HYPERKALEMIA AND HYPOKALEMIA

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INTRODUCTION

- Physiology of Potassium
- Roles of Potassium
- Hyperkalemia
- Hypokalemia

PHYSIOLOGY OF POTASSIUM

- Potassium is a major intracellular cation
- Total body K+ content in a normal adult : 3000- 4000mEq
- 98% Intracellular, 2% in ECF
- Normal homeostatic mechanisms maintain the serum K level within a narrow range (3.5-5.5 mEq/L).
- The intracellular concentration of Potassium is roughly 150 mEq/L, which is maintained by **Na^+/K^+ ATPase pump**.

ROLES OF POTASSIUM

- **Electrolyte Balance**: maintains fluid balance inside and outside cells
- **Nerve Function**: helps generate and regulate electrical signals necessary for nerve cell communication
- **Muscle Function**: regulates balance between contraction & relaxation of muscles
- **Blood Pressure Regulation**: lowers BP by vasodilation
- **Acid-Base balance**: acts as buffer to maintain an optimal pH range for proper physiological function
- **Metabolism & Energy production**: involved in various enzymatic reactions. (eg: conversion of glucose into glycogen)

- It's important to note that both low and high potassium levels can have adverse effects on health.
  - **Hypokalemia** (low potassium levels i.e. <3.5 mEq/L)
  - **Hyperkalemia** (high potassium levels i.e. >5.5 mEq/L)

  These can disrupt normal physiological processes and require appropriate medical management.

HYPOKALEMIA

Defined as plasma [K^+] <3.5meq/L

ETIOLOGY

Decreased Intake:
- Starvation
- Clay Ingestion

Redistribution into cell:
- Acid Base: Metabolic Alkalosis
- Anabolic State
  - Vit B12/Folic acid administration
  - Total Parenteral Nutrition
- Hormonal:
  - Insulin
  - Downstream stimulation of Na^+/K^+ ATPase: Theophylline
  - Beta-2 adrenergic agonist
- Others:
  - Pseudohypokalemia
  - Hypothermia

ETIOLOGY

<u>Increased Loss</u>:

- Non Renal
  - Gastro-intestinal Loss (Diarrhoea)
  - Integumentary Loss (Sweat)
- Renal
  - Diuretics, Osmotic diuretics
  - Increased secretion of K+ : Mineralocorticoid excess
  - Magnesium Toxicity
  - Vomiting, Diabetic Ketoacidosis, Proximal Renal Tubular Acidosis
  - Drugs:
    - Penicillin
    - Dicloxacilin

CLINICAL FEATURES

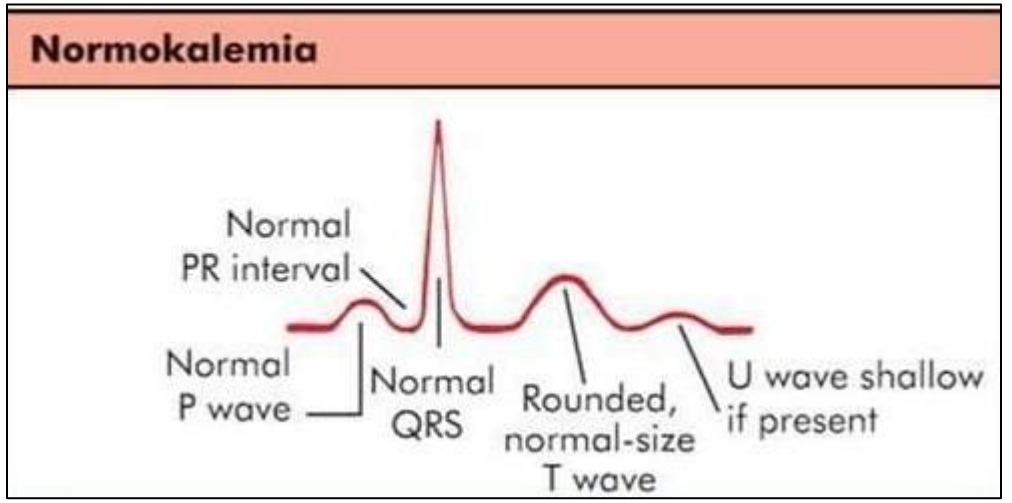
- Fatigue
- Myalgias
- Muscular weakness or cramps
- Constipation
- Frank paralytic ileus
- In **Severe Hypokalemia** :
  - Complete paralysis
  - Hypoventilation
  - Rhabdomyolysis

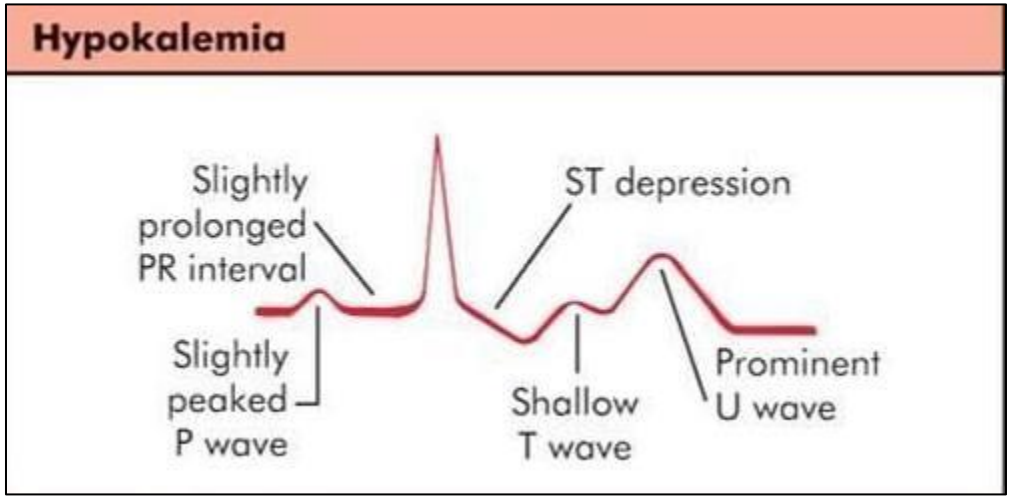
DIAGNOSTIC TESTING

- Urine Potassium level: measured by 24hr urine collection or by multiplying spot urine [K^+] by total daily urine output. (<15mEq/L suggests appropriate K^+ conservation)
- Alternatively, **TTKG** (Trans tubular potassium gradient)
  - TTKG= (Urine K x Plasma osm) / (Plasma K x Urine osm)
  - TTKG<2 suggests a non renal source
  - TTKG>4 suggests inappropriate renal potassium excretion.
- A Urine Potassium-to-creatinine ratio of <13 indicates transcellular potassium shift, GI losses, diuretic use or poor dietary intake; if >13, indicates renal potassium wasting.
- Acid-Base status: mostly associated with metabolic alkalosis

ECG CHANGES

- Flattening or Inversion T-wave
- Prominent U-wave
- ST segment depression
- Premature atrial complexes
- Premature ventricular beats
- Prolonged QU interval
- In **Severe K^+ depletion**:
  - Prolonged PR interval
  - Decreased voltage
  - Widening of QRS complex





TREATMENT

A total body Potassium deficit of **200-400 mEq** is required to lower the serum potassium levels by 1mEq/L. Hence, correction needs to be targeted to total body deficit.

Oral Therapy

- Oral replacement with KCl (Mainstay of therapy)
- Potassium phosphate (Oral or IV)
- Potassium bicarbonate/citrate (in pt. with concomitant metabolic acidosis)
- DOSE : Oral dose of 40mEq every 4 hrly.

IV Therapy

- IV replacement with KCl (<=40 mEq/L via peripheral veins; 100mEq/L via central line)
- Ideally KCl mixed with NS because dextrose might exacerbate hypokalemia
- Maximal daily dose: 240-400 mEq/day
- Rate of infusion <=20mEq/hour (unless paralysis or malignant ventricular arrhythmia
- Potassium sparing diuretics (Spironolactone, Amiloride, Triamterene, Eplerenone)

HYPERKALEMIA

Defined as plasma [K^+] >5.5meq/L

Pseudohyperkalemia

- Cellular efflux Thrombocytosis
- Hereditary RBC membrane defect

ETIOLOGY

Intra to Extracellular Shift

- Acidosis
- Hyperosmolarity
- Beta-2-adrenergic antagonist
- Digoxin

ETIOLOGY

Inadequate Excretion

- Inhibition of renin-angiotensin-aldosterone axis
  - ACE inhibitors, ARBs, ENaC blocker (Amiloride, Trimethoprim)
- Decreased distal delivery
  - Congestive Heart Failure
  - Volume Depletion
- Hyporeninemic hypoaldosteronism
  - Tubulointerstitial disease (SLE)
  - Diabetic nephropathy
  - Drugs: NSAIDs

- Renal resistance to mineralocorticoid
  - Hereditary: defect in ENaC
- Advanced renal insufficiency (CKD)
- Primary Adrenal insufficiency
  - Autoimmune: Addisons disease
  - Infectious: HIV, TB
  - Drugs: Heparin, LMWH

CLINICAL FEATURES

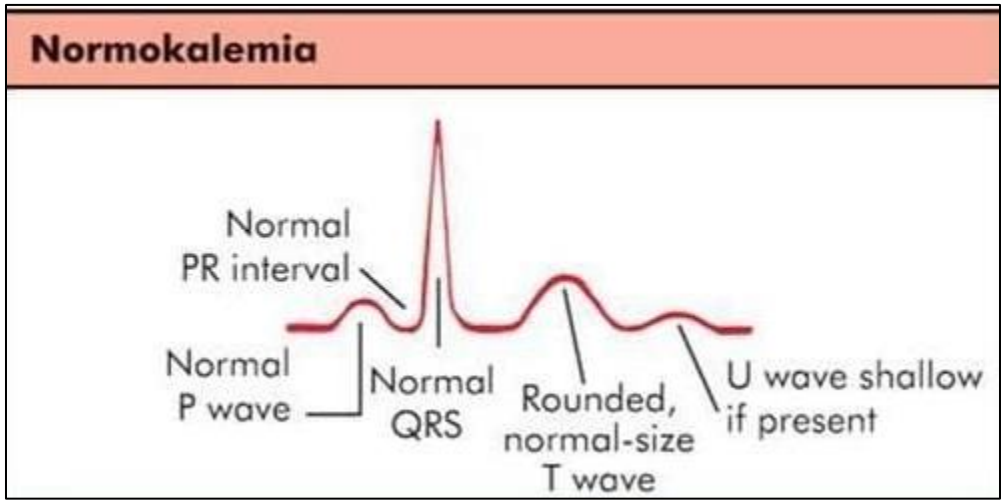
- Palpitations
- Syncope
- Cardiac Arrhythmogenesis (may lead to sudden cardiac death)
- **In severe hyperkalemia**
  - Weakness
  - Flaccid paralysis
  - Hypoventilation (if respiratory muscles involved)

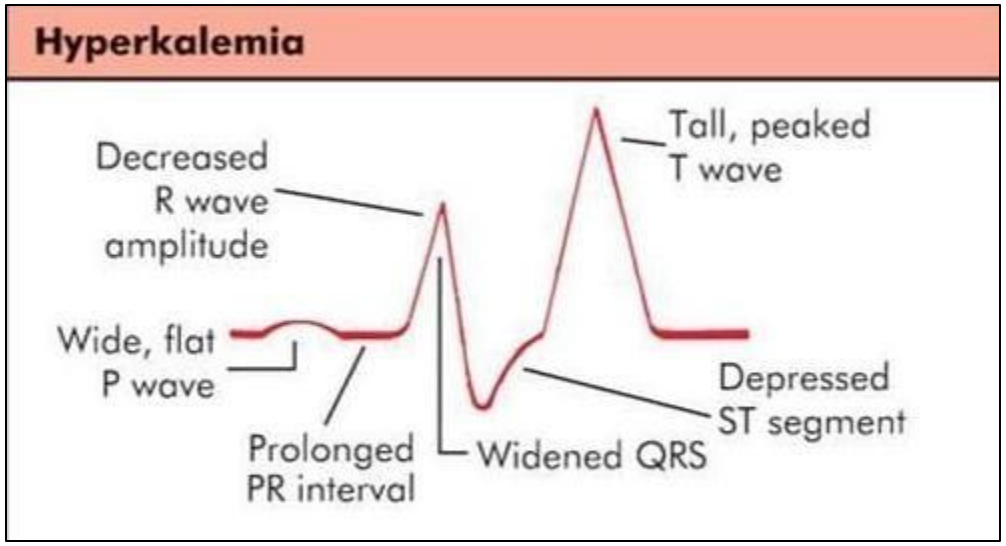
DIAGNOSTIC TESTING

- Pseudohyperkalemia should be excluded by rechecking laboratory data
- High Aldosterone levels
  - High renin levels suggest Aldosterone resistance (Pseudohypoaldosteronism)
- Low Aldosterone levels
  - Adrenal disease (Renin levels elevated)
  - Hyporeninemic Hypoaldosteronism (Renin level low) occurs with Type 4 Renal Tubular Acidosis

ECG CHANGES

Serum Potassium (mEq/L)	ECG
5.5-6.5	Tall, peaked T-waves
6.5-7.5	Loss of P-waves
7-8	Widening of QRS complex
8-10	Cardiac arrhythmia, sine wave pattern and ventricular fibrillation/asystole





TREATMENT

Acute Therapy

- **Calcium Gluconate** 1000mg(10ml) of a 10% infused over 2-3 minute, dose can be repeated if no ECG improvement seen after 5-10 minutes.
- **Sodium Bicarbonate**: 3 ampoules of NaHCO3 in 1 Liter of 5% Dextrose.
- **Beta-2-adrenergic agonist**: Albuterol (10-20mg neb. Over 30-60mins)
- **Insulin:** causes Potassium shift into the cell & temporarily lowers the plasma potassium.
    - 10-20 units of regular insulin (with 10/25/50% Dextrose IV)

Long term Therapy

- Administration of Saline in Pt. who appear volume depleted, otherwise Furosemide can be used if renal function is adequate
- **Dialysis** (Patients with renal failure)
- **Chronic Therapy**
  - Dietary modification to avoid high potassium foods
- Cation exchange resins(promotes excretion of Potassium in GI tract):
  - Sodium Polystyrene Sulphonate (SPS): 25-50gm
  - Zirconium cyclo-silicate
  - Patiromer: 8.4gm mixed with 100ml of water, given daily


Thank You