacidosis.

   Prolonged administration of sodium bicarbonate may lower potassium but at the expense of a large sodium load. There are also potential risks in giving sodium bicarbonate in terms of volume overload and tetany in patients with chronic renal failure & co-existent hypocalcaemia.

2. **Frusemide** - initially 10-20mg IV (ONLY ifclinically appropriate).
3. **Haemodialysis/ haemofiltration** should be considered as definitive treatment if other options fail. Adequate time should be allowed to organize this, depending on the services & facilities available in the institution.

NON-SEVERE HYPERKALAEMIA

Stop further potassium accumulation

Stop all potentially offending drugs or infusions immediately.

- Place the patient on a low potassium diet.
- Remove potassium from the body using a **cation-exchange resin** - Calcium Resonium® / Kayexalate®:

  (Each gram of Calcium Resonium® removes approximately 1mmol/l K+ from the gut.)

  Give Calcium polystyrene sulphonate resin (**Calcium Resonium®) orally, 30g followed by 15g orally 4 times daily** with regular lactulose to increase gut losses of potassium. It should be given in a small amount of water, or made into a paste with some sweetened vehicle.

  Do not use fruit juice as this has high potassium content.

  OR give Calcium resonium **rectally**. This must be retained for 9 hours followed by irrigation to remove resin from the colon to prevent faecal impaction. Most serious adverse effect is intestinal necrosis which can occur when given orally or rectally.

  The onset of action is slow (> 2hours) therefore other methods such as insulin dextrose and salbutamol nebulisation should be used in the interim to lower potassium.

MONITORING

- Serum potassium should be monitored closely to assess efficacy of treatment as well as rebound hyperkalaemia. It can be suggested that it is done atleast 1,2,4,6 and 24 hours since identification.
- Blood sugar monitoring should be carried out at regular intervals in those who had insulin-glucose infusion. It is recommended that blood glucose is done at 0, 15,30,60,90, 120 minutes and hourly thereafter till minimum of 6 hours.
  Treat hypoglycaemia with a bolus of 25-50g of glucose.
- Renal function tests (twice a day or daily)
- Other relevant investigations to diagnose a precipitant
- Vital signs
- Urine output
- Fluid balance
- Creatine Phosphokinase (CPK)

Hyperkalaemic cardiac arrest :

Hyperkalaemia should be considered in all patients who have a cardiac arrest as part of identifying & treating a reversible cause.

The presenting cardiac arrest rhythm associated with hyperkalaemic cardiac arrest maybe shockable (pulseless ventricular tachycardia or ventricular fibrillation) or non shockable (pulseless electrical activity or asystole).

Patients could be refractory to defibrillation until the potassium is controlled. Thus resuscitation efforts are frequently prolonged.

Dialysis should be considered if resistant to medical therapy.

HYPOKALAEMIA

Hypokalaemia is defined as **serum potassium <3.5 mmol/l.**

Severe hypokalaemia is when serum potassium < 2.5 mmol/l

Causes of Hypokalaemia

- Increased potassium loss
    1. Drugs: diuretics (thiazides, loop diuretics), laxatives, glucocorticoids, fludrocortisone, penicillins, amphotericin, aminoglycosides
    2. GI losses: diarrhoea, vomiting, ileostomy, intestinal fistula
    3. Renal losses: renal tubular disorders, dialysis, diabetes insipidus
    4. Endocrine disorders: hyperaldosteronism (Conn's syndrome), Cushing's syndrome
- Trans-cellular shift
    1. Insulin/glucose therapy
    2. Salbutamol and other beta-agonists
    3. Theophylline
    4. Metabolic alkalosis
- Decreased potassium intake
- Magnesium depletion (associated with increased renal potassium loss)

Clinical Assessment

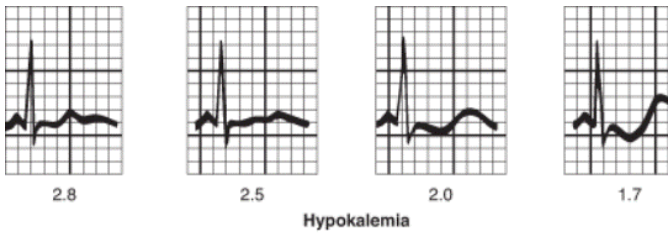
Assess the patient's clinical status using the ABCDE approach and here again dysrhythmias are the major risk. Some patients are at a greater risk of the effects than others; such as patient's on digoxin, those with heart failure, ischaemia etc. which has to be elicited in the history.

Common symptoms are fatigue, weakness, leg cramps, paralytic ileus, constipation etc and if the potassium is less than 2.5 mmol/l and prolonged hypokalaemia was present, they can have rhabdomyolysis, paralysis & respiratory difficulty. Cardiovascular effects like bradycardia, tachycardia, hypotension & arrhythmias

Do an ECG, especially in severe symptomatic hyperkalaemia, undelying cardiac disease & renal impaired.

ECG features seen are,

- U waves
- T wave flattening
- ST segment changes
- Arrhythmias(VF/VT, PEA & asystole are all possible; characteristically torsade de pointes)
- Reduced p wave amplitude



Severity of the clinical effects depends not only on the potassium level, but also on whether the fall is acute or chronic; thus review previous reports if available.

In addition to correcting the serum potassium level, further analysis may be necessary to determine the cause (as listed in page 8) & prevent further episodes.

Further investigations that may be done(depending on the availability) to determine the cause are,

- Urine K+
- Serum Magnesium
- Blood urea, Serum Creatinine, Chloride level, Bicarbonate (ABG), Blood Sugar
- Drug screen of urine & serum for diuretics, amphetamines & sympathomimetics
- Serum Renin, Aldosterone & Cortisol
- 24 hour urine for Renin, Aldosterone, Potassium & Sodium
- Imaging
  - Pituitary for Cushing's syndrome
  - Adrenals for adenoma
  - Renal Artery Stenosis
- 17-beta hydroxylase assay
- Thyroid function

MANAGEMENT

Treatment aims should be to reduce losses, replenish potassium stores, evaluate potential toxicities and determine the cause so that further hypokalaemia can be prevented.

- Thus the cause has to be identified & corrected as much as possible.
- Gradual replacement of potassium (via oral route) is preferred, if clinically appropriate.
- Potassium must be replaced cautiously in patients with renal impairment as there is risk of hyperkalaemia secondary to impaired potassium excretion.
- Oral potassium should be taken with plenty of fluid, with or after feeds.
- Use Intravenous route in patients with severe nausea, vomiting or abdominal distress.
- 0.9% sodium chloride is the preferred infusion fluid as 5% glucose may cause trans-cellular shift of potassium into cells.
- Check magnesium levels - repletion of magnesium stores will facilitate more rapid correction of hypokalaemia.

Mild to Moderate Hypokalaemia (2.5-3.5 mmol/l)

Replace orally unless symptomatic.

Oral KCl 2 tablets 3-4 times daily

Monitor serum potassium level daily and replace appropriately.

Intravenous replacement should be done if oral/nasogastric feeds are not tolerated or if there is associated ECG abnormalities/arrhythmias

IV KCl 10 mmol/hour with regular serum levels being done.

If via a central access - concentration can be 20 mmol/100ml

If via a peripheral access - concentration should not be more than 20mmol/500ml

Select the largest possible vein

Monitor the vein for extravasation, phlebitis etc

Use an infusion pump whenever available

Severe Hypokalaemia (<2.5 mmol/l or higher serum levels with symptoms)

Intravenous replacement should be done while monitoring the patient closely.

Maximum rate of infusion should be **40 mmol/hour** when there is severe symptomatic hypokalaemia.

Generally 20 mmol/ hour should be adequate and repeat doses can be given while monitoring the serum level.

Concentration of the solution should depend on type of intravenous access, peripheral venous access requiring more dilute solutions.

Peripheral access - 40mmol per litre

Central access- 20 mmol per 100 ml (higher concentrations have been used, but this is the highest concentration with an evidence base)

Potassium deficit is given by the following formula[8]

Potassium deficit in mEq= {(3.5 - patient's K) body weight}0.4

In severe hypokalaemia, Serum Magnesium levels should be assessed and Mg replaced at the same time.

Monitoring when potassium being replaced:

ECG monitoring -for infusions of >10 mmol/hour.

Monitor the IV access site for extravasation.

Monitor blood glucose levels & acid base status regularly.

Once serum potassium is replaced to normal limits or till symptoms are resolved, regular dose of replacement should be prescribed if appropriate.

Correction of the possible cause is important to prevent further episodes of hypokalaemia.

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APPENDIX 1

ABCDE Approach

A – Airway –

Recognise & treat airway obstruction.

B – Breathing –

Assess adequacy of ventilation; clinical examination, respiratory rate, O_2 saturation, arterial blood gas.

Give oxygen aiming for an oxygen saturation appropriate for the patient. Provide ventilator support if necessary.

C – Circulation –

Assess cardiovascular status; colour, pulse, BP, volume status, peripheral circulation, urine output (check for palpable bladder), cardiac rhythm (ECG), electrolytes (Urea, electrolytes, Mg^{2+}, Ca^{2+}, Phosphate).

Consider fluid bolus (with care), vasopressors, inotropes, treatment of arrhythmia, correct electrolyte abnormalities.

D – Disability –

Assess neurological function (GCS) and blood glucose

Correct underlying cause.

E – Exposure –

Head to toe assessment and look for evidence of cause – eg: signs of injuries, compartment syndrome,palpable bladder, skin rashes, dialysis access (dialysis catheter, AV fistula).

Check temperature.