Whittington Health NHS

Hyperkalaemia

Subject:	Hyperkalaemia
Policy Number	N/A
Ratified By:	Clinical Guidelines Committee
Date Ratified:	June 2016
Version:	4.0
Policy Executive Owner:	Clinical Director MFNS ICSU
Designation of Author:	Consultant, Acute and General Medicine
Name of Assurance Committee:	Clinical Guidelines Committee
Date Issued:	July 2016
Review Date:	3 years hence
Target Audience:	All medical staff
Key Words:	Hyperkalaemia, Dialysis, Haemofiltration, Renal Failure, Acute Kidney Injury, Chronic Kidney Disease

Version Control Sheet

Version	Date	Author	Status	Comment
1.0	March 2010	Dr Richard Fish and Dr Lok Yap	Off-line	New guideline approved at CGC
2.0	June 2010	Dr Richard Fish and Dr Lok Yap	Off-line	Minor amendments
3.0	May 2012	Dr Lok Yap	Current	Reviewed. No change
4.0	March 2016	Dr Ilana Samson (Consultant) Rebecca Edwards (Clinical Biochemist)		Reviewed. New international/ national guidelines considered Added the incorporation of hyperkalaemia into the escalation patient/ level 3 alert system which is now in place Sections rewritten are: Background/ intro; clinical management; contacts and references. Appendices left unchanged.

➢ Criteria for use

For use when a patient has a serum Potassium (K) level over 5.5 mmol/L.

➢ Background/ introduction

Hyperkalaemia is a common biochemical abnormality which can lead to life-threatening cardiac arrhythmias. A serum K+ of >5.5 is the widely accepted definition but hyperkalaemia is a spectrum with the incidence of complications rising with severity and rate of rise of the serum K+, as well as the medical history of the patient. Co-existing electrolyte and metabolic disturbances (eg hyponatraemia, hypocalcaemia and acidaemia) can exacerbate the effects of hyperkalaemia so it is essential that all electrolyte and metabolic abnormalities are taken into account.

It is useful to use the European Resuscitation Guideline definition of hyperkalaemia:

Mild	5.5 - 5.9 mmol/L
Moderate	6.0 - 6.5 mmol/L
Severe	>6.5 mmol/L

Causes of hyperkalaemia:

This is commonly associated with AKI, chronic renal impairment or drugs which affect the renin-angiotensin-aldosterone axis (RAA) axis, but may also be due to redistribution of intracellular potassium into the serum in uncontrolled hyperglycaemia such as DKA. Identifying the potential cause is important in considering the management, especially as many of the immediate treatments of hyperkalaemia act to redistribute rather than excrete potassium.

Specific risk factors for hyperkalaemia include:

1. Dialysis dependency (haemodialysis or peritoneal dialysis)
2. Chronic Kidney Disease Stages 4 & 5 (CKD, eGFR < 30 ml/min/1.73m2)
3. Nephrotoxic medications (e.g. renin-angiotensin drugs, non-steroidal anti-inflammatory drugs)
4. Cardiac failure (e.g. renin-angiotensin drugs)
5. Diabetes mellitus (e.g. renin-angiotensin drugs, diabetic keto-acidosis)
6. Liver disease (e.g. spironolactone, hepato-renal failure)
7. Adrenal insufficiency

ECG changes:

Hyperkalaemia causes a rapid reduction in resting membrane potential leading to increased cardiac depolarization, and muscle excitability. The ECG changes

associated with hyperkalaemia can progress rapidly and do not behave in a dose-dependent manner. They include in approximate progression:

Peaked T waves with a shortened QT interval, prolonged PR and QRS complex, disappearance of p wave and widening of QRS, eventually progressing to a sine wave before asystole.

The objective of this guideline is to provide brief guidance on the management of acute hyperkalaemia.

- **Clinical management**

There are 3 key areas of management

a. *Stabilising the cardiac membrane with calcium ions.*
b. *Driving extracellular potassium into the cells*
c. *Removing excess potassium from the body*

A. Confirm Hyperkalaemia: Re-check K+ level

1. Recheck K+ urgently: via a blood gas analyser (Emergency Department or Biochemistry lab) from a venous blood sample for an immediate result.

If the result is >0.8 mmol different, consider pseudo-hyperkalaemia, (see Appendix 1). Please note that there is an expected slight difference in results due to the use of serum in the laboratory method vs anti-coagulated blood in the blood gas analyser (as potassium is released from platelets during the clotting process, a higher platelet count may give a larger difference between the results).

2. If pseudohyperkalaemia is suspected, then a paired lithium heparin and clotted (yellow top) should be taken from a large vein using gentle traction and sent immediately to the laboratory for comparison (the lab should be informed of this).

B. Management of hyperkalaemia and ensuring patient safety

1. SEVERE hyperkalaemia (K+ > 6.5 mmol/L)

This is a Level 3 Escalation Alert and you should inform the ITU SpR (Bleep 2613) +/- CCOT (see Deteriorating Patient Policy)

a) Stabilising the cardiac membrane with calcium ions.

1. ECG & Cardiac monitoring (see above for expected ECG changes)
2. Administration of **10ml 10% Calcium carbonate iv (on resus trolley)** given as a bolus (this acts to stabilize the myocardium and **will not** alter serum K

concentration). This should be repeated after 5 minutes if there are ECG changes which recur or fail to correct after initial bolus.

***Note - extravasation of calcium salts can cause tissue necrosis** so should be given through a large vein. They should not be co-administered with bicarbonate as this will cause precipitation of calcium carbonate.*

Note - Caution should be used in patients who have suspected or known digoxin toxicity, as calcium salts can potentiate digoxin causing fatal arrythmias. In such cases, calcium should be given over 30 minutes with continuous monitoring.

b) Driving extracellular potassium into the cells

1. Administration of 50ml of Glucose 50% containing 10units of Actrapid insulin infused over 20 minutes. This will require half hourly monitoring of capillary blood glucose (CBG) for 4 hours after. Insulin acts to drive potassium into cells and as it brings down the serum potassium, it also carries a risk of hypoglycaemia. If necessary, an infusion of 10% dextrose should be started.

   This should bring down the serum K+ by approximately 1mmol and the effect lasts between 3-6 hours. The dose can be repeated several times if necessary, but should not delay definitive management.

   Note that if repeated doses are given, then this may reduce the effectiveness of subsequent dialysis as the majority of the K+ will have been driven intracellularly and can cause a rebound post-dialysis hyperkalaemia, so multiple doses should only be given in agreement with ITU or the renal team.

2. **Consider** giving 10-20 mg nebulised (not intravenous) salbultamol, which can provide an additive effect to insulin-dextrose in driving K+ into cells. This should **only be given as adjuvent therapy to insulin-dextrose, never as monotherapy.**

   ***Note** - this carries a **small risk of precipitating angina in patients with IHD**, particularly if they are tachycardic or have ESRF, so this should be used cautiously in such patients.

3. **Consider** - if the patient has CKD and a serum bicarbonate of <18, you could consider the use of 1.26% HCO3 solution given slowly over 4-6 hours *if the patient is not fluid overloaded or at risk of pulmonary oedema, but there is currently no trial evidence to support this practice.*

4. **Re-check K level** at 1-2 hours and again at 4-6 hours (or sooner if there is reason to suspect ongoing hyperkalaemia eg rhabdomyolysis, tumour lysis syndrome).

c. Removing excess potassium from the body

1. The definitive method of excess K+ removal is haemofiltration (in ITU) or dialysis. The ITU team should be contacted to evaluate the patient's suitability if these are necessary.

2. Treating the cause of hyperkalaemia is the most important step after the initial emergency management, which will often be adequate to prevent ongoing hyperkalaemia.

3. Other options for less severe hyperkalaemia include use of loop or thiazide diuretic (in normal or mild CKD only), possibly in combination with a saline infusion, to enhance renal potassium excretion. This is only possible if there is no dehydration, AKI or hypotension).

4. Calcium resonium should not be used in the acute setting as it acts to reduce upper GI potassium absorption and is only effective with repeated doses. It causes severe constipation and has been associated with intestinal necrosis, so should be used with caution and laxatives should be prescribed.

2. Mild -moderate hyperkalaemia (K levels of 5.5-6.5 mmol/L):

1. ECG - if this shows signs consistent with hyperkalaemia, then proceed as for severe hyperkalaemia.

2. If there are no ECG changes, then the key to management is to identify if this is in context of an acute severe illness such as AKI, DKA or rhabdomyolysis or in the context of a chronic illness (CKD) or other cause (eg drugs) in a well patient.

If the patient is well, the treatment options include

- Withdrawal of offending drug (e.g. ACE inhibitors, ARBs, Direct renin inhibitors, aldosterone antagonists, NSAIDS and non selective beta blockers)
- Loop or thiazide diuretic
- Oral bicarbonate if the patient has CKD 4+ and renal acidosis (HCO3 <22)
- Dietary modification (See appendix 2)
- Consideration of calcium resonium (with caution)

There is limited evidence for most of these strategies and decisions should be discussed with the renal team where possible.

- **Contacts (inside and outside the Trust including out-of-hours contacts)**

- ITU SpR (24 hours): Bleep 2613
- Whittington Renal Registrar (In hours): Bleep 3106
- Whittington DMR (24 hours): Bleep 3300
- Royal Free Renal Registrar (24 hours): contact via Whittington switchboard

Appendix 1

Causes of pseudo hyperkalaemia are:

- Sample from a drip arm
- Sample left for a prolonged period before analysis (K leak from cells)
- Haemolysis (the analyser in biochemistry detects haemolysis, and the lab will not report haemolysed samples)
- Contamination with EDTA from FBC samples
- High platelet counts spuriously high K results. This can be checked by repeating on a plasma sample (green top tube).

It is crucial to ascertain why the K is in all cases. In the majority of true hyperkalaemia cases at least one of the following situations is likely to be present:

Impaired excretion (common causes):

- **Worsening renal function** (check previous creatinine readings)
- **Dehydration** (Decreased Na/K exchange in distal nephron)
- **Drugs** (Numerous culprits exist but commonly Spironolactone and ACEi/A2RBs.

Common unrecognised agents include NSAIDs and Trimethoprim):

- **Systemic acidosis** (check serum bicarbonate level)
- **Corticosteroid or Mineralocorticoid deficiency or insensitivity**

Excessive loading into circulation (often in the context of poor renal function):

- **Supplements** (oral or intravenous)
- **Release from cells** (e.g. necrosis, haemolysis, diabetic keto-acidoisis)
- **High K diet** (only an issue if very low GFR)

Appendix 2

Low Potassium Diet:

Potassium is a mineral found in most foods. It plays an important role in keeping your heartbeat regular and your muscles working well. If your potassium levels are too high you may be advised to follow a low potassium diet.

This information sheet is to help you decrease the amount of potassium in your diet before you see the dietitian. This is not an exhaustive list. It is important that a dietitian assesses you and tailors advice to your individual needs.

Fruit and vegetables: contain potassium and so you are advised to limit your intake to 2 serving of vegetables & 2 serving of fruits per day	
One serving of Fruit would be:	One serving of Vegetables would be:
1 medium apple ½ medium orange ½ small banana Tinned fruit – any kind but no juice 4 oz. glass fruit/vegetable juice 10 Strawberries or Grapes ½ cup Raspberries	1 tablespoon of broccoli 3 tablespoons of carrots 3 tablespoons of onions 3 tablespoons of frozen peas, pumpkin, seakale, spring greens or swede ½ tomato, 15 slices of cucumber
Vegetables should be boiled and water thrown away – avoid steaming or using a microwave or pressure cooker to cook veg	

Starchy: include something starchy at each meal but choose the lower potassium options listed below	
Lower potassium alternatives	**High in Potassium**
White bread, chapatti, noodles, white pasta, rice, cereal eg puffed rice, cornflakes	Wholemeal bread, naan, Malt bread, soda bread, fruit bread, ciabatta, rye bread, chips, potatoes, potato waffles, cereals with dried fruit, chocolate or nuts eg fruit and fibre, muesli, coco pops
Potatoes, yam or cassava should be boiled and water thrown away. Aim to limit intake to 3 egg sized potatoes or one small portion of cassava or yam per day as a maximum	

Dairy: dairy foods also contain potassium and so you will need to be careful with the amounts you eat. See below for daily limits
200ml (1/3 pint) of milk per day or 1 medium pot of yogurt per day or 30g of cheese per day If you have milk alternatives such as rice or oat milk, you do not need to limit your intake. Soya milk is lower in potassium compared to cow's milk; you can have up to 400ml/day of soya milk.

Sweets and savoury: Aim to choose the lower potassium alternatives

	Lower in potassium alternatives	High in potassium
Cakes, Biscuits and sweets	Plain sponge cakes, plain biscuits. Boiled sweets, jelly sweets, mints	Biscuits or cakes containing dried fruit and nuts. Solid Milk Chocolate, toffee, caramels, fudge, liquorice, Burfi
Condiments/ Seasoning	Fresh Herbs, salad cream, pepper, mayonnaise, mustard.	Yeast extracts, beef extracts, stock cubes, bottled sauces, ketchups, pickles, chutneys, dried herbs. SALT Substitutes All Soups –packet, tinned and homemade
Savoury Snacks	Snacks made from wheat, corn or rice	Potato snacks, all kinds of nuts. Bharji, Bombay Mix, Nuts

Beverages: Aim to choose the lower potassium alternatives	
Lower potassium alternatives	**High in potassium**
Tea (including fruit teas), fizzy drinks and dilutes squash	All fruit juices, High Juice drinks, chocolate/Cocoa, malted drinks, brewed coffee, beer, lager, stout, cider and sherry.

- **References (evidence upon which the guideline is based)**

1. **CLINICAL PRACTICE GUIDELINES TREATMENT OF ACUTE HYPERKALAEMIA IN ADULTS** UK Renal Association 2014. Lead Author: Dr A Alfonzo. Posted at www.renal.org/guidelines

2. Treatment and prevention of hyperkalemia in adults, David B Mount MD. Article on UpToDate.com, last updated 28/07/2015. Accessed 6/3/16.

- **Compliance with this guideline (how and when the guideline will be monitored e.g. audit and which committee the results will be reported to) Please use the tool provided at the end of this template**

An audit will be conducted following the publication of the guideline, which will be presented at the Trust Audit meeting.

To be completed and attached to any procedural document when submitted to the appropriate committee for consideration and approval

		Yes/No	Comments
1.	**Does the procedural document affect one group less or more favourably than another on the basis of:**		
	• Race	No	
	• Ethnic origins (including gypsies and travellers)	No	
	• Nationality	No	
	• Gender	No	
	• Culture	No	
	• Religion or belief	No	
	• Sexual orientation including lesbian, gay and bisexual people	No	
	• Age	No	
	• Disability - learning disabilities, physical disability, sensory impairment and mental health problems	No	
2.	**Is there any evidence that some groups are affected differently?**	No	
3.	**If you have identified potential discrimination, are any exceptions valid, legal and/or justifiable?**	No	
4.	**Is the impact of the procedural document likely to be negative?**	No	
5.	**If so can the impact be avoided?**	N/A	
6.	**What alternatives are there to achieving the procedural document without the impact?**	N/A	
7.	**Can we reduce the impact by taking different action?**	N/A	

If you have identified a potential discriminatory impact of this procedural document, please refer it to the Director of Human Resources, together with any suggestions as to the action required to avoid/reduce this impact.

For advice in respect of answering the above questions, please contact the Director of

Human Resources.

Checklist for the Review and Approval of Procedural Document

To be completed and attached to any procedural document when submitted to the relevant committee for consideration and approval.

	Title of document being reviewed:	Yes/No	Comments
1.	**Title**		
	Is the title clear and unambiguous?	Yes	
	Is it clear whether the document is a guideline, policy, protocol or standard?	Yes	
2.	**Rationale**		
	Are reasons for development of the document stated?	Yes	
3.	**Development Process**		
	Is it clear that the relevant people/groups have been involved in the development of the document?	Yes	
	Are people involved in the development?	Yes	
	Is there evidence of consultation with stakeholders and users?	Yes	
4.	**Content**		
	Is the objective of the document clear?	Yes	
	Is the target population clear and unambiguous?	Yes	
	Are the intended outcomes described?	Yes	
5.	**Evidence Base**		
	Are key references cited in full?	N/A	
	Are supporting documents referenced?	N/A	
6.	**Approval**		
	Does the document identify which committee/ group will approve it?	Yes	
7.	**Dissemination and Implementation**		
	Is there an outline/plan to identify how this will be done?	Yes	
8.	**Document Control**		
	Does the document identify where it will be held?	Yes	
9.	**Process to Monitor Compliance and Effectiveness**		
	Are there measurable standards or KPIs to support the monitoring of compliance with and	Yes	

	Title of document being reviewed:	Yes/No	Comments
	effectiveness of the document?		
	Is there a plan to review or audit compliance with the document?	Yes	
10.	**Review Date**		
	Is the review date identified?	Yes	
	Is the frequency of review identified? If so is it acceptable?	Yes	
11.	**Overall Responsibility for the Document**		
	Is it clear who will be responsible for co-ordinating the dissemination, implementation and review of the document?	Yes	

Executive Sponsor Approval			
If you approve the document, please sign and date it and forward to the author. Procedural documents will not be forwarded for ratification without Executive Sponsor Approval			
Name		Date	
Signature			
Relevant Committee Approval			
The Director of Nursing and Patient Experience's signature below confirms that this procedural document was ratified by the appropriate Governance Committee.			
Name		Date	
Signature			
Responsible Committee Approval – only applies to reviewed procedural documents with minor changes			
The Committee Chair's signature below confirms that this procedural document was ratified by the responsible Committee			
Name		Date	
Name of Committee		Name & role of Committee Chair	
Signature			

Tool to Develop Monitoring Arrangements for Policies and guidelines

What key element(s) need(s) monitoring as per local approved policy or guidance?	Who will lead on this aspect of monitoring? Name the lead and what is the role of the multidisciplinary team or others if any.	What tool will be used to monitor/check/observe/Asses s/inspect/ authenticate that everything is working according to this key element from the approved policy?	How often is the need to monitor each element? How often is the need complete a report ? How often is the need to share the report?	What committee will the completed report go to?
Element to be monitored	**Lead**	**Tool**	**Frequency**	**Reporting arrangements**