Royal United Hospitals Bath
NHS Foundation Trust

# Information for Clinicians

## Clinical Biochemistry Department

# Hypokalaemia – a guide for GPs

## Overview

**Patients with severe hypokalaemia OR symptomatic moderate/mild hypokalaemia require urgent admission. Severe hypokalaemia can cause life threatening arrhythmias.**

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|---|---|
| Mild | $K^+$= 3.1-3.5 mmol/L |
| Moderate | $K^+$= 2.5-3.0 mmol/L |
| Severe/ Symptomatic | $K^+$ <2.5 mmol/L |

**All results <2.5 mmol/L will be phoned by the laboratory to the requesting location or out of hours (OOH) service, consider urgent admission in these cases.**
GP Acute Medical Referrals: 07824 334450, MAU Consultant Advice Line: 07818 013823

98% of body's potassium is intracellular. The intra-extracellular gradient is crucial to maintaining resting membrane potential and normal nerve function. A small decrease in extracellular potassium due to gastrointestinal or renal losses, redistribution between intra- and extracellular concentrations or reduced intake can have serious effects on heart and skeletal muscles.

Patients on digoxin and underlying cardiac conditions with even mild hypokalaemia have increased risk of digoxin toxicity and arrhythmia.

## Symptoms of low potassium include:

- Muscle cramps and pain with rhabdomyolysis
- Weakness and fatigue
- Palpitations and syncope
- Cardiac arrhythmias or rhabdomyolysis
- Muscle weakness – leading to constipation, dyspnoea.
- Psychological symptoms: delirium, hallucinations
- Symptoms that present in mild hypokalaemia are often from underlying cause of the hypokalaemia rather than hypokalaemia itself.

## Causes of low potassium

The patient history is often helpful to clarify the likely cause of hypokalaemia. Common causes include:

**Gastrointestinal loss of potassium**

- Gastrointestinal Losses (51%) (diarrhoea, vomiting, fistulae, villous adenoma – very rare)
- Laxative abuse

**Increased renal loss of potassium**

- Diuretic treatment (47%)
- Hypomagnesaemia
- Mineralocorticoid excess (Cushing's syndrome, Primary hyperaldosteronism, Secondary hyperaldosteronism : e.g. volume depletion and congestive heart failure, Congenital Adrenal Hyperplasia)
- Exogenous mineralocorticoid excess (steroids, liquorice, Renal Tubular Acidosis I and II)
- Increased urine flow (osmotic diuresis)
- Renal tubular transport defects: Bartters, Gitelmans or Liddle syndrome

**Redistribution of potassium from extracellular to intracellular space**

- Redistribution due to Insulin treatment for diabetic ketoacidosis
- Uptake of potassium into cells due to B12 or folate replacement
- Refeeding: following prolonged starvation, alcoholism, eating disorders
- Alkalosis
- Hypokalaemic periodic paralysis and thyrotoxic periodic paralysis

**Pseudo hypokalaemia**

- Seasonal pseudohypokalaemia – delayed analysis of venous samples in warmer weather
- Significant leukocytosis due to the in vitro uptake of potassium by white blood cells.

**Various drugs can cause hypokalaemia:**

- Loop or thiazide diuretics (furosemide, Indapamide)
- Laxatives (consider hidden laxative abuse)
- Insulin or glucose administration
- Corticosteroids (hydrocortisone, prednisone)
- Beta-agonists (salbutamol, formoterol)
- Xanthines (Theophylline, aminophylline, caffeine)

## Investigations

**Please note in warmer weather delays in sample reaching lab can lead to spurious (pseudo) hypokalaemia.**

First line investigations:

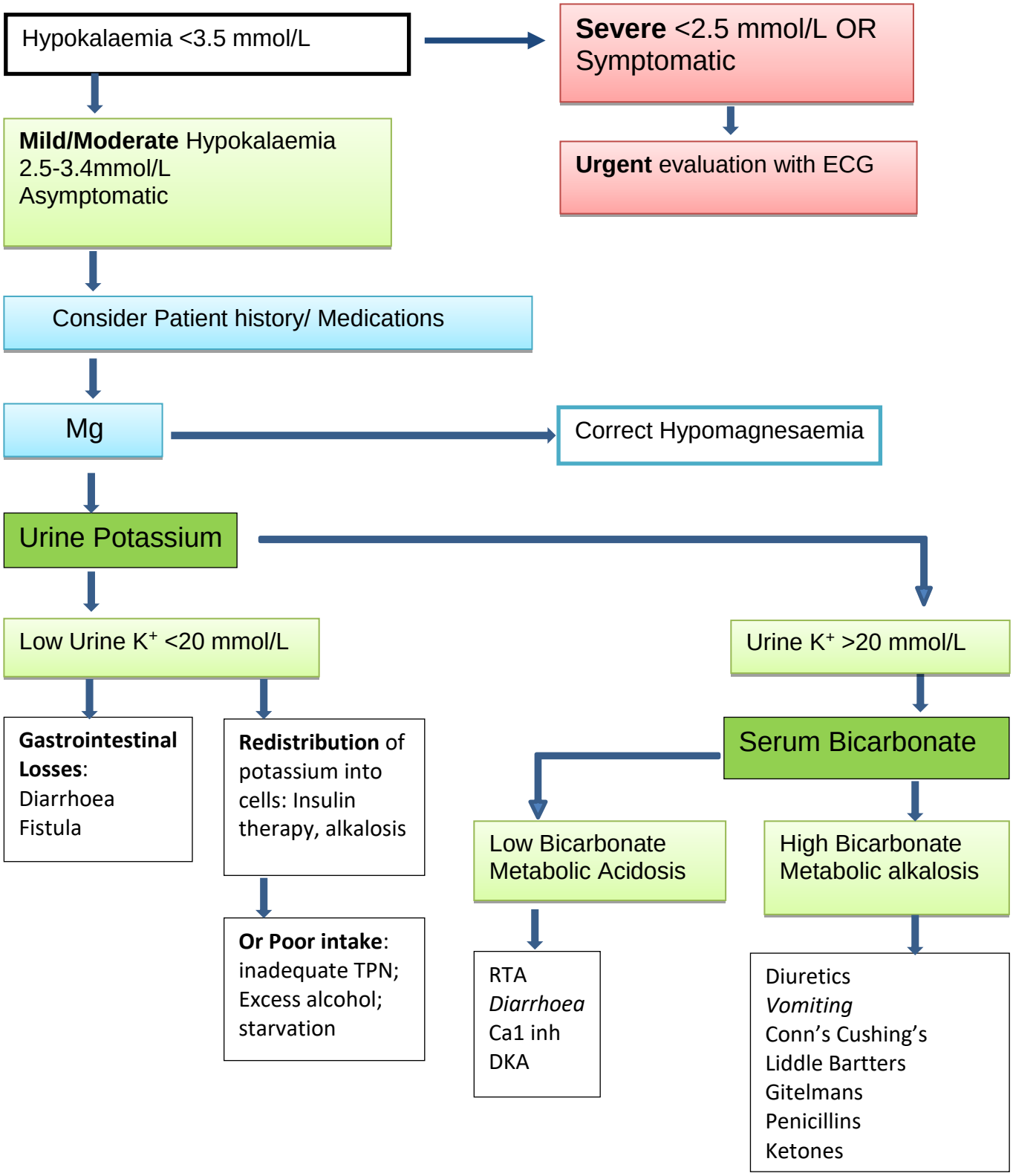
- Serum electrolytes and renal function
- Magnesium. Hypokalaemia is often refractory to treatment unless hypomagnesaemia is corrected
- Bicarbonate to assess acid-base status (please ensure sample arrives to laboratory within 4 hours)
- FBC (high WCC can cause an increase or decrease to potassium in vitro and mask true potassium status)
- If clinical suggestion of hyperthyroidism consider TFTs
- If myalgia exclude rhabdomyolysis with CK
- ECG if $K^+$ <3 mmol/L. ECG changes include small p waves, U waves, prolonged QT, ST depression and T wave flattening

Second line investigations:

- If no obvious cause, it is essential to assess if there is renal loss of potassium: 24 hr urine collection is the gold standard (for collection container telephone 01225 824712) or if this is not possible send paired random urine sample (with serum sample) for potassium, taken at time patient is hypokalaemic and not on supplements.
- Further second line investigations can be complex so it is advised to telephone the duty biochemist if persistent hypokalaemia that cannot be explained.

See flow chart below for help investigating the cause of hypokalaemia.

To discuss any patients and their results please contact the Duty Clinical Biochemist on 01225 824050 (available 9 am-5 pm Mon-Fri) or via consultant connect. For queries OOH the Duty Clinical Biochemist may be contacted via the Biochemistry Laboratory Biomedical Scientist, contactable through RUH switchboard.



## Management

The underlying cause of low potassium should be identified and managed appropriately.

Magnesium

- Hypokalaemia and hypomagnesaemia often co-exist. Treatment of hypokalaemia unlikely to be successful without correction of hypomagnesaemia.

Oral potassium replacement:

- Dietary sources of potassium-Tomato juice, coffee, nuts, fruit, bananas and chocolate.
- Smaller doses must be used if there is **renal insufficiency** to reduce risk of hyperkalaemia.
- Potassium sparing diuretics are recommended (rather than potassium supplements) for prevention of hypokalaemia due to diuretics such as frusemide or thiazides.
- Oral supplementation is well absorbed, but large doses are limited by nausea / vomiting
- Mild: use Sando-K (12 mmol each) 1 tab tds *or* Kay-Cee-L syrup 10ml (10 mmol) tds
- Moderate: Sando-K 2 tabs tds *or* Kay-Cee-L syrup 20ml tds
- Slow-K (8 mmol each) 1 - 2 tabs tds is an alternative but are large tablets
- Monitor weekly to several times a week dependent on severity. Prescribe for 3 days maximum and then review.

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## References

Oram RA, Vaidya B. Investigating Hypokalaemia. *BMJ*. 2013;347:f5137

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Bath RUH Acute Medicine Clinical Guidelines: Hypokalaemia

Veltri KT, Mason C. Medication-induced hypokalemia. *P T*. 2015;40(3):185–190.

# Amendment History

| Issue | Status | Date | Reason for Change | Authorised |
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