HEALTH HOLDING

HAFER ALBATIN HEALTH CLUSTER
MATERNITY AND CHILDREN HOSPITAL

Department:	Neonatal Intensive Care Unit (NICU)		
Document:	Departmental Policy and Procedure		
Title:	Guidelines for Management of Neonatal Non – Oliguric Hyperkalemia		
Applies To:	All NICU Staff		
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1. PURPOSE:

1.1 To provide guidelines for the safe and effective management of hyperkalemia.

1.2 Just Culture Principles: Maternity and Children Hospital Staff will not be held accountable for organizational or system issues or for human error that is not cause by deliberate reckless or risky behaviours. Just culture balances organizational accountability, system flaws and professional accountability and promotes learning from adverse events and near misses.

2. DEFINITONS:

2.1 **Hyperkalemia** – is defined as serum K>6.4 µmol/L in non – hemolysed arterial or venous blood sample. It is common for K+ to be greater than 6.4 µmol/L on a capillary specimen. Therefore, hyperkalemia should always be confirmed with a non – hemolysed arterial or venous specimen.

3. POLICY:

3.1 Hyperkalemia [serum potassium (K+) 6.4 µmol/L] has been reported in up to 50% ELBW premature infants. It may be associated with ECG disturbances (prolonged atrioventricular and ventricular conduction) and arrhythmias (sinus bradycardia or ventricular tachycardia).

3.2 Ninety – eight percent (98%) of total body K+ is intracellular at a concentration of 150 µmol/L, with the extracellular concentration 30 times lower.

3.3 **Hyperkalemia may result from:**

- 3.3.1 Increased K+ intake
- 3.3.2 Decrease excretion of K+
- 3.3.3 Shift of K+ from intracellular to extracellular space due to immature function of the erythrocyte Na+/K+ - ATPase (Sodium/Potassium adenosine triphosphatase). This is the etiology in non – oliguric hyperkalemia in preterm infant in the first few days after birth, which is unrelated to leakage following cell disruption associated with hypoxia, acidosis, hypoglycaemia, bruising, intracranial haemorrhage or hemolysis.
- 3.3.4 Extreme Prematurity
- 3.3.5 Renal Failure
- 3.3.6 Adrenal Insufficiency

3.4 In general, serum K+ less than 6.4µmol/L is well tolerated in preterm infants.

3.5 Calcium ions antagonize the membrane effects/arrythmogenicity of hyperkalemia.

3.6 Acidosis may increase hyperkalemia by shifting potassium from the intra- to extracellular space.

3.7 Treatment of hyperkalemia aims to:

3.8 Reduce the likelihood of arrythmia

3.9 Redistribute K+ into intracellular space and,

3.10 Remove K+ from the body.

4. PROCEDURE:

4.1 **Treatment (Appendix 7.1):**

4.1.1 Hyperkalemia should be treated when Potassium is >6.4µmol/L.

4.1.2 If no ECG abnormalities, confirm hyperkalemia on non – hemolysed arterial or venous specimen.

4.1.3 In presence of peak T wave or arrhythmia on ECG:

4.1.3 Calcium Gluconate 10% 0.11mmol/kg (0.5 ml/kg over 10 – 20 minutes).

4.1.3 Sodium Bicarbonate 4.2% (2µmol/kg) over 10 – 20 minutes (not simultaneously via the same line).

4.1.4 Discontinue any IV fluids containing K+.

4.1.5 Commence Insulin (0.1 units/kg/hour) with glucose 25% (250 microgram/kg/hour) via a central or umbilical line. Gradually reduce insulin and glucose over hours to prevent rebound hypoglycaemia and hyperkalemia.

4.1.6 Correct concomitant hypocalcemia. Salbutamol is a ß – adrenergic agonist that causes cellular potassium uptake by stimulating membrane bound Na+/K+ - ATPase (Sodium/ Potassium Adenosine Triphosphatase).

4.1.6.1 Infusion: 100 nanogram/kg/min over 24 hours or 4 – 5 microgram/kg over 15 – 20 minutes.

4.1.6.2 Inhalation: when compared with nebulised saline, serum K+ was reduced at 4 hours and at 8 hours after initiation of treatment. Inhaled Salbutamol appeared to be tolerated, but potential side effects include tachycardia, hypertension, tremor and hyperglycemia.

4.1.7 Investigate and treat underlying cause of acidosis.

4.1.8 Avoid oral and rectal resonium.

4.1.9 Cation – exchange resin (Resonium) does not effectively reduce serum K+ in non oliguric hyperkalemia in preterm infants. In addition, complications of rectal ion exchange resins include impaction and/or rectal perforation.

4.1.10 Insulin and glucose decreases serum K+ by transporting potassium into the intracellular space.

4.1.11 Exchange transfusion may be considered as a last resort where all other therapeutic option have failed. However, along preparation time is required to use the recommended saline washed RBC.

4.1.12 Peritoneal dialysis may also be considered as a last resort.

5. MATERIALS AND EQUIPMENT:

N/A

6. RESPONSIBILITIES:

6.1 Physician

6.2 Nurse

7. APPENDICES:

7.1 Algorithm for the Treatment of Neonatal Hyperkalemia

8. REFERENCES:

8.1 Kingdom of Saudi Arabia, Ministry of National Guard – Health Affairs, August – 2009.

8.2 Royal Women's Hospital Clinical Practice Guidelines (CPGs) 5 February 2009.

8.3 R Isaac, M Tajik, J Martin; September 2019.

9. APPROVALS:

	Name	Title	Signature	Date
Prepared by:	Ms. Afrah Saud Al Shammari	NICU Head Nurse		January 05, 2025
Prepared by:	Dr. Falah Nabhan Al Shammari	NICU Quality Coordinator		January 05, 2025
Reviewed by:	Mr. Sabah Turayhib Al - Harbi	Director of Nursing		January 07, 2025
Reviewed by:	Dr. Sarhan Hamdan Al Shammari	NICU Head of the Department		January 08, 2025
Reviewed by:	Mr. Abdulellah Ayed Al Mutairi	QM&PS Director		January 09, 2025
Reviewed by:	Dr. Tamer Mohamed Naguib	Medical Director		January 12, 2025
Approved by:	Mr. Fahad Hazam Al -Shammari	Hospital Director		January 19, 2025

APPENDIX I

Algorithm for the Treatment of Neonatal Hyperkalemia

Assess ABCDE

Perform 12 lead ECG or monitor continuous ECG
Repeat K^+ in laboratory
Stop all K^+ containing IV fluids/drugs affecting K^+

ECG Changes: Peaked/tented T waves; wide QRS; long PR interval; diphasic QRS ('sine wave'); flat/loss of P waves; VF/Asystole

Assess

Patient Age	SEVERE ECG changes / symptomatic	MODERATE No ECG changes / asymptomatic	MILD No ECG changes / asymptomatic
Neonate	$K^+ \geq$ 7.6mmol/L	K^+ 7.1-7.5mmol/L	K^+ 6.5-7mmol/L
	⇒ STEP 1+2+3	**⇒ STEP 2+3**	**⇒ STEP 3**

STEP 1

STEP 1: Most urgent! *Always* start with step 1 if severe hyperkalaemia, then continue to steps 2 and 3

Patient Age	**Give IV calcium** (stabilises the cardiac membrane to prevent arrhythmias) Can be repeated- see full guideline
Neonate	Calcium Gluconate 10% 0.5ml/kg over 10 minutes Give centrally whenever possible, via most distal lumen available If no central access available, dilute x 5 volume with Sodium Chloride 0.9%

STEP 2

STEP 2: Start with step 2 in cases of moderate hyperkalaemia, then continue to step 3

Patient Age	**Give Salbutamol** (moves K^+ into cells) Nebs can be rptd- see full guideline	**Give Insulin/Glucose** (moves K^+ into cells) Monitor blood glucose every 30 mins for 6 hours
Neonate	Give Nebulised Salbutamol as in Step 3 *or* IV Salbutamol 4 microgram/kg/dose over 5 minutes, diluted to 2ml with Sodium Chloride 0.9%	Add 0.1units/kg soluble insulin (actrapid) to 1g/kg of glucose. Give over 10 minutes. *(see Monograph/Appendix C for preparation)*
		Add 10 units of soluble Insulin (actrapid) to 50ml Glucose 50% or 125ml glucose 20% .Give over 5-10 minutes.

STEP 3

STEP 3: Start with step 3 in cases of mild hyperkalaemia

Patient Age	Nebulised Salbutamol (moves K^+ into cells)	IV Furosemide (increases K^+ elimination)	Calcium Resonium (increases K^+ elimination)
Neonate	2.5mg	1mg/kg/dose over 5 mins	Rectally 125-250mg/kg/dose four times a day

Monitor

Use the following table to record and assess response to treatment:

Monitor K^+/gluc until K^+ is: < 6.5 in those $\leq^1/_{12}$ < 5.5 in those $>^1/_{12}$	0min Time: ... : ...	15min Time: ... : ...	30min Time: ... : ...	45min Time: ... : ...	60min Time: ... : ...	90min Time: ... : ...	120min Time: ... : ...	4hours Time: ... : ...	6hours Time: ... : ...
K^+ (mmol/L)									
Glucose (mmol/L)									

Consider Causes of Hyperkalaemia:

Renal failure
DKA
Adrenal insufficiency (e.g. Addison's disease, CAH)
Pseudohyperkalaemia (esp. from haemolysis)
Drugs (e.g. K supplements, ACE inhibitors, β-blockers, suxamethonium, trimethoprim, diuretics)
Cell lysis (tumour lysis syndrome, rhabdomyolysis, severe burns, trauma)

Hyperkalaemia Management Guideline v1.0 Sept 2019; Review Sept 2021; R Isaac, M Tajik, J Martin