SydPath
ST VINCENT'S PATHOLOGY

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POTASSIUM INFORMATION SHEET

PHYSIOLOGY

Potassium is the major intracellular cation present at concentrations of about 150 mmol/L. Total body potassium is about 3000 mmol of which 98% is intracellular. An average diet includes about 100 mmol of potassium per day. Extracellular potassium is about 4 mmol/L. 60mmol/day is secreted into the gastro-intestinal tract, of which about 90% is resorbed (ie fecal losses are about 10-20 mmol/day). About 800 mmol of potassium is filtered at the glomerulus each day, of which nearly all is resorbed in the proximal tubule and Loop of Henle. Potassium excretion is controlled in the distal tubule and collecting ducts under the influence of aldosterone and other mineralocorticoids involving exchange with sodium or hydrogen ions. The obligatory daily urinary potassium loss is about 20-30 mmol with the average urinary excretion matching daily intake minus gastrointestinal losses. Changes in plasma potassium may be caused by changes in potassium intake or excretion, or by exchange with intracellular compartments. This may occur by cell lysis, pH changes and other factors such as insulin or digoxin.

HYPERKALAEMIA

Hyperkalaemia is often multifactorial, with renal failure and increased intake the most common causes. There are many causes of artefactual hyperkalaemia and if there is any doubt a repeat sample should be collected.

CAUSES OF HYPERKALAEMIA

Artefacts:

- Drip arm, EDTA contamination of sample
- Difficult collection, prolonged fist-clenching
- Thrombocytosis. Only occurs with serum (gold top tube), if suspected collect a heparin tube (lime-top)
- Leukocytosis (only in some leukaemias; may occur only in heparin tube (lime top), using serum tube (gold top) may avoid this issue in some patients)
- Delayed separation from red cells
- Haemolysis during collection (this is detected int he laboratory and results with-held)

Increased Intake:

- Oral (eg excessive fruit or vegetable juices)
- Intravenous (old blood, drugs eg potassium penicillin, potassium therapy)

Decreased output:

- Renal failure
- Hyponatraemia
- Addisons Disease or other mineralocorticoid deficiency
- Drugs (spironolactone, triamterene, amiloride, captopril, enalapril, heparin, NSAIDS)
- Hypoxia

Redistribution:

- Acidosis
- Tissue damage (eg tumour lysis syndrome, rhabdomyolysis)
- Insulin deficiency (eg during diabetic ketoacidosis)
- Digoxin overdose
- Intravascular haemolysis

EFFECTS OF HYPERKALAEMIA

Hyperkalaemia initially causes membrane excitability due to partial membrane depolarisation, then as level of serum potassium increases it may cause muscle weakness, arrhythmias, and eventually cardiac arrest. The ECG changes are: peaked T waves, decreased p waves, QRS widening, ST-T slurring, Sine wave, arrest.
Due to the prevalence of artefactual hyperkalaemia elevated levels should be checked, particularly if the finding is contrary to clinical expectations. With markedly elevated levels an ECG can be used to rapidly confirm the presence of hyperkalaemia.

ARTEFACTUAL HYPERKALAEMIA

Artefactual causes of hyperkalaemia are relatively common. An artefactual cause for elevated serum or plasma potassium should always be considered in a patient with high serum potassium in the presence normal renal function (eGFR>60 mL/min/1.73m2). Where possible the SydPath laboratory will identify samples with artefactual causes of hyperkalaemia and withhold results which may be affected (eg haemolysis, delayed separation, EDTA contamination) however full information is not always available. If in doubt please contact the laboratory for advice.

HYPOKALAEMIA

Hypokalaemia is most commonly due to reduced intake or increased renal losses. The effect of drugs, especially diuretics, should always be considered.

CAUSES OF HYPOKALAEMIA

Artefact:

- Drip arm dilution of sample

Reduced Intake

- "Tea and toast" diet
- Inadequate intravenous supply

Increased Output – Renal (spot Urine K > 20 mmol/L)

- Drugs: especially diuretics, amphoterocin, carbenicillin
- Mineralo-corticoid excess: eg aldosteronism (primary or secondary), Cushings syndrome, Bartters syndrome
- Osmotic diuresis
- Renal tubular acidosis

Increased Output – GIT

- Diarrhoea and vomiting
- Resonium treatment
- Fistulae

Redistribution

- Alkalosis
- Glucose/insulin
- Catecholamines
- Salbutamol
- Hypokalaemic periodic paralysis
- Thyrotoxic periodic paralysis