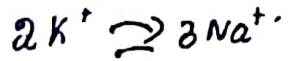


Hyperkalemia.

- Common electrolyte disorder, serum potassium $> 5 \text{ mmol/L}$ (3.5 to 5 mmol/L).

Cause Potassium inside cell - 98%, outside - 2%.



1) External Balance Shift.

- 1) Artefactual $\rightarrow$ During Venous puncture,
- 2) Increased intake
 $\rightarrow$ Diet
 $\rightarrow$ ↑ Intravenous fluids.
- 3) Decreased urinary excretion
 $\rightarrow$ Addison's disease
 $\rightarrow$ Kidney injury
 $\rightarrow$ Drugs inhibiting Renin, Angiotensin
 $\rightarrow$ Tubulointerstitial disease.

2) Internal Balance Shift.

- 1) Diabetes Mellitus Type I
- 2) Acidosis
- 3) Hyperosmolality
- 4) β_2 adrenergic receptor blocker
- 5) Cell lysis
- 6) Exercise.

Clinical features ↑ Resting Membrane Potential $\rightarrow$ ↑ Depolarization (Contractile)
 $\downarrow$ Repolarization

1) Smooth Muscle.

$\downarrow$
Severe Intestinal Cramps

2) Skeletal muscle

- $\downarrow$
- i) Weakness
 - ii) Flaccid paralysis (Starts from lower and upward)

iii) Cardiac muscle

- $\downarrow$
- Arrhythmia
 - Cardiac arrest.

Investigations

ECG $\rightarrow$ ↑ T wave, ST depression, Short QT interval,
 $\downarrow$ Sodium $\rightarrow$ Addison's disease, Renal function.

Treatment

$\rightarrow$ Insulin, Glucose, β adrenergic agonists, α Sodium Bicarbonate $\rightarrow$ Shift K^+ into cells.

Severe
 $\downarrow$
Prolonged PR Interval
Absent P wave
Widened QRS complex

1) External Balance Shift

1) Antefactual $\rightarrow$ During venous puncture or thrombocytosis
 $\rightarrow$ Injury to cells $\rightarrow$ $\uparrow$ Potassium outflow in blood.

2) Intake $\rightarrow$ Potassium rich intravenous fluids.
Diet $\rightarrow$ Rich in potassium.

3) $\downarrow$ Excretion $\rightarrow$

1) Hypoaldosteronism.
(Adrenal insufficiency)

2) Adrenocortical disease
(Primary adrenocortical insufficiency)

$\downarrow$ Aldosterone from zona glomerulosa
 $\downarrow$ $\uparrow$ Hyperkalemia & $\downarrow$ Hypokalemia.

2) Drugs inhibiting Renin, angiotensin, aldosterone.
 $\rightarrow$ Renin inhibitors, ACE inhibitors, Angiotensin II inhibitors,
selective aldosterone inhibitors, Potassium sparing diuretics.

3) Acute & Chronic Kidney Injury
 $\rightarrow \downarrow$ GFR $\rightarrow \downarrow$ oliguria & Hyperkalemia.

4) Tubulointerstitial disease
 $\rightarrow$ Interstitial nephritis, Diabetic nephropathy.

2) Internal Balance Shift

1) ^{Diabetes Type I} Insulin deficiency $\rightarrow \downarrow$ Intake of Glucose, ~~by~~ &
Potassium by cells.

2) Acidosis $\rightarrow$ Too acidic ($\downarrow$ pH) $\rightarrow \uparrow$ H^+ ions in blood.

$\hookrightarrow$ Compensatory mechanism $\rightarrow \uparrow$ H^+ ions inside cells
to ~~reduce~~ $\uparrow$ H^+ outside cells
increase pH in Blood

3) β_2 adrenergic receptors usually stimulate Na^+/K^+ pump
increased Na^+ outside cell by catecholamines

↓
 β_2 antagonists (β Blockers).

4) Hypertonicity inside cell $\rightarrow$ throws it out in Blood

5) Cell lysis (Burns, Rhabdomyolysis, Tumor lysis
↓ Breakdown of skeletal muscle Radiotherapy)

6) During Exercise $\rightarrow$ Potassium comes outside cell
due to usage of ATP.

Treatment

1) Calcium $\rightarrow$ stabilize myocardial cell membrane.

2) IV Insulin, IV Glucose, inhal β -adrenergic agonists, IV Sodium
Bicarbonate $\rightarrow$ shifts K^+ into cells.

3) Potassium wasting Diuretics,

4) Excretion $\rightarrow$ Dialysis

Resin binding to K^+ and excrete
through GIT.

↓
shifting
into cells.