Hypokalemia

Dr Ummar

- Normal range of potassium is **3.5 to 5 mEq/L**
- Potassium is essential for Muscles, cardiovascular system, Central nervous system, respiratory system.
  - Maintain osmolarity of ECF and ICF and hence cellular volume
  - Regulation of acid base balance along with cellular growth, protein synthesis and hormonal secretion
  - Vital for cell excitability and muscle contraction
  - Maintainance of transmembrane electric potential.

Physiological Hemostasis:

- 98% of total body K+ is intracellular and chiefly in muscles. In a healthy individual steady state K+ excreted 90% in urine and 10% in feces.
- K+ absorbs from small intestine; through duodenum, jejunum and ileum. K+ mainly required for the below mentioned channels:
  - Na+K+ ATPase: almost all cells contains this pump; required for maintenance of ICF and ECF through electromechanical gradient (3 Na+ out & 2 K+ in)
  - H+K+ATPase: In GI cells and renal tubules (H+ out and K+ in)
  - Na+K+Cl- co transport: in salivary gland, GI tract and Renal tubules; brings 1 Na+, 1 K+ & 2 Cl- inside cell
  - K+Cl- Co transport: plays role in maintaining volume of erythrocytes.

Renal handling of potassium:

- Proximal convoluated tubules: 60% of K+ reabsorbption occurs through paracellualr K+ channels.
- In ascending thick part of Loop of henle K+ is reabsorbed by Na+K+Cl- co transport channel.
- Distal convoluted tubules and collecting duct:
  - major determinants of urinary K+ levels as K+ is secreted by principle cells.
  - Potassium secretion occurs in ‘principal cells’ by active uptake across the basolateral membrane by Na+/K+-ATPase and passive diffusion into the lumen across the apical membrane by K+ channels or using a K+ Cl_ cotransport. Increased concentration of Na+ makes K+ to secrete in lumen because increase in Na+ concentration potential difference across cell membrane and that makes K+ drive out of the cells.

- Reabsoption of K+ occurs through H+K+ATPase and Na+K+Cl- co transport through intercalated cells.
- Potassium homesostatsis is mainly done by renal system.
- In case of hypokalemia it reabsorbs the filtered K+ and in hyperkalemia it promotes secretion of K+ by principle cells.

- Pottasium rich diet:

- Bananaas, Kiwi, Mango, Oranges, Papaya, coconut water, fruit juice, spinach, sweet potato, tomato, pickles, beet, dry fruits, chocolate coffee.

- Serum level < 3.5 mEq/L defined as a hypokalemia

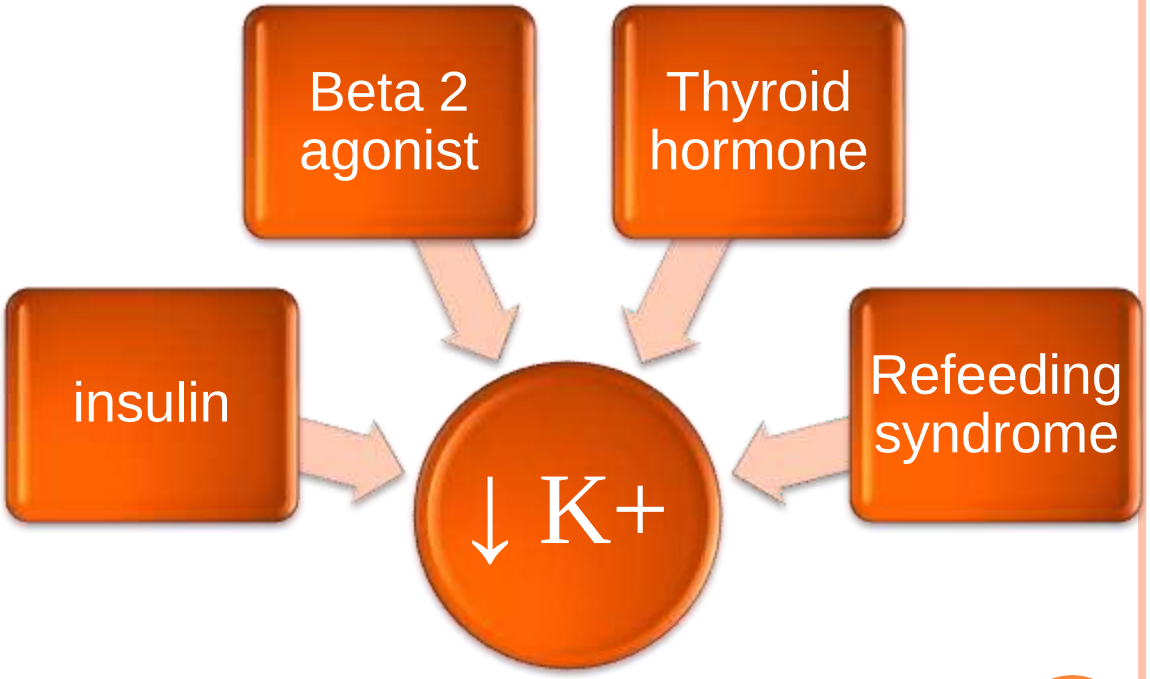
Causes:

- Psuedohypokalemia: Drip arm sample
- Reduced intake: starvation and dietary deficiency
- Magnesium deficiency: treatment resistance hypoK+
- Redistributive hypokalemia:
- Acid base disorder
  - Metabolic alkalosis

Redistributive Hypokalemia

- Increased cellular uptake

through Na+K+ ATPase



Alteration in sympathetic activity

- Alcohol withdrawl
- Thyrotoxic periodic paralysis
- Acute MI
- Head injury
- Sympathomimetic drugs:
  - Ephedrine
  - pseudoephedrine

  } **as in cough syrup**
- Rarely theophylline and caffine can cause downregulation of beta 2 receptor (Na+K+ATPase) causing hypokalemia
- Hypothermia, Familial Hypokalemic Periodic Paralysis, barium toxicity (inhibition of leak K+ channel)

Increased potassium loss

- **Renal loss:**
- **Increased distal Na delivery:**
  - Diuretics
  - Osmotic diuretics
  - Salt wasting nephropathies
  - Antibiotics: penicillin related, aminoglycosides, AMP B, cisplatin, ifofosphomide
- **Non renal loss:**
- **Infectious:** Diarrhoea and vomiting
- **Non infectious:**
  - Celiac disease
  - Ileostomy
  - Villous adenoma
  - VIPoma
  - Chronic laxative abuse
  - Colonic pseudo-obstruction

Increased secretion of K+
Hyperaldosteronism

Primary

- Genetic :
- Congenital adrenal hyperplasia
- Acquired:
- Aldosterone producing adenomas
- Adrenal hyperplasia
- Idiopathic

Secondary

- Malignant hypertension
- Renal artery stenosis
- Renin secreting tumour

Mineralocoticoid excess:

- Cushings syndrome
- Barters syndrome
- Liddle’s syndrome
- Gitelman’s syndrome.

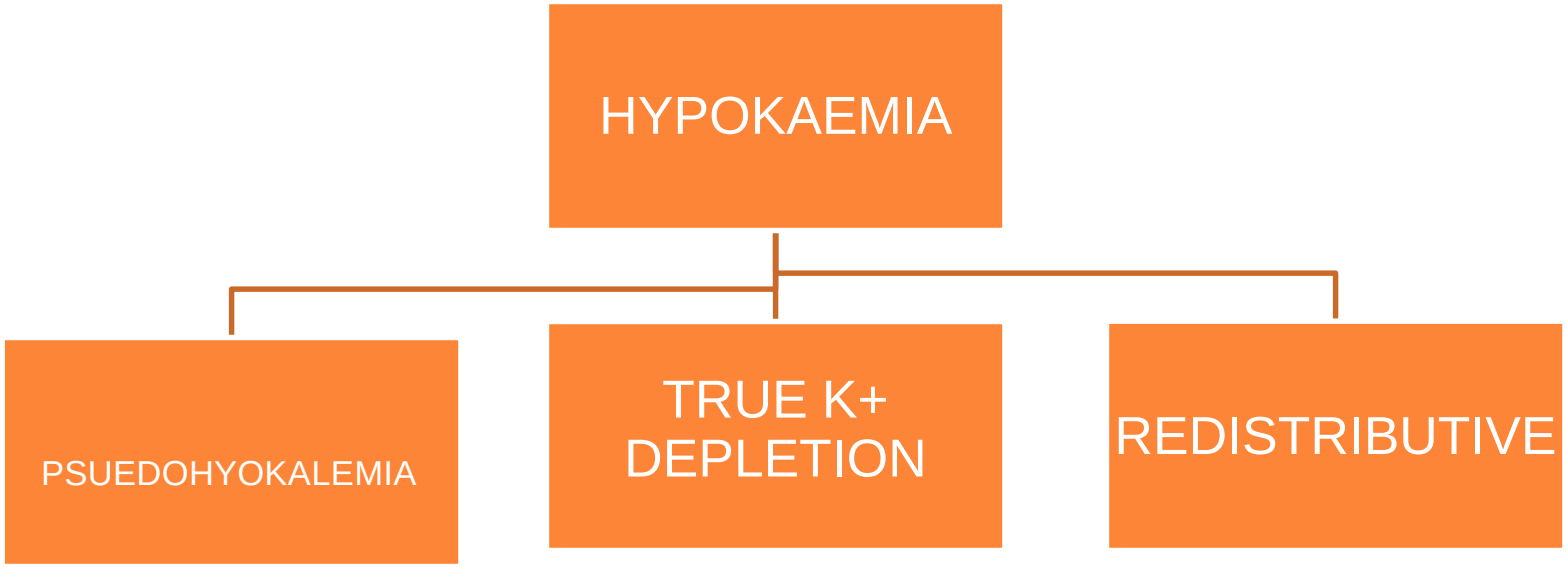
Clinical features:

- May be asymptomatic
- Fatigue, myalgia, LL weakness with depressed DTR
- Paralytic ileus, constipation
- Respiratory muscle weakness and complete paralysis
- Increased risk of arrythmia and heart failure; esp patients on digitalis treatment.
- No neurological presentation

ECG CHANGES IN HYPOKALEMIA (DO NOT CORRELATE WITH S. K+ LEVEL)

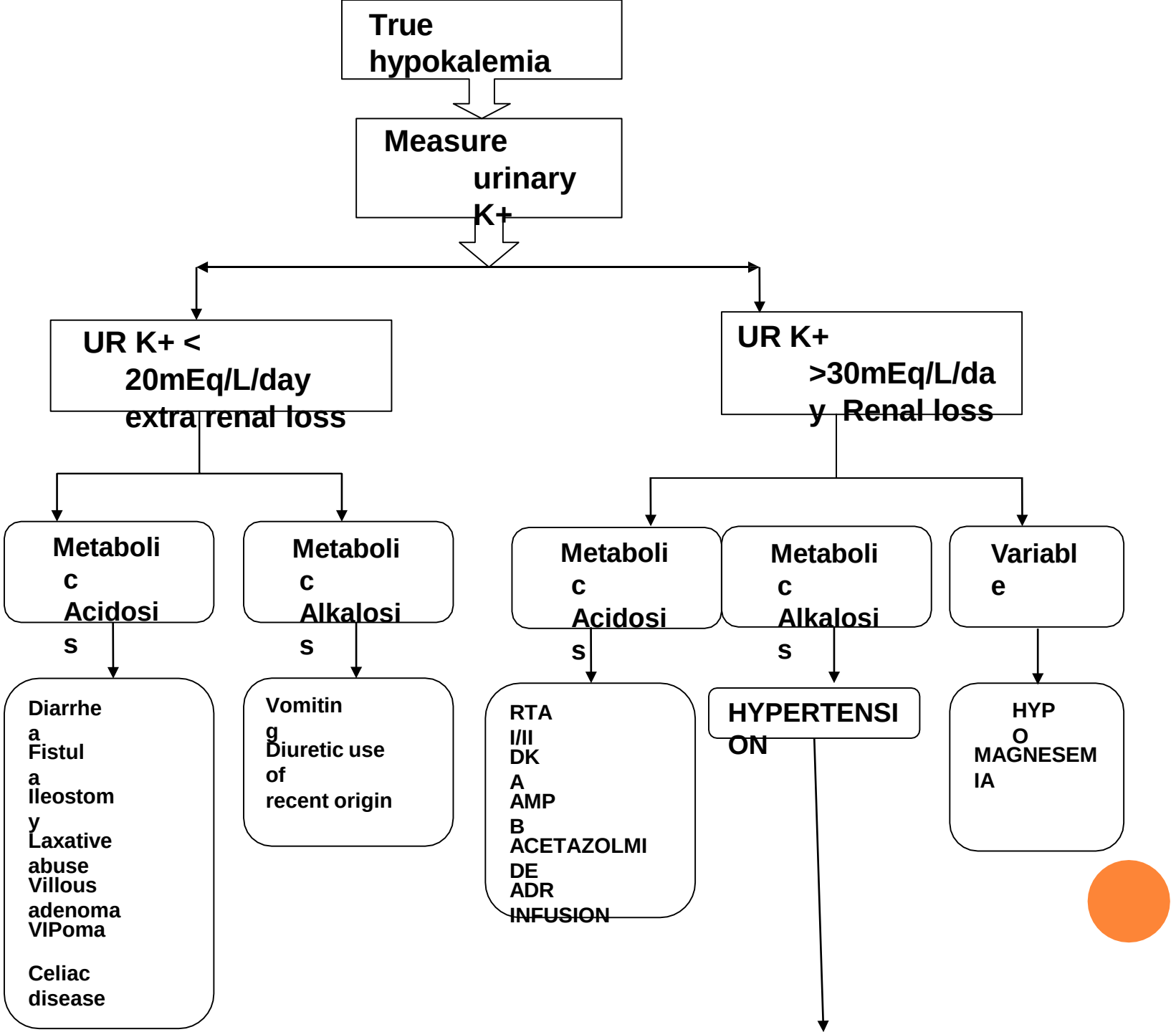
- **Early changes:**
- T wave inversion or flattening
- U wave
- ST segment depression
- Prolong QT interval
- **Severe K+ depletion**
- Prolong PR
- Low voltage ECG
- Wide QRS comples
- Ventricular arrythmia

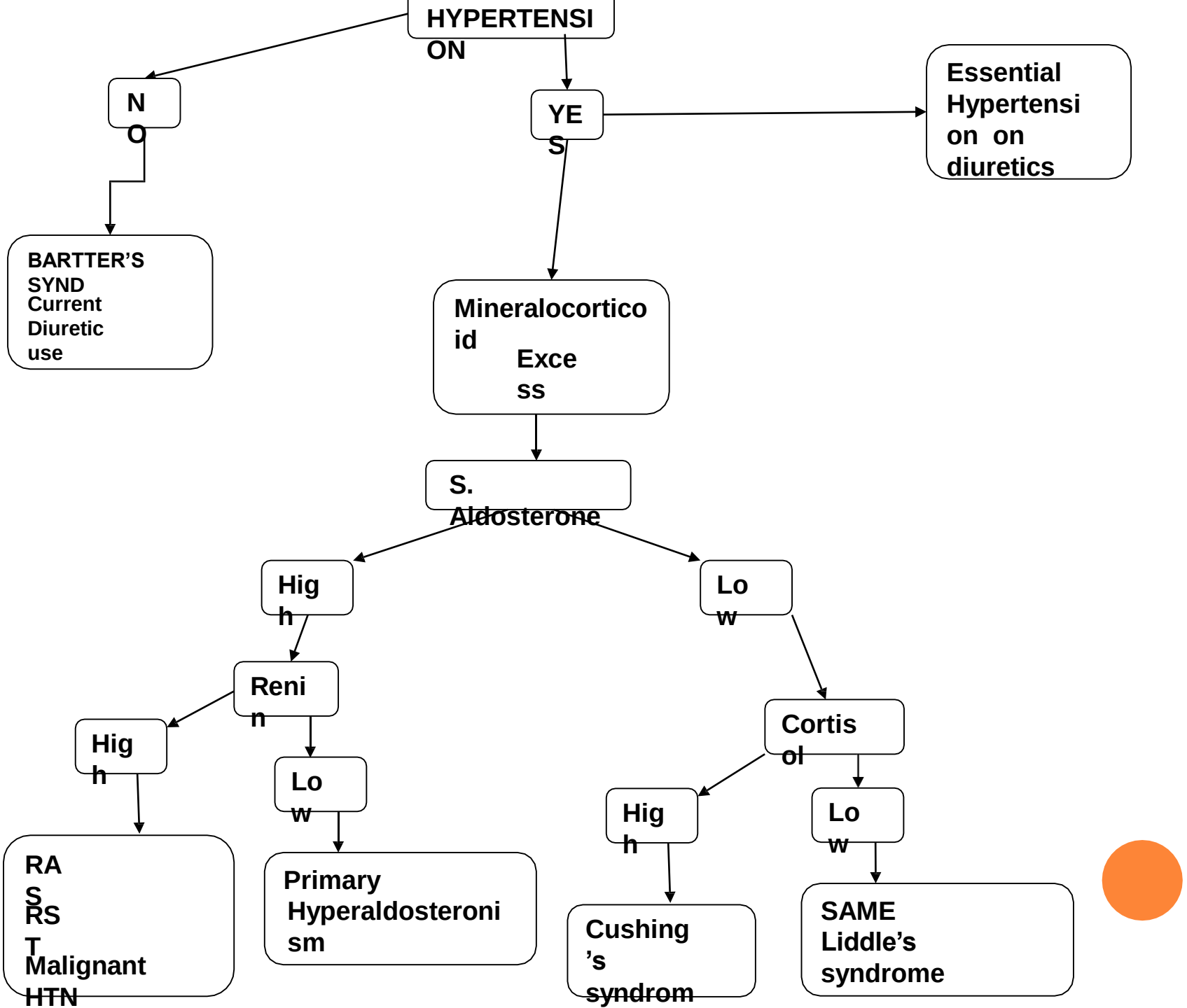
DIAGNOSTIC APPROACH TO HYPOKALEMIA



- Drip arm sample
- No treatment required
- Confirmed with repeat lab

Find out the cause for redistribution and treat accordingly


True hypokalemia
Measure urinary K+
UR K+ < 20mEq/L/day extra renal loss
UR K+ >30mEq/L/day Renal loss
Metabolic Acidosis
Metabolic Alkalosis
Metabolic Acidosis
Metabolic Alkalosis
Variable
Diarrhea
Fistula
Ileostomy
Laxative abuse
Villous adenoma
VIPoma
Celiac disease
Vomiting
Diuretic use of recent origin
RTA I/II
DKA
AMPB
ACETAZOLMIDE
ADR
INFUSION
HYPERTENSION
HYPO MAGNESEMIA


HYPERTENSION
NO
YES
Essential Hypertension on diuretics
BARTTER'S SYND
Current Diuretic use
Mineralocorticoid Excess
S. Aldosterone
High
Low
Renin
High
Low
RAS
RST
Malignant HTN
Primary Hyperaldosteronism
Cortisol
High
Low
Cushing's syndrom
SAME
Liddle's syndrome

THERAPEUTIC GOALS FOR TREATING HYPOKALEMIA

- Prevent life threatening complications like arrhythmia and respiratory failure
- Correction of underlying etiology
- Correct K+ deficiet
- Minimize ongoing losses
- Prevention of hypokalemia

Prevention of hypokalemia

- Normal daily intke: 60 mEq/L
- **Prophylactic K+ Supplements in patients taking**
- Digitalis
- Long term use of diuretics
- Larger doses of Steroids
- **Prevention of hypokalemia required in:**
- Digitalis therapy
- Hepatic failure
- Prev MI or IHD
- DM
- **Post op on TPN or IV fluid: Give 40-50 mEq/ day of K+**

Treatment Guidelines:

- **K+ level 3 to 3.5 mEq/L:**
- Treat in special high risk groups:
  - Risk of arrhythmia
  - Hepatic failure/ Congestive Cardiac Failure
  - Digitalis therapy
  - IHD/DM

- **K+ level below 3 mEq/L:**
- Definitive treatment thorugh IV route

Precautions:

- **Never give K+**
- In oligouric or anuric patient
- **Cautious use of K+:**
- pt on K+ sparing diuretics, ACEi, pt with renal failure
- Pt on digitalis therapy IV K+ infusion rate shoud be < 20 mEq/hr
- If rate > 20 mEq/hr, every pt needs to have continouos ECG monitoring and frequent S. K+ Level.

- Roughly fall of 1 mEq/L of S. K+ = 200 – 400 mEq body deficit
- When deficit of K+ about 200 to 400 mEq; 50 to 100 mEq/day of K+ slowly but adequately corrects deficit.
- KCl (potassium chloride) salts are the preapartion of choice for treating hypokalemia. It will correct hypokalemia and also metabolic alkalosis.
- Potassium bicarb and citrate will alkalize the patients and more appropriate for hypokalemia asso with chronic diarrhoea and RTA
- Oral potassium therapy safer than IV as it carries less risk of hyperkalemia

IV POTASSIUM

- Iv route carries high risk for hyperkalemia
- Reserved only for severe symptomatic hypokalemia or for the patients who can't take oral feeds.
- Always monitor IV therapy with cont EC monitoring and frequent K measurements
- Avoid IV till U/O is established
- **Don't Give**
- **> 10-20 mEqL/hr**
- **> 40 mEq/Litre**
- **>240 mEq/day**

- **Never give:**
  - **Direct Inj. KCL IV; can cause sudden cardiac arrest**
  - **Add KCL to Isolyte M**

- Rapid IV correction can cause dangerous hyperkalemia; hypo is better than hyper
- Avoid treating Meta. Acidosis; asso with Hypokalemia; with IV $NaHCO_3$ as it may aggravate hypokalemia
- In severe hypoK, add KCl in isotonic saline; not in D5% as diluent.

- DKA and non ketotic hyperosmolar hypergylcemia are the commenest indication for IV potassium therapy.
- 100 mEq of K+ mixed in 1 litre of isotonic saline at rate of 100 ml/hour (25 macro or 100 micro drops) will deliver 10 mEq KCl per hour.
- **IV potassium max rate of infusion: Central line 60 mEq/L and peripheral line 40 mEq/L.**
- > 40 mEq/L can cause thrombophlebitis
- Avg rise in S. K+ level ins 0.25 mEq/L when 20 mEq/l given in one hour.
- As soon as cardiac rhythm returns to normal or respiratory muscle strength is restored to normal; IV potassium drip is to be tapered and switch to oral potassium therapy.

Asso Magnesium deficiency

- **Always suspect if:**
- Malnutrition/ alcoholic
- Diarrhoea
- Diuretics
- Not responding to replacement of hypokalemia even with adequate doses
- Associated hypocalcemia
- DM
- Aminoglycoside use

Oral K+ Salts:

- Oral salts are safer as having minimal risk of hyperkalemia
- Mild to mod hypo K+ (3 to 3.5 mEq/L): avg dose is 20 mEq 3 to 4 times a day along with treatment of underlying disorder
- Potassium chloride solution contains 20 mEq per 15 ml solution.
- KCl Tab contains 8 mEq per tab.
- May cause frequent GI Irritation; so advised to take solution with proper dilution with water and after food
- Oesophageal or small bowel erosion or stricture are uncommon side effects.

Thank you