

Information for Clinicians

Clinical Biochemistry Department

Hypokalaemia – a guide for GPs

Overview

Patients with severe hypokalaemia can require urgent admission. Severe hypokalaemia can cause life threatening arrhythmias.

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 whether urgent admission is required 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
- 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

Patients are often asymptomatic and symptoms are from the underlying cause of hypokalaemia. Common causes include:

Gastrointestinal loss of potassium

- Gastrointestinal Losses (51%) (diarrhoea, vomiting, fistulae, villous adenoma – very rare)
- Laxative abuse
- Chronic purging/ eating disorders

Increased renal loss of potassium

- Diuretic treatment (47%)
- Hypomagnesaemia
- Increased urine flow (osmotic diuresis)
- 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)
- 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:

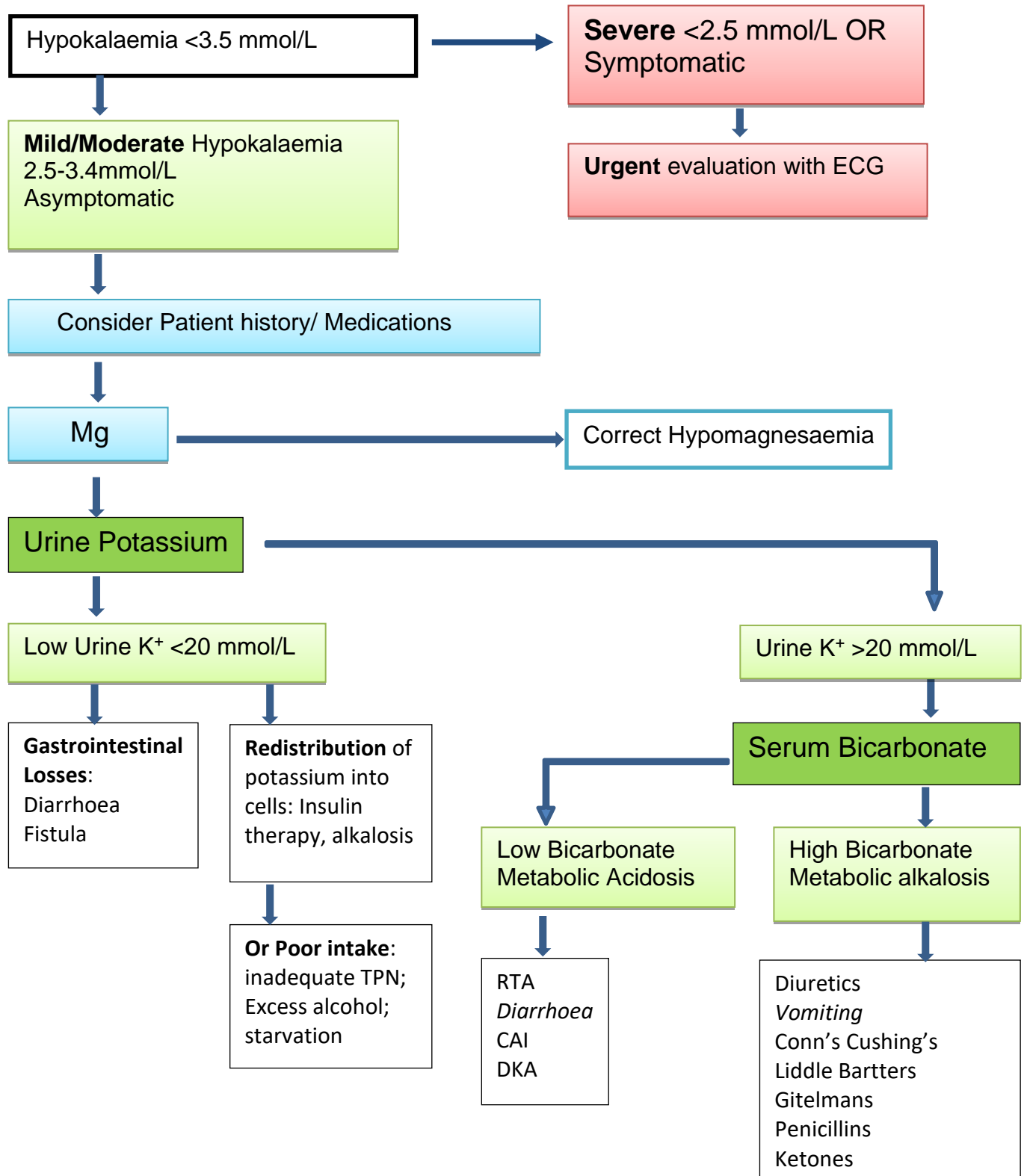
- Serum electrolytes and renal function
- Magnesium. Hypokalaemia is often refractory to treatment unless hypomagnesaemia is corrected
- Bicarbonate to assess acid-base status (please ensure sample arrives to laboratory within 4 hours).
- FBC (high WCC can cause an increase or decrease to potassium in 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 Cinapsis. 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 bd.
- Moderate: Sando-K 2 tabs bd.
- Slow-K (8 mmol each) 1 - 2 tabs tds is an alternative but are large tablets
- Initially prescribe for 3-4 days and then monitor potassium levels, the supplementation should be titrated according to the response. Monitor weekly to several times a week dependent on severity.

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Severe/ Symptomatic	K ⁺ <2.5 mmol/L	Acute Medical care. IV K ⁺

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
2		11.7.24	No changes	M O'Doherty
3		5.11.24	Remove Kay-Cee-L syrup	M O'Doherty