HYPERKALAEMIA

Hyperkalaemia is defined as a serum $K^+ > 5.5$ mmol/L.

AETIOLOGY.

The most common cause is *factitious hyperkalaemia* due to haemolysis during venesection.

It rarely occurs from excessive K^+ intake.

Renal insufficiency is a major cause including;

- defects in tubular secretion
- hypoaldosteronism
- Addison's disease
- Drugs;
    - ACEi
    - NSAIDS
    - Diuretics

Transcellular shifts with acidosis or beta blockade.

Drugs including digitalis and suxamethonium.

Hyperkalaemia is the most common metabolic cause of death in patients with ARF & results from an inability to excrete endogenous and exogenous potassium loads.

BOX 123-6 CAUSES OF HYPERKALEMIA

- Pseudohyperkalemia
  - Hemolysis of sample
  - Thrombocytosis
  - Leukocytosis
  - Laboratory error
- Increased potassium intake and absorption
  - Potassium supplements (oral and parenteral)
  - Dietary (salt substitutes)
  - Stored blood
  - Potassium-containing medications
- Impaired renal excretion
  - Acute renal failure
  - Chronic renal failure
  - Tubular defect in potassium secretion
    - Renal allograft
    - Analgesic nephropathy
    - Sickle cell disease
    - Obstructive uropathy
    - Interstitial nephritis
    - Chronic pyelonephritis
    - Potassium-sparing diuretics
    - Miscellaneous (lead, systemic lupus erythematosus, pseudohypoaldosteronism)
  - Hypoaldosteronism
    - Primary (Addison's disease)
    - Secondary
      - Hyporeninemic hypoaldosteronism (renal tubular acidosis type 4)
      - Congenital adrenal hyperplasia
      - Drug-induced
        - Nonsteroidal anti-inflammatory drugs
        - Angiotensin-converting enzyme
        - Heparin
        - Cyclosporine
- Transcellular shifts
  - Acidosis
  - Hypertonicity
  - Insulin deficiency
  - Drugs
    - Beta-blockers
    - Digitalis toxicity
    - Succinylcholine
  - Exercise
  - Hyperkalemic periodic paralysis
- Cellular injury
  - Rhabdomyolysis
  - Severe intravascular hemolysis
  - Acute tumor lysis syndrome
  - Burns and crush injuries

CLINICAL FEATURES.

CVS & neurological dysfunction are the primary manifestations of hyperkalaemia.

They may have a variety of dysrhythmias (including 2nd & 3rd degree HB, wide-complex tachycardia, VF and asystole).

- The ECG is invaluable in alerting the presence of hyperkalaemia.

Neuromuscular signs & symptoms of hyperkalaemia include muscle cramps, weakness, paralysis, paraesthesias, tetany & focal deficits.

ECG Changes Associated with Hyperkalemia

$[K^+]$ (mEq/L)	ECG Changes
6.5–7.5	Prolonged PR interval, tall peaked T waves, short QT interval
7.5–8.0	Flattening of the P wave, QRS widening
10–12	QRS complex degradation into a sinusoidal pattern

More on ECGs & Hyperkalaemia...

The earliest & best known manifestation = ***tall, symmetrically peaked T-waves***.

With escalating K^+ levels, there is further loss of conduction.

- Impairment of SA & AV node conduction = ***loss of P-waves***.

Progressive loss of transmembrane gradient = ***widening of QRS.***

Eventually, the QRS merges with the T-wave = ***sine wave***.

- Rapid deterioration to VF & asystole.

58-1 • ELECTROCARDIOGRAPHIC MANIFESTATIONS RELATED TO HYPERKALEMIA

Potassium Concentration	ECG Abnormality
Mild elevation: [K+] 5.5–6.5 mEq/L	Tall, symmetric, peaked T waves
Moderate elevation: [K+] 6.5–8.0 mEq/L	P wave amplitude decreases PR interval lengthens QRS complex widens Peaked T waves persist
Severe elevation: [K+] >8.0 mEq/L	P wave absent Intraventricular, fascicular, bundle branch blocks QRS complex widens, progressing to "sine wave" Ventricular fibrillation Asystole

MANAGEMENT.

All patients with suggested hyperkalaemia should receive;

- Cardiovascular monitoring
- Telemetry
- 12-lead ECG
- IV access
- Venous blood gas / point-of-care K^+
- Formal electrolytes / renal function etc.

Treatment of the hyperkalaemia is divided into three phases;

1. Membrane stabilisation (esp cardiac tissue)
2. Intracellular shift of K^+
3. Removal / excretion of K^+ from the body.

Calcium gluconate / chloride.

- Immediate antagonism of K^+ at cardiac membrane

Dose:

- Gluconate = 10-20mL of 10% solution (10mL = 1gram or 2.2mmol of calcium)
- Chloride = 5-10mL of 10% solution (10mL = *6.8mmol of calcium !!!)*

Onset = 1-3 minutes.
Lasts = 30-50 minutes.

Sodium Bicarbonate.

- Promotes intracellular shift of K^+ (*in the setting of metabolic acidosis*).

Dose:

- One ampule (50mL of 8.4% solution) = 50mmol.
    - Can be repeated 1-2 hours later.

Onset = 5-10 minutes.
Lasts = 1-2 hours.

Insulin & Dextrose.

- Induces cellular uptake of K^+.

Dose

- 5-10 units of regular IV insulin (Actrapid) *with*
- 1 ampule (50mL) of 50% dextrose = 25g glucose.

Onset = 30 minutes.
Lasts = 4-6 hours.

Beta-agonists.

- Causes movement of K^+ into cells.

Dose

- 5-20mg nebulised Salbutamol

Onset = 15-30 minutes.
Lasts = 2-4 hours.

Frusemide.

- Enhances renal excretion (*if passing urine*) with varying effect.

Dose

- 40mg IV.

Exchange Resins.

- Definitive treatment via increasing GI excretion

Dose

- 15-30grams PO or PR (Resonium) TDS-QID.

Onset = 1-2 hours.
Lasts = 4-6 hours.

Haemodialysis.

- Corrects K^+ rapidly, with removal from blood.
- Often used in severe lift-threatening causes and in rhabdomyolysis.
- Early consultation with Nephrologist.

CORRECT the UNDERLYING or PRECIPITATING CAUSE.

- Fluids for dehydration / pre-renal causes
- Removal of nephrotoxic agents (drugs in particular)
- Corticosteroids for Addison's.
- Digoxin-binding antibodies for Dig-overdose / toxicity
    - In severe hyperkalaemia, Calcium should be avoided in patients taking digoxin due to potential cardiac toxicity.