A Clinical Guideline for the Management of Hyperkalaemia in Adults

Document Control:

For Use In:	Norfolk and Norwich University Hospitals NHS Foundation Trust and James Paget University Hospitals NHS Foundation Trust		
	Emergency departments and all adult wards		
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Version	Date	Author	Reason/Change
JCG0020 v1	31 July 2014	THCGAP	Change of header and reference to joint hospital version
JCG0020 v2	03 March 2017	Dr C Ross, Mr N Weavers	Addition of an alternative insulin / glucose treatment option (8 units actrapid in 100mL 20% glucose)
JCG0020 v3	01 October 2018	Dr C Ross	Change in dose of Calcium Gluconate. Advice on (K^+) and (Glucose) monitoring.
CG9078 v4	30 June 2022	Dr R Varma Miss N Korn	Change of trust template to newest version, change from joint clinical guideline to NNUHFT trust

			guideline Change in insulin/glucose treatment option based on renal association hyperkalaemia guideline Addition of sodium zirconium cyclosilicate
CG9078v 5	25 July 2023	Dr R Varma Dr J Patrick Miss N Korn	Change in administration rate for calcium gluconate Change in monitoring frequency for potassium in moderate or severe hyperkalaemia
CG9078v 6	July 2024	Miss N Korn	Transferred to procedural document template

Previous Titles for this Document:

Previous Title/Amalgamated Titles	Date Revised
None	Not applicable

Distribution Control

Printed copies of this document should be considered out of date. The most up to date version is available from the Trust Intranet.

Consultation

The following were consulted during the development of this document:
Dr Mahzuz Karim – Consultant – Renal
Dr M Karim, Dr M Mahdi Althaf, Dr M Andrews, Dr A Friedla, Dr M Todd, Dr T Marshall, Ms H Willimott, Ms B Tedder.

Monitoring and Review of Procedural Document

The document owner is responsible for monitoring and reviewing the effectiveness of this Procedural Document. This review is continuous however as a minimum will be achieved at the point this procedural document requires a review e.g. changes in legislation, findings from incidents or document expiry.

Relationship of this document to other procedural documents

This document is a clinical guideline applicable to Norfolk and Norwich University Hospitals NHS Foundation Trust please refer to local Trust's procedural documents for further guidance, as noted in Section 5.

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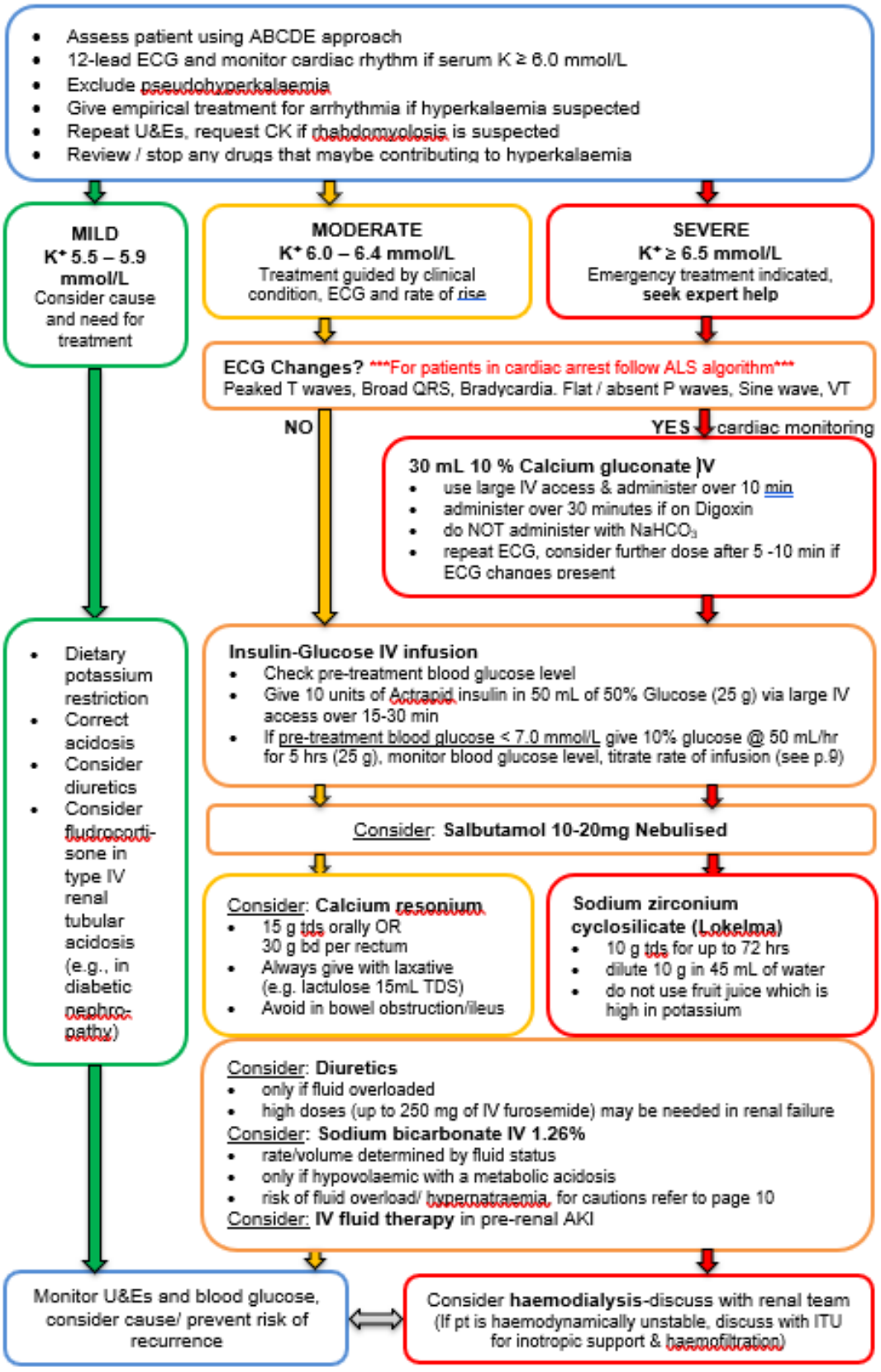

1. Introduction

1.1 Rationale

Hyperkalaemia is a life-threatening condition, primarily due to its effect on the heart. It is defined as a serum potassium level higher than 5.5 mmol/L. It is seen in 1.1% to 10% of all hospitalised patients, with approximately 1% having significant hyperkalaemia of greater than 6.0 mmol/L and is associated with a high mortality rate (14.3% to 41%).

In most patients, the pathophysiology of hyperkalaemia is multifactorial, with reduced renal function, medications, acidosis, and hyperglycaemia being the most common contributing factors.

1.2 Objective/s

To improve the management of hyperkalaemia in adult patients (18 years or over) in the Trust.

1.3 Scope

Treatment of hyperkalaemic cardiac arrest is outside the scope of this guideline, the relevant algorithms and guidelines should be followed.

1.1 Glossary

The following terms and abbreviations have been used within this document:

Term	Definition
AKI	Acute kidney injury
bd	Twice daily
CK	Creatin kinase
ECG	Electrocardiogram
g	gram
hr / hrs	Hour / hours
ITU	Intensive therapy unit
IV	intravenous
K	Potassium
L	litres
mmol	millimoles
Na	Sodium
$NaHCO_3$	Sodium bicarbonate
min	minutes
mL	millilitres
tds	Three times daily
U&Es	Urea and electrolytes
VT	Ventricular tachycardia

2. Responsibilities

1.4 Medical staff

Medical staff are responsible for prescribing and monitoring of medicines used for treatment of hyperkalaemia according to this guideline

1.5 Nursing staff

Nursing staff are responsible for administering medicines used for treatment of hyperkalaemia according to this guideline.

1.6 Pharmacists

Pharmacists are responsible for advising medical staff and checking prescriptions, monitoring and administration of medicines used for treatment of hyperkalaemia as per recommendations within this guideline.

2. Background

2.1 Causes of hyperkalaemia

Pseudohyperkalaemia

This is an artefactual increase in serum K^+ due to its release from cells during or after venepuncture. Potential causes include:

- Increased K^+ efflux from local muscle due to fist clenching or prolonged tourniquet time.
- Thrombocytosis, leucocytosis and/or erythrocytosis
- Delay in processing of sample leading to cell lysis

Excessive potassium intake

This is usually only a problem if potassium excretion is impaired, e.g. in patients with renal failure, those with a type IV renal tubular acidosis (e.g. diabetic nephropathy), or patients on drugs such as potassium-sparing diuretics, ACE inhibitors, or angiotensin receptor blockers, renin inhibitors.

Red cell transfusion is a well-described cause of hyperkalaemia, typically seen in children or in massive transfusions, but also in patients with significant renal dysfunction. Risk factors for transfusion-related hyperkalaemia include the rate and volume of transfusion, the use of central venous infusion and/or pressure pumping, the use of irradiated blood, and the age of the blood infused.

It is worth noting that some commonly used laxatives contain potassium – Movicol®, Laxido®, Klean-Prep® and Fybogel®.

Lo-Salt is a commercial preparation containing 66% potassium chloride (KCl) . Patients with or at risk of hyperkalaemia should be advised not to use this as a salt substitute.

Some herbal medicines (e.g. alfalfa, dandelion, horsetail, milk weed, nettle and others) also contain potassium and should be avoided in patients with or at risk of hyperkalaemia.

Re-distribution

Several different mechanisms can result in an efflux of intracellular potassium, resulting in hyperkalaemia:

- Increased serum osmolarity as in people with diabetes with severe hyperglycaemia or as a result of hypertonic mannitol.
- Rhabdomyolysis may cause hyperkalaemia.
- Metabolic acidosis associated with inorganic ions is associated with hyperkalaemia. Acidosis causes extracellular movement of K^+: this is more profound with 'fixed' rather than organic acidosis.
- Digoxin inhibits Na^+/K^+-ATPase and therefore impairs uptake of potassium by skeletal muscle; thus digoxin overdose can result in hyperkalaemia.
- Non-selective ß-blockers can cause hyperkalaemia in part by inhibiting cellular uptake (but also through effects on renin-aldosterone system)
- Agents that depolarise skeletal muscle, such as succinylcholine, or that activate potassium-dependent amino acid exchangers, such as lysine or arginine, can also lead to hyperkalaemia.

Impaired renal potassium excretion

Renal failure of any cause will usually lead to impaired potassium excretion.

Hypoaldosteronsim will result in hyperkalaemia. Aldosterone promotes both kaliuresis and proton excretion in the cortical and medullary collecting ducts by a variety of mechanisms. Aldosterone deficiency or resistance (e.g. Addison's disease, pseudohypoaldosteronism, type IV renal tubular acidosis) will therefore result in hyperkalaemia.

Several drugs cause impaired renal potassium excretion.

Class	**Examples**	**Mechanism**
ACE inhibitors	Lisinopril, ramipril, perindopril	Inhibit conversion of angiotensin I to angiotensin II
Angiotensin receptor blockers	Losartan, candesartan, irbesartan	Inhibit activation of angiotensin receptor by angiotensin II
Aldosterone receptor antagonists	Spironolactone, eplerenone, drospirenone	Block aldosterone receptor activation
Potassium-sparing	Amiloride, triamterene (trimethoprim, co-trimoxazole	Block/inhibit collecting duct apical Na^+-Cl^- symport channels,

diuretics	and pentamidine are structurally similar to amiloride and can have similar effect)	decreasing gradient for potassium secretion
NSAIDs	Ibuprofen, diclofenac	Inhibit prostaglandin stimulation of collecting duct potassium secretion, inhibit renin release, can cause renal failure
Calcineurin inhibitors	Cyclosporin, tacrolimus	Inhibit basolateral Na^+/K^+-ATPase in collecting duct; also inhibit apical secretory potassium channels
Heparin and LMWH	Heparin sodium, dalteparin, enoxaparin	Inhibit aldosterone synthase, rate-limiting enzyme for aldosterone synthesis

3. Processes to be followed

3.1 Broad recommendations

Management of hyperkalaemia is dictated by the potassium level and the severity of ECG changes. There is an overlap between conservative (see later) and emergency treatment of hyperkalaemia. **For patients presenting with cardiac arrest – please follow ALS algorithm and relevant guidelines, medical treatment for hyperkalaemia should be given as per hyperkalaemic cardiac arrest algorithm.**

Emergency treatment is indicated if:

- There is **severe hyperkalaemia** (K^+ > 6.5 mmol/L)
- There are **hyperkalaemic ECG changes** – loss of P waves, prolonged PR interval, peaked T waves, widened QRS complexes, and sine wave development (hyperkalaemia may also be associated with bradycardia or complete heart block – in this situation, a temporary pacing wire and urgent dialysis is often necessary)

In situations where artefactual hyperkalaemia is a possibility, repeat U&Es should be taken, **but do not delay treatment whilst waiting for result** if any of the above are present.

Urgent management of hyperkalaemia is considered in three steps:

STEP 1: Antagonism of the cardiac effects of hyperkalaemia

STEP 2: Rapid reduction in serum potassium by redistribution into cells

STEP 3: Removal of potassium from the body

Note 1: It is mandatory that all patients with hyperkalaemia (K level ≥6.0 mmol/L) should have an urgent 12-lead ECG performed and, if there are any hyperkalaemic changes or if K level ≥ 6.5 mmol/L, should be on a cardiac monitor (minimum 3-lead continuous ECG monitoring).

Note 2: It is recommended that serum potassium is assessed at least 1, 2, 4, 6 and 24 hours after identification and treatment of moderate or severe hyperkalaemia..

Note 3: The following therapies are listed to explain the physiology and posology. A recommendation for a hierarchy of use is as per the attached algorithm.

Note 4: Some therapies are essentially holding-manoeuvres (Calcium, glucose/insulin, and salbutamol). In the absence of another treatable pathology (e.g. overt dehydration), definitive therapy – dialysis – will be necessary.

3.2 STEP 1: Antagonism of cardiac effects:

Calcium

Calcium reduces myocardial excitability in the face of hyperkalaemia. It is available as 10% calcium gluconate. **The dose is 30 mL of 10% of calcium gluconate, administered intravenously over 10 minutes via large IV access with continuous cardiac monitoring.** The effect is seen within 3 minutes and lasts 30-60 minutes. The dose may be repeated – after 5 to 10 minutes – if no effect is seen or if ECG changes recur after initial improvement. It MUST NOT be administered via a line containing bicarbonate as it will precipitate as calcium carbonate.

As an alternative to 10% calcium gluconate, 10% calcium chloride may be administered. The dose of 10% calcium chloride is 10 mL as it contains 3 times more calcium than calcium gluconate. Calcium chloride has been recommended in the setting of haemodynamic instability, including cardiac arrest.

Calcium should be used cautiously in patients taking digoxin as hypercalcaemia potentiates the action of digoxin and may precipitate myocardial toxicity. In this case, it is necessary to infuse it more slowly (over 30 min in 100 mL of 5% glucose) to allow for an even distribution of calcium in the extracellular compartment.

3.3 STEP 2: Redistribution of potassium into cells

Insulin and Glucose (off label use)

Insulin lowers serum potassium by stimulation of the Na^+/K^+-ATPase. This effect is reliable, reproducible, dose-dependent and effective.

In recent years, there have been multiple published reports on a high incidence of iatrogenic hypoglycaemia after administration of insulin and glucose. The most consistent risk factor for iatrogenic hypoglycaemia is a low pre-treatment blood glucose. Reducing the dose of insulin alone did not consistently reduce hypoglycaemic episodes, there is more evidence to support increasing the total glucose load to 50g. The lowest risk of severe hypoglycaemia was associated with continuous delivery of glucose.

The treatment regimen has been altered to reduce the incidence of iatrogenic hypoglycaemia.

The recommended treatment regimen is:

- Check pre-treatment blood glucose level prior to insulin and glucose administration
- Administer **10 units of actrapid insulin** (using an **insulin syringe**) with **50 mL of 50% glucose (25 g of glucose)**. This should be administered intravenously **via a central venous access device/large vein via a syringe pump** over 15-30 minutes. Monitor for phlebitis if 50% glucose is administered via a large peripheral vein.

  (NB-for alternative glucose regimens see box below)
- **If pre-treatment blood glucose < 7.0 mmol/L administer 10% glucose at 50 mL/hour for 5 hours (25 g)**
  - target blood glucose 4.0 – 7.0 mmol/L
  - titrate rate of infusion if required
- Monitor blood glucose as per advice below
- Anticipate and treat hypoglycaemia promptly

Alternative Glucose regimens providing 25 g of glucose include 125 mL of 20% glucose or 250 mL of 10% glucose**.** The 10 units of soluble insulin should be added to the total volume of glucose required and drawn up into 50 mL intravenous syringes (syringe pump maximum volume is 50 mL). If alternative glucose regimens are used the syringe pump will need to be changed promptly several times through treatment.

The effect on serum potassium begins in 10-20 minutes, peaks at 30-60 min, and lasts for 4-6 hours. In most patients, the serum potassium drops by 0.5-1.2 mmol/L with this treatment. The dose may be repeated if necessary.

Close monitoring of capillary blood glucose levels, for a minimum of 12 hours after administration of the insulin-glucose infusion, should be done at 0, 30, 60, 90, 120, 180, 240, 300, and 360, 480 and 720 minutes. If the patient has been started on a continuous glucose infusion titrate rate of infusion according to blood glucose levels. Seek advice from senior medical doctor if patient becomes hypoglycaemic (capillary blood glucose < 4 mmoL/L). If patient becomes hyperglycaemic whilst on 10% glucose infusion stop infusion and contact senior medical doctor for advice.

In hyperglycaemic patients with hyperkalaemia, insulin may be administered without glucose (in the case of diabetic ketoacidosis as a fixed rate insulin infusion) with close monitoring of serum glucose levels. Discuss with SpR or Consultant. For patients with diabetic ketoacidosis follow the national guideline for the management of adult patients with diabetic ketoacidosis. (Trust document ID: 1140)

$ß_2$-Adrenergic Agonists (Salbutamol) (off label use)

Salbutamol exerts its effects via activation of Na^+/K^+-ATPase.

The recommended dose is 10- 20 mg of nebulised salbutamol. Its effects are seen at about 30 minutes and peak at 90 minutes, lasting for 2-6 hours. It reduces serum potassium levels by 0.5-1.0 mmol/L. However, several studies have shown that a subset of patients is not responsive to the potassium-lowering effects of salbutamol, and as such it should not be used as a single agent in the management of hyperkalaemia. The effects of salbutamol are also attenuated in patients on ß-blockers and digoxin. It is unclear whether treatment with salbutamol has a significant additive effect to insulin on its own.

Treatment with salbutamol may cause a significant tachycardia and should be used in caution in those with ischaemic heart disease.

Sodium bicarbonate

Previously used routinely in the treatment of hyperkalaemia, sodium bicarbonate is now reserved for hyperkalaemia associated with renal failure and acidosis.

The recommended dose is 1.26% sodium bicarbonate infused intravenously at a rate determined by the patient's fluid status and degree of acidosis.

Hyperkalaemia and metabolic acidosis with cardiac arrest should be treated with 50 mL of 8.4% sodium bicarbonate (which is available on the arrest trolley).

Sodium bicarbonate should be used with extreme caution in the following situations:

- In anuric or hypervolaemic patients – the significant sodium load may result in symptomatic fluid overload
- Hypocalcaemia – bicarbonate causes precipitation of calcium; the resultant fall in ionised calcium may result in tetany or fits
- Patients with type 2 respiratory failure – potential for paradoxical acidosis within the central nervous system

3.4 STEP 3: Removal of potassium from the body

Intravenous fluids

Most cases of acute (or acute on chronic) kidney injury in hospital are a result of intravascular volume depletion (i.e. pre-renal). In these situations, correction of volume status with intravenous fluids may be sufficient to restore renal function and promote a kaliuresis.

Compound sodium lactate (Hartmann's solution) should not be used when hyperkalaemia is present as it contains 5 mmol/Litre of potassium).

Diuretics

In certain cases, increasing renal potassium elimination with diuretics may be adequate to lower total body potassium. However, in the setting of renal insufficiency (chronic or acute) the effectiveness of diuretic therapy may be limited. **The use of diuretics is only indicated for those patients who are fluid replete.**

In the acute setting, the diuretic most often used is intravenous furosemide. The dose will vary depending on renal function, but in those with significant renal impairment, up to 250 mg may be used to try and promote a kaliuresis: the effect is mild.

Sodium zirconium cyclosilicate

Sodium Zirconium Cyclosilicate (SZC) is a non-absorbed potassium binder that preferentially exchanges H+ and Na+ for K+ and ammonium ions throughout the entire gastrointestinal tract. The K+-binding capacity of SZC is up to 9 times greater than that of sodium polystyrenesulfonate. NICE has approved SZC as an option in the treatment of acute life-threatening hyperkalaemia alongside standard care in hospitalised patients

Correction phase: the recommended dose is 10 g orally three times a day until normokalaemia (serum K 4.0-5.0 mmol/L) is achieved. Usual treatment duration is 24-48 hours, maximum duration is 72 hours. Sodium Zirconium Cyclosilicate should be discontinued after 72 hours if normokalaemia not achieved.

Maintenance phase: a dose of 5 g daily can be prescribed once normokalaemia is achieved, this can be titratred up to 10 g per day or down to 5 g alternate days depending on serum K levels. The maintenance dose should be discontinued once hypokalaemia develops (serum K level < 4.0 mmol/L)

The contents of the sachet should be emptied into a glass containing approximately 45 mL of water and stirred well. The powder will not dissolve. Advise the patient to drink the tasteless liquid while still cloudy, if the suspension settles it should be stirred again.

Cation-exchange resins

Ion-exchange resins are cross-linked polymers containing acidic or basic structural units that can exchange either anions or cations on contact with a solution. The most commonly used cation-exchange resin used is Calcium Resonium®. The onset of action is slow (> 4 hours) and efficacy is unpredictable therefore it should only be used in conjunction with other measures in the management of acute hyperkalaemia. It is also poorly tolerated due to taste and constipation. Cation exchange resins have been associated with colonic necrosis (most commonly seen with sodium polystyrene sulfonate used in conjunction with sorbitol). Therefore, they should not be used in those with bowel obstruction or an ileus.

The recommended dose is 15 g orally three times a day; each dose should be given with 15 mL of lactulose to prevent constipation and to facilitate passage of the resin through the gut.

Calcium Resonium® may also be administered rectally in those unable to take or tolerate it orally. **The recommended dose is 30 g as an enema retained for 9 hours and followed by irrigation**. (NB: For rectal administration please refer to summary of product characteristics, it can be administered rectally as a suspension of 30 g resin in 150 ml of water or 10% dextrose, as a daily retention enema.)

Extracorporeal potassium wasting – dialysis

All modes of renal replacement therapy are effective in removing potassium, with haemodialysis being the most rapid. Haemodialysis is indicated when hyperkalaemia is refractory to medical management.

If haemodialysis is likely to be necessary, it is important to enlist the help of the renal team at an early stage.

In patients who are haemodynamically unstable, haemodialysis may not be appropriate, and they may instead need haemofiltration – early consultation with the ITU team will be necessary.

Other measures

If a rapidly reversible cause of renal failure is identified, such as obstructive uropathy, then treatment of the underlying cause with close observation may be adequate to treat the hyperkalaemia.

All potentially offending drugs should be stopped immediately.

Although not indicated for acute hyperkalaemia, mineralocorticoid therapy (e.g. fludrocortisone starting at 50 microgram daily) may be indicated as chronic management of hyperkalaemia in patients with type IV renal tubular acidosis (e.g. diabetic nephropathy, sickle cell disease).

Patients with chronic renal failure and a chronic metabolic acidosis may benefit from long-term oral sodium bicarbonate (usually started at 500mg TDS) for chronic management of hyperkalaemia.

All patients should be placed on a low potassium diet. Patients have a sign "low potassium diet" added to beds/handover for catering to be alerted, not all patients remain on a low potassium diet once hyperkalaemia resolved hence it is not appropriate to give a leaflet to all patients. If renal fct doesn't improve and the patient remains under care of nephrology most patient will be seen by a renal dietician at some point (in particular if commencing renal replacement therapy); the available leaflets:

- Kidney Disease: Controlling your Potassium if you have Diabetes – Trust Doc ID 14863.

- Kidney Disease - Controlling Your Potassium (Trust Doc ID: 107) will be handed out to relevant patients in a clinic environment/ by dieticians if appropriate

4. References

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5. Monitoring Compliance

Compliance with the process will be monitored through the following:

Key elements	Process for Monitoring	By Whom (Individual / group /committee)	Responsible Governance Committee /dept	Frequency of monitoring
The management of hyperkalaemia in (non-dialysis) patients with a [K+] >6.5 (identified via ICE).	Audit	Renal Clinical Governance Lead	Renal	3 yearly

The audit results are to be discussed at relevant governance meetings to review the results and recommendations for further action. Then sent to Clinical Standard Group and Effectiveness Sub-Board who will ensure that the actions and recommendations are suitable and sufficient.

6. Appendices

There are no appendices for this document.

7. Equality Impact Assessment (EIA)

Type of function or policy	Existing

Division	1 - Medicine	**Department**	Nephrology
Name of person completing form	Nicola Korn	**Date**	July 2024

Equality Area	Potential Negative Impact	Impact Positive Impact	Which groups are affected	Full Impact Assessment Required YES/NO
Race	No	No	n/a	No
Pregnancy & Maternity	No	No	n/a	No
Disability	No	No	n/a	No
Religion and beliefs	No	No	n/a	No
Sex	No	No	n/a	No
Gender reassignment	No	No	n/a	No
Sexual Orientation	No	No	n/a	No
Age	No	No	n/a	No
Marriage & Civil Partnership	No	No	n/a	No
EDS2 – How does this change impact the Equality and Diversity Strategic plan (contact HR or see EDS2 plan)?		n/a		

- **A full assessment will only be required if: The impact is potentially discriminatory under the general equality duty**
- **Any groups of patients/staff/visitors or communities could be potentially disadvantaged by the policy or function/service**
- **The policy or function/service is assessed to be of high significance**

IF IN DOUBT A FULL IMPACT ASSESSMENT FORM IS REQUIRED

The review of the existing policy re-affirms the rights of all groups and clarifies the individual, managerial and organisational responsibilities in line with statutory and best practice guidance.