DISORDERS OF POTASSIUM BALANCE

Potassium is the major intracellular cation

98% of total body potassium is intracellular, whereas only 2% is extracellular

which is crucial to normal functioning of nerve, muscle and cardiac tissues

The major regulators of this internal redistribution . are: (a) insulin, (b) catecholamines and (c) .mineralocorticoids

- the kidney is responsible for the excretion of some
 90% of the daily intake of potassium,
- typically 80-100 mmol/day. •
- The movement of potassium from blood to lumen is dependent on active uptake across the basal cell •membrane by the Na,K-ATPase

Factors modifying transcellular K+ distribution

Acid base status • Pancreatic hormones : insulin , glucagon • Catecholamines • Aldosterone • Plasma Osmolality • Exercise • Cellular K+ content •

Disorders of potassium homeostasis

(3.5-5.5)

Hypokalemia: Serum potassium <3.5 ● mmol/L (Potassium depletion: ICF or total potassium ↓)

Hyperkalemia: Serum potassium >5.5 mmol/L•



Acid Base Status

Alkalemia promotes K+ uptake by cells • Acidemia diminishes K+ uptake by cells •

Pancreatic Hormones

Insulin stimulates cellular uptake of K+ by • activating Na+K+ATPase (decreasing plasma K+)

Insulin affects K+ transport independently • of glucose uptake

Glucagon increase plasma K+ independently • of changes in plasma glucose / insulin

Catecholamines

Beta 2 adrenergic activity – hypokalemia •

Alpha adrenergic antagonists – • hypokalemia

Osmolality

Hyperosmolality (Mannitol infusion / • hyperglycemia in DM) : increase plasma K+

Each 10 mOsm / Kg rise in plasma • osmolality, increases plasma K+ by 0.6 mmol/l

Exercise

- Recurrent contraction increases K+ egress from muscle
- Modest exercise : high K+ in ECF in local environment produces vasodilatation & thereby increased regional blood flow
- Severe exercise : increase plasma K+ modestly •
- Physical training increases Na+K+ATPase activity in skeletal muscle which helps skeletal muscle to take up K+ again

Hyperkalemia is defined as a serum K⁺ level greater than 5.5 mmol per liter . (serum potassium >5.5mmol/L)

(2) Causes:

A. Diminished renal excretion

a. reduced GFR:

oliguria phase of acute renal failure terminal phase of chronic renal failure

 b. reduced tubular secretion of K⁺ addison's disease; hypoaldosteronism application of potassium sparing diuretic(spironolactone,

triamterene).

B. Increased input of potassium

excessive or rapid parenteral infusion of KCl solution

C. Extracellular shifts

- a. acidosis
- b. cell destruction(trauma, burns, hemolysis, tumor lysis, rhabdomyolysis
- c. Familial hyperkalemic periodic paralysis

Manifestations of hyperkalemia

Gastrointestinal manifestations •

Anorexia, nausea, vomitting, intestinal – cramps, diarrhea

Cardiovascular manifestations •

- Ventricular fibrillation and cardiac arrest –
- **Neuromuscular manifestations** •
- Paresthesias –
- Weakness –
- Muscle cramps –

Severe Hyperkalemia- • Absence of P waves Intraventricular blocks,BBB, Progressive widening of QRS complex Sine wave pattern ventricular fibrillation,asystole



EKG Changes Peaked T Waves

Symptomatic Hyperkalemia



EKG Changes Widening of QRS Complex

Severe Hyperkalemia



Treatment

- 1- Stabilize myocardial membrane •
- 2- Drive extracellular potassium into the cells
- 3- Removal of Potassium from the body •

TREATMENT OF HYPERKALAEMIA •

1-Stabilise cell membrane potential •

Intravenous calcium gluconate (10 ml of 10% • solution

2-Shift K into cells (•

Inhaled β agonist, e.g. salbutamol • Intravenous glucose (50 ml of 50% solution) and (insulin (5 U Actrapid Intravenous sodium bicarbonate 100ml of 8.4% solution

3-Remove K from body •

Intravenous furosemide and normal saline • 4-Ion-exchange resin (e.g. Resonium) orally or rectally 5-Dialysis

One commonly used regimen for • administering insulin and glucose is 10 units of **regular insulin** in 500 mL of 10 percent dextrose, given over 60 minutes. Another regimen consists of a bolus • injection of 10 units of regular insulin, followed immediately by 50 mL of 50 percent dextrose (25 g of glucose).

Hypokalemia Concept:() Serum potassium<3.5mmol/L

Causes:

a. Inadequate intake: Inability to eat;

Fast and anorexia;

Administration of K⁺ -free parenteral solutions

b. Excessive losses:

Gastrointestinal losses – Vomiting; Diarrhea; suction; Fistula. Renal losses – Polyurine phase of acute renal failure, Diuretic

therapy (except triamterene and spironolactone).

Increased mineralocorticoid levels – primary and second aldosteronism

Cushing's syndrome

Treatment with glucocorticoid hormones

c. Intracellular shift:

Alkalosis

Insulin treatment

Familial hypokalemic periodic paralysis

Barium poisoning

ECG changes with potassium imbalance.



Manifestations of hypokalemia

Neuromuscular manifestations

Muscle flabbiness, weakness and fatigue –

Muscle cramps and tenderness –

Paresthesia and paralysis –

Impaired kidney's ability to concentrate the urine • polyuria, urine with low osmolality, polydipsia (ECF – osmolality↑)

Gastrointestinal manifestations •

Anorexia, nausea, vomitting, –

Constipation, abdominal distension, paralytic ileus –

Cardiovascular manifestations •

Arrhythmias, increased sensitivity to digitalis toxicity – Metabolic alkalosis •

(4) Principle of treatment

a. In mild hypokalemia:

replenishment of KCl by the oral route is preferable because it is safer than I.V. administration.

b. In severe hypokalemia:

adding KCl to glucose water intravenously at a final concentration of 40 mmol/L, and infusing KCl at a rate of 10-20 mmol/h, since KCl is an irritating substance. Usually the repair of hypokalemia needs several days. Never give KCl solution by direct intravenous injection to patients.