

# Hyperkalaemia in Primary Care: Investigation and management (adults)

Authors: Colin Jones<sup>1</sup>, David Border<sup>1</sup>, Alison Jones<sup>2</sup>, Fiona Lloyd<sup>3</sup>.

<sup>1</sup>Consultant in Renal Medicine, <sup>2</sup>Consultant in Clinical Biochemistry, <sup>3</sup>General Practitioner

## Aims of the guideline

This guideline is intended to support primary care clinicians to safely manage high blood potassium results. Secondary care clinicians should refer to the advice that is available on Staff Room on managing acute (Protocol for management of acute hyperkalaemia in adults) and chronic hyperkalaemia (Chronic hyperkalaemia management guideline).

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 (see Appendix 3). This information will assist laboratory staff in providing the most appropriate advice.

Laboratory staff may telephone a high potassium result through to primary care. This does not automatically mean patients require admission to hospital (see guidance below).

The **clinical urgency** of the situation is mainly dependent on two factors:

### 1. The severity of the hyperkalaemia

Normal range for serum potassium	3.5 – 5.3 mmol/L
Mild hyperkalaemia	5.4 – 5.9
Moderate hyperkalaemia	6.0 – 6.4
Severe hyperkalaemia	≥6.5

NB this scale of severity is arbitrary and serves only as a guide - the severity of hyperkalaemia is dependent on the impact on the patient.

### 2. The rate of change in potassium and any change in serum creatinine/eGFR.

Rapid rises in potassium or significantly impaired renal function increase the likelihood of an impact on the patient and the need for intervention in the community and/or hospital admission.

Significant changes are:-

- a) a sudden (within 1 week) rise in potassium
- b) a >10% increase in serum creatinine or >10% decrease in eGFR

If the potassium is elevated in the presence of either normal kidney function or stable chronic kidney disease, you may need to consider the following explanations for a high potassium:

- 1) spurious hyperkalaemia – see Appendix 1
- 2) hyperkalaemia related to a metabolic acidosis – serum bicarbonate is automatically added on to all potassium results greater than 6.0mmol/L in York and Scarborough hospitals.

## Arrangements for reporting raised potassium results

The duty biochemist will review all potassium results  $>5.8\text{mmol/L}$  between 9am and 5pm. Results  $>6.5\text{mmol/L}$  are always phoned through to the GP surgery. Results  $<6.5\text{mmol/L}$  may be rung through to the GP surgery at the discretion of the biochemist.

Out of hours, all potassium results  $>6.5\text{mmol/L}$  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 to leave the result to the next day or not is based upon clinical details on the request form, other laboratory results (current and previous) and collection time and date of the sample.

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

Potassium results between  $5.8$  and  $6.5\text{mmol/L}$  will be reviewed by the duty biochemist at 9am the next morning. Results may be telephoned through to the GP in the morning, at their discretion.



## Management of abnormal Potassium levels

1. Check if the result is spurious (Appendix 1)
2. Consider causes of hyperkalaemia (Appendix 2)

- **Potassium 5.4 – 5.9mmol/L**

Action A)

If eGFR has not decreased by >10%

**And**

The rise in K<sup>+</sup> is not recent (within 1 week)

**Then**

Review medications and consider any possible changes to be made (Appendix 3)

Repeat U&E within 1 week

Action B)

If eGFR has decreased by > 10%

**Or**

The rise in K<sup>+</sup> is recent (within 1 week)

**Then**

Review potential causes and repeat U&E within 48 hours

Consider discussion with a Renal Physician if not improving

*The majority of these patients do not need emergency admission.*

- **Potassium 6.0 – 6.4mmol/L**

(May have been telephoned same day or next working day)

Assess for the presence of “high risk” features (Appendix 4)

Action A)

If high risk features – arrange emergency assessment (Emergency Department)

Action B)

If no high risk features are present and potassium result is believed to be genuine contact the patient and assess them (Appendix 5)

Review medication and consider if any drugs could be stopped safely (see Appendix 3)

Repeat U&E within 24 to 48 hours, depending on clinical context (as per this document)

- **Potassium  $\geq$  6.5**

(Result telephoned to requestor)

If the potassium result is believed to be genuine, **this is high risk hyperkalaemia**. Contact the patient and arrange repeat potassium and ECG as emergency (Emergency Department)

**Do not** offer patients with hyperkalaemia dietary advice. Except in end stage kidney disease, dietary restriction is rarely appropriate in the management of high potassium and may lead to inappropriate restriction of healthy food choices when diet is rarely the cause of the hyperkalaemia (except in ESKD). Patients with ESKD should be supervised by a renal dietitian, who can both identify potential sources of high potassium foods and ensure that nutritional needs are met.

New oral potassium exchange agents (Patiromer and Zirconium) have been approved by NICE to manage hyperkalaemia in specific circumstances (see Appendix 3). **Do not** prescribe calcium resonium. It has no place in the acute or long term management of hyperkalaemia.

## Appendix 1: Causes of a spuriously high potassium result

**Haemolysis** (see <https://tinyurl.com/BiochemInfo> for more information)

The laboratory will not report a K<sup>+</sup> result if the sample is grossly haemolysed (K<sup>+</sup> result will appear as NA with a comment stating haemolysed sample). The laboratory will report a numerical K<sup>+</sup> result along with a cautionary comment if a sample is slightly haemolysed. At this level of haemolysis the contribution to K<sup>+</sup> measured in serum will be very small (~0.2 mmol/L).

Haemolysis has various causes e.g. difficult sample collection, shaking of the tube etc.

### **EDTA contamination**

Collection of blood samples in reverse traffic light order, i.e. green topped tube first, then amber, lastly red, prevents EDTA contamination of the sample. Gross EDTA contamination is usually detected within the laboratory and the K<sup>+</sup> result will be reported as NA with an appropriate comment.

**Delay in centrifugation / receipt in lab** (see <https://tinyurl.com/BiochemInfo> for more information). If there has been a delay in processing the sample, K<sup>+</sup> will move from the red blood cells into the serum and contribute to high K<sup>+</sup> results. This effect is more pronounced at low temperatures and so there is increased incidence in winter. Serum samples for K<sup>+</sup> should NEVER be refrigerated. It is useful for the labs to have the time the sample was taken written on the blood bottle and request form, to help detect when this effect may have occurred. The laboratory will usually comment on reports if they consider a delay in receipt may have contributed to high K<sup>+</sup>.

### **Abnormally high Red cell count / White cell count / Platelet count**

Patients with abnormally high red cell, white cell or platelet counts may have spurious hyperkalaemia. A repeat blood test should be obtained in a lithium heparin tube obtained and taken at the hospital phlebotomy service to remove any transport effects. Please contact the Duty Biochemist to discuss.

## Appendix 2: Causes of genuinely raised serum potassium

1. Medications – see Appendix 3

2. Abnormal kidney function:

- Acute Kidney Injury
- Chronic Kidney Disease (usually stage 4/5)

3. Other causes

- Increased intake – very unusual, except in dialysis patients
- Redistribution
  - Diabetic ketoacidosis (before treatment)
  - Severe metabolic acidosis
  - Diabetic nephropathy (patients with longstanding diabetes and moderate renal impairment)
  - Trauma/burns
  - Rhabdomyolysis and/or tumour lysis syndrome
- Decreased excretion
  - Hypoadrenalism/Mineralocorticoid deficiency



### Appendix 3: Medications associated with hyperkalaemia

This list is not exhaustive but covers the majority of commonly implicated substances

ACE inhibitors

Angiotensin-2 receptor antagonists

Entresto (which contains valsartan)

Aldosterone antagonists e.g. spironolactone/eplerenone

Potassium supplements

Potassium sparing diuretics

NSAIDS

Trimethoprim

Heparin

Beta blockers

Ciclosporin

Tacrolimus

Ketoconazole

Theophylline

Digoxin

Dietary foodstuffs, including Losalt

Herbal remedies

When reviewing medication, it is important to consider the indication for the medication and the potential consequences of stopping the medication. For example, medications that block the renin-angiotensin-aldosterone system (e.g. ACE-inhibitors, angiotensin-2 receptor antagonists, Entresto and mineralocorticoid antagonists) improve symptoms, decrease hospital admissions and decrease mortality in people with complications of

conditions which include diabetes, chronic kidney disease and heart failure. These medicines should be continued wherever possible. They should not be stopped unnecessarily.

NICE has approved both Patiromer<sup>1</sup> and Zirconium<sup>2</sup> for long term use to manage hyperkalaemia in adults only if used for people with persistent hyperkalaemia and chronic kidney disease stage 3b to 5 or heart failure, if they:

- have a confirmed serum potassium level of at least 6.0mmol/L *and*
- because of hyperkalaemia, are not taking an optimised dosage of renin-angiotensin-aldosterone system (RAAS) inhibitor *and*
- are not on dialysis.

These medicines can be prescribed in primary care under current shared care guidelines: [York and Scarborough Formulary](#)

<sup>1</sup> [Overview | Patiromer for treating hyperkalaemia | Guidance | NICE](#)

<sup>2</sup> [Overview | Sodium zirconium cyclosilicate for treating hyperkalaemia | Guidance | NICE](#)

## Appendix 4: High risk hyperkalaemia

1) Any ECG changes – peaked T waves, widened QRS complex, absent P wave, sine wave

2)  $K^+ \geq 6.5\text{mmol/l}$

3)  $K^+ > 6.0\text{mmol/l}$  *and any of the following*

Fall in eGFR  $> 10\text{ml/min}$  or by  $> 25\%$  since last test

**And/or**

Acute Kidney Injury (AKI) alert

**And/or**

Bicarbonate  $\leq 15\text{mmol/L}$

**And/or**

new eGFR  $< 45\text{ ml/min}$

## Appendix 5: Clinical features of hyperkalaemia

### *Symptoms*

- Muscle weakness
- Fatigue
- Otherwise unexplained breathlessness
- Muscular paralysis
- Palpitations
- Chest pain

### *Signs*

- Bradycardia (secondary to heart block)
- Tachypnoea (from respiratory muscle weakness)
- Muscle weakness and flaccid paralysis
- Depressed/absent tendon reflexes

### *Examination required*

- Temperature
- Pulse
- Blood pressure
- Respiratory rate

(Pulse <50 or >100 and/or RR >16 are indicators that the patient should be assessed in hospital).