

APPROACH TO HYPOKALEMIA

Fb/Nurse Info

HYPOKALEMIA

```
graph TD; A[HYPOKALEMIA] --> B[urine potassium < 25 mEq/day]; A --> C[urine potassium > 30 mEq/day]
```

urine potassium < 25 mEq/day

urine potassium > 30 mEq/day

Urinary conservation +

urine potassium < 25 mEq/day

Acid base status

Normal acid base

- Profound sweating
- Prolonged decreased Intake
- Vomiting, NG suction ,
remote diuretic use

Metabolic acidosis

Lower GI loss

Urinary conservation -

urine potassium >30 mEq/day

Acid base status

Metabolic alkalosis

Normal acid base

Metabolic acidosis

- **Post ATN/ post obstructive diuresis**
- **Osmotic diuresis**
- **Gentle diuretic use**
- **Decreased magnesium ions**
- **High dose penicillin**
- **Polydipsia/ diabetes insipidus**

- **Type 1 RTA**
- **Type 2 RTA**
- **DKA**
- **Amphotericin B**
- **acetazolamide**

HYPOKALEMIA

urine potassium >30 mEq/day

Metabolic alkalosis

Urine chloride <20 mEq/liter

- Nasogastric suction
- vomiting

HYPERTENSIVE

Normal / low aldosterone

- CUSHINGS
- CAH
- LIDDLE'S SYNDROME
- APPARENT Mineralocorticoid excess
- HYPERALDOSTERONISM (1' and 2')

Urine chloride >20 mEq/liter

Blood pressure

NORMOTENSIVE/ HYPOTENSIVE

- *BARTTER'S SYNDROME*
- *GITELMAN'S SYNDROME*
- *AGGRESSIVE DIURETIC USE*

- **HYPOKALEMIA:**

REDUCED INTAKE

RENAL WASTING- most common cause

TRANS CELLULAR SHIFT.

Hypokalemia

plasma K⁺ concentration <3.5 mmol/L,

• CAUSES:

I. Decreased intake (Seldom a sole cause for K⁺ depletion)

- A. Starvation
- B. Clay ingestion (Geophagia – binds dietary K⁺ and Fe)

II. Redistribution into cells

- A. Acid-base

1. Metabolic alkalosis (1. K^+ redistribution into cell 2. Excess renal loss)

- B. Hormonal

1. Insulin (Stimulation of Na H antiporter and secondary activation of Na K ATP ase pump)

2. beta-Adrenergic agonists (1. Directly induces cellular uptake of K^+ 2. Stimulates insulin secretion)

3. alpha-Adrenergic antagonists

C. Anabolic state (D/T K+ shift into newly formed cells)

- 1. Vitamin B₁₂ or folic acid
(RBC production)
- 2. Granulocyte-macrophage colony stimulating factor
(white blood cell production)
- 3. Total parenteral nutrition

- D. Other

- 1. Pseudo hypokalemia
- 2. Hypothermia
- 3. Hypokalemic periodic paralysis (Calcium Channelopathy)
- 4. Barium toxicity

pseudohypokalemia

- Prevented storing the blood sample on ice or rapidly separating the plasma from the cells.
- e.g., acute myeloid leukemia
-low measured plasma K^+ concentration d/t white blood cell uptake of K^+ at room temperature

- III. Increased loss

- A. Non renal

- 1. Gastrointestinal loss (1. diarrhea – per se loss of K^+)...profuse diarrhea, villous adenomas, VIP oma, laxative abuse
2. Vomitting - mainly d/t $\wedge$ renal K^+ excretion caused by volume depletion and metabolic alkalosis)
- 2. Integumentary loss (sweat) (ECF contraction...Aldosterone secretion)

- **B. Renal**

- **1. Increased distal flow:**

- diuretics
- osmotic diuresis
- salt-wasting nephropathies

- **2. Increased secretion of potassium**
- **I. Mineralocorticoid excess:**
- **Primary hyperaldosteronism** (ca, Conn's , hyperplasia)
...**low plasma renin activity**
- **Secondary hyperaldosteronism** (1.malignant hypertension, 2.renin-secreting tumors –RCC, Ovarian Ca, Wilm's tumor, 3.renal artery stenosis, 4.hypovolemia)...**Hyperreninemia**
- **Bartter's s..** (Na K 2 Cl pump mutation...Volume depletion...hyperaldosteronism...K⁺ secreted

- **Apparent Mineralocorticoid excess** (licorice, chewing tobacco, carbenoxolone) ...cortisol occupies the aldosterone receptor for action...cortisol to cortisone does not occur d/t 11 beta HSDH deficiency/inhibition...– **low renin and aldosterone**
- **Congenital adrenal hyperplasia** (D/T non aldosterone Mineralocorticoids – corticosterone, deoxycorticosterone)
- **Cushing's s..** (cortisol formed overwhelms the activity of 11 beta HSDH)

- **II. Distal delivery of non-reabsorbed anions:**
- vomiting ... HCO_3^- in urine
- nasogastric suction
- proximal (type 2) renal tubular acidosis
- diabetic ketoacidosis ...Beta OH butyrate
- glue-sniffing (toluene abuse) ...Hippurate
- penicillin derivatives ...(1. Secrete K^+ ; 2. Osmotic diuresis)

- **III. Other:**

- **Amphotericin B**...(^ distal nephron K^+ permeability)
- **Liddle's syndrome**... (ENaC up regulation in CCD... negative Electrical gradient in lumen... H^+ & K^+ secretion)
- **Hypomagnesaemia**...(Resistant to treatment)

Genetic disorders

- ⌘ Hypokalemic periodic paralysis
- ⌘ Bartter's syndrome
- ⌘ Gitelman's syndrome
- ⌘ Liddle's syndrome
- ⌘ Apparent mineralocorticoid excess
- ⌘ Glucocorticoid-remediable hyperaldosteronism

HYPOKALEMIA

urine potassium >30 mEq/day

Metabolic alkalosis

Urine chloride <20 mEq/liter

- Nasogastric suction
- vomiting

HYPERTENSIVE

Normal / low aldosterone

- CUSHINGS
- CAH
- LIDDLE'S SYNDROME
- APPARENT Mineralocorticoid excess
- HYPERALDOSTERONISM (1' and 2')

Urine chloride >20 mEq/liter

Blood pressure

NORMOTENSIVE/ HYPOTENSIVE

- *BARTTER'S SYNDROME*
- *GITELMAN'S SYNDROME*
- *AGGRESSIVE DIURETIC USE*