Guidelines for dealing with adult patients with hypokalaemia in the community

Aims of the guideline

This guideline is intended to support primary care clinicians with the safe management of patients with hypokalaemia, including the decision whether treatment should be managed in a primary care setting or if an admission to secondary care is required. Secondary care clinicians should refer to the advice that is available on Staff Room (Hypokalaemia diagnosis and management).

The purpose of this guidance is to support decision making. It cannot replace the need for sound clinical judgement.

Relevant clinical information and a drug history should be included on the request form when requesting renal function and electrolytes. This information will assist laboratory staff in providing the most appropriate advice.
Laboratory staff may telephone a low potassium result through to primary care. This does not automatically mean patients require admission to hospital. The **clinical urgency** of the situation may depend on:

1. **The severity of the hypokalaemia**
   - Normal range for serum potassium: 3.5 – 5.3 mmol/L
   - Mild hypokalaemia: 3.1 – 3.4 mmol/L
   - Moderate hypokalaemia: 2.5 – 3.0 mmol/L
   - Severe hypokalaemia: <2.5 mmol/L

NB this scale of ‘biochemical’ severity is arbitrary and serves only as a guide - the severity of hypokalaemia is predominantly defined by symptoms and ECG changes.

2. **The presence of any symptoms**

<table>
<thead>
<tr>
<th>Neuromuscular:</th>
<th>Muscle weakness, myalgia, ascending paralysis, rhabdomyolysis, muscle cramps, numbness, tingling, paraesthesia, hyporeflexia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gastro-intestinal tract:</td>
<td>Abdominal distension, constipation, paralytic ileus</td>
</tr>
<tr>
<td>Kidneys:</td>
<td>Polyuria (↓ concentrating ability)</td>
</tr>
<tr>
<td>Heart muscle and nerve conduction system:</td>
<td>Dysrhythmias (AF, VT, torsade de pointes, digoxin toxicity)</td>
</tr>
<tr>
<td>Respiratory System:</td>
<td>Respiratory muscle weakness</td>
</tr>
<tr>
<td>Genitourinary tract:</td>
<td>Hypotonic bladder, urinary retention</td>
</tr>
<tr>
<td>Endocrine System:</td>
<td>Insulin resistance and impaired insulin release</td>
</tr>
</tbody>
</table>

^ indicates severe hypokalaemia

3. **The presence of any ECG changes indicates severe hypokalaemia**

- Depression of ST segment
- Flattening of T wave/ T wave inversion
- Appearance of a prominent U wave
- Prolongation of PR interval
- QT prolongation (VT torsade, AF)
Arrangements for reporting low potassium results

The duty biochemist will review all potassium results <2.9mmol/L between 9am and 5pm. A new finding of potassium <2.5mmol/L is always phoned through to the GP surgery. Results 2.5-2.8mmol/L may be rung through to the GP surgery, at the discretion of the clinical biochemist. Providing appropriate clinical information on the request form will help with this judgement.

Out of hours, all potassium results <2.5mmol/L are actioned immediately by the laboratory staff. Usually, this will mean that the result is telephoned to the Out of hours (OOH) GP service. An on-call biochemistry consultant may review the result and decide that the result can be safely left until the next day to telephone to the GP practice. The decision as to whether the leave the result to the next day or not is based upon clinical details on the request form and other laboratory results (current and previous).

Once a low potassium result has been telephoned to the OOH service, responsibility passes to the OOH GP to decide on a course of action and contact the patient if appropriate.

Potassium results between 2.5-2.8mmol/L will be reviewed by the duty biochemist at 9am the next morning. Results are normally telephoned through to the GP on the morning if the low potassium level is a new finding.
Contacts for advice or admission at York and Scarborough Hospitals

If hypokalaemia is associated with severe renal impairment or if a patient is on dialysis, the on call nephrologist should be the first point of contact. They will co-ordinate management if needed (NB most people with severe kidney disease have a high potassium; low potassium on a POST-dialysis blood sample should NEVER be corrected without discussion with a renal physician).

Duty Nephrologist
01904 631313 (York hospital switchboard)

Bed manager
01904 631313 (York hospital switchboard)
bleep 998

Duty Biochemist (9am-5pm)
01904 726366 or via 01904 726802

On call consultant via hospital switchboard (01904 631313)

Biochemist (5pm – 9am)
Investigation and management of abnormal low potassium levels

1. Assess for critical complications:
   **Dysrhythmias, muscle weakness/paralysis, respiratory failure, paralytic ileus**
   If any of these are present, arrange **urgent admission** to secondary care.

   An ECG is MANDATORY in patients with severe symptomatic hypokalaemia and in people who are taking digoxin. These patients may need to be referred to the emergency department if an ECG cannot be arranged and reviewed quickly.

2. Identify the cause of hypokalaemia:
   a) History (including **medications** – see appendices 1 & 2)
      Most causes are evident from history and examination.

   b) If unexplained, refractory or diuretic-induced hypokalaemia, check the serum **magnesium**. In most cases, this will have been added by the laboratory. Replacement of potassium will be ineffective and will not result in a sustained increase in serum potassium if the serum magnesium is also low.

   c) If the cause of hypokalaemia remains unclear, measure **urinary potassium excretion** with a 24h urine collection (ideally) and **acid-base status** on a venous blood sample for **U&E and bicarbonate**. These samples should be collected on the same day.
      - Urinary potassium excretion <20mmol/24h (or <15mmol/L in a random sample) suggests a **Non-renal loss of potassium**
      - Urinary potassium excretion >20mmol/24h (or >15mmol/L in a random sample) suggests a **Renal loss of potassium**

3. Address the underlying cause of hypokalaemia and replace potassium (see appendix 3)

   **NB:**
   Hypokalaemia may not resolve in the presence of hypomagnesaemia.
   Hypokalemia will recur if the underlying cause has not been appropriately identified and corrected.
Appendix 1. Causes of hypokalaemia

- **Hypokalaemia**
  - Urinary K excretion >20 mmol/24h (random sample >15 mmol/L) *(Renal loss)*
  - Urinary K excretion <20 mmol/24h (random sample <15 mmol/L)

  **BP**

- **HIGH**
  - Hyperaldosteronism
  - Glucocorticoid excess
  - Genetic

- **NORMAL / LOW**
  - Alkalosis *(raised bicarb.)*
  - Diuretics
  - Response to vomiting*
  - Bartter’s syndrome
  - Gitelman’s syndrome
  - Acidosis *(low bicarb.)*
  - Hypomagnasaemia
  - Response to DKA
  - Renal Tubular Acidosis

- **PRIOR RENAL POTASSIUM LOSS**
  - (now potassium deplete)
  - History of diuretic use
  - History of prolonged vomiting*

- **NON-RENAL POTASSIUM LOSS**
  - Diarrhoea / laxatives* *(acidosis)*
  - Villous adenoma *(alkalosis)*

- **TRANSCELLULAR POTASSIUM SHIFT**
  - Acute cell proliferation *(e.g. following B12 or folate administration to treat megaloblastic anaemia)*
  - Acute myeloid leukemia
  - Other cause of extremely high WCC
  - Familial / thyrotoxic hypokalaemia paralysis

* Denotes symptoms that can occur with hypokalaemia.
### Appendix 2. Medications associated with hypokalaemia

<table>
<thead>
<tr>
<th>Renal losses</th>
<th>GI Losses</th>
<th>Transcellular shifts</th>
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<tbody>
<tr>
<td>Thiazide diuretics*</td>
<td>Laxatives*</td>
<td>Insulin</td>
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<tr>
<td>Loop diuretics*</td>
<td></td>
<td>β-agonists (e.g. salbutamol)</td>
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<tr>
<td>Glucocorticoids</td>
<td></td>
<td>Theophylline (acute intoxication)</td>
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<tr>
<td>Aminoglycosides</td>
<td>(e.g. gentamicin)</td>
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<td>Penicillin (in high doses)</td>
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<tr>
<td>Amphotericin B</td>
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<tr>
<td>Fludrocortisone</td>
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<tr>
<td>Cisplatin</td>
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</tbody>
</table>

* Commonest causes

When reviewing medication, it is important to consider the indication for the medication and the potential consequences of stopping the medication.
Appendix 3 – Oral potassium replacement


Gradual replacement of potassium (via oral route) is the preferred treatment if the patient has a functioning bowel UNLESS severe hypokalaemia is present (mainly defined by symptoms and ECG changes, rather than the serum potassium). If there is severe hypokalaemia or if the patient will not tolerate oral replacement, ADMIT to hospital for intravenous treatment.

Potassium should only ever be replaced cautiously in patients with renal impairment (eGFR less than 45 mL/min) or any level of renal impairment and taking medication(s) which interferes with intra/extracellular potassium distribution OR inhibits potassium excretion, because of the risk of causing hyperkalaemia. NEVER treat a low potassium result obtained after dialysis treatment in a patient with end stage kidney disease. Contact the Renal Team if a patient is on dialysis or has severe renal impairment (eGFR less than 15 mL/min).

Oral potassium should be taken with plenty of fluid, with or after meals. Sando-K is the most widely available oral potassium preparation. Each Sando-K tablet contains 12 mmol of potassium. Sando-K also contains bicarbonate and may not be appropriate in patients with a significant metabolic alkalosis.

Patients with mild to moderate hypokalaemia may need 60-80mmol/day. Two tablets three times a day may be an appropriate replacement dose. The duration of treatment and the timing of blood tests to monitor response to treatment will depend on the underlying cause of hypokalaemia and whether or not the cause has resolved or is still present. This is a clinical judgement and has to be made on a case by case basis.

**Side effects of oral potassium administration:**
Nausea and vomiting, abdominal pain, diarrhoea, flatulence, GI obstruction, hyperkalaemia.
References

1. York & Scarborough Teaching Hospital Foundation Trust
   Hypokalaemia Diagnosis & Management – October 2019 to October 2022
   T. Pawlak and C. Jones

Authors

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