Hypokalemia in children

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بسم الله الرحمن الرحيم
• Hypokalemia is defined as a serum or plasma potassium that is less than the normal value.

• The lower pediatric limit of normal serum potassium is between 3 and 3.5 mEq/L.

• However, symptoms are unlikely to occur in most healthy children until serum potassium is below 3 mEq/L.
Hypokalemia is relatively common among hospitalized pediatric patients, especially those who are critically ill.

- **severe** hypokalemia, defined as potassium level less than 2.5 mEq/L
- **moderate** hypokalemia, defined as potassium level 2.5 to less than 3 mEq/L
- **mild** hypokalemia, defined as potassium level from 3 to less than 3.5 mEq/L.
• In developing countries, severe hypokalemia (potassium level <2.5 mEq/L) is often observed in children with diarrhea and severe acute malnutrition, and is associated with an increased risk of mortality.
• 4 basic mechanisms of hypokalemia

• 1- Spurious hypokalemia occurs in patients with leukemia and very elevated WBC counts

• 2- If plasma for analysis is left at room temperature, permitting the WBCs to take up K+ from the plasma.

• 3- With a transcellular shift, there is no change in total body K+, although there may be concomitant potassium depletion resulting from other factors.

• 4- Decreased intake, extrarenal losses, and renal losses are all associated with total body K+ depletion
• Causes of Hypokalemia:
• Spurious
• Laboratory Value High white blood cell count
• Transcellular Shifts
• Alkalemia
• Insulin
• α-Adrenergic agonists
• Drugs/toxins (theophylline, barium, toluene, cesium chloride, hydroxychloroquine) Hypokalemic periodic paralysis
• Thyrotoxic period paralysis
• Refeeding syndrome
• Decreased Intake
• Anorexia nervosa
• Extrarenal Losses
• Diarrhea
• Laxative abuse
• Sweating
• Sodium polystyrene sulfonate (Kayexalate) or clay ingestion
Renal Losses

• **With Metabolic Acidosis:**
  - Distal RTA
  - Proximal RTA
  - Ureterosigmoidostomy
  - DKA

• **Without Specific Acid–Base Disturbance:**
  - Tubular toxins: amphotericin, cisplatin, aminoglycosides
  - Interstitial nephritis
  - Diuretic phase of ATN
  - Postobstructive diuresis
  - Hypomagnesemia
  - High urine anions (e.g., penicillin or penicillin derivatives)
With Metabolic Alkalosis

- Emesis or NGT
- CF
- Posthypercapnia
- Previous loop or thiazide diuretic use
- Adrenal adenoma or hyperplasia Glucocorticoid-remediable aldosteronism Renovascular disease
- Liddle syndrome
Clinical Manifestations

- **Heart and skeletal muscle** are especially vulnerable to hypokalemia.
- **ECG changes:**
  - flattened T wave,
  - depressed ST segment
  - appearance of a **U wave**, which is located between the T wave (if still visible) and the P wave.
- **Ventricular fibrillation and torsades de pointes** may occur, although usually only in context of underlying heart disease.
- Hypokalemia makes heart especially susceptible to digitalis-induced arrhythmias (**SVT, VT, heart block**).
ECG changes in Hypokalemia

- Slightly prolonged PR interval
- ST depressed and prolonged
- Slightly peaked P wave
- Prominent U wave
- Depressed T wave (may be inverted)

ECG changes: ST Depression, flattened T waves, prolonged P-R intervals, prominent U waves
• muscle weakness and cramps.
• Paralysis is a possible complication, generally only at [K<2.5 meq/l ]
• It usually starts in the legs and moves to the arms.
• Respiratory paralysis may require mechanical ventilation.
• Some patients have rhabdomyolysis;
• Hypokalemia slows GI motility. This effect manifests as constipation; with K+ <2.5 meq/l an ileus may occur.
• impairs bladder function, potentially leading to urinary retention.
• polyuria and polydipsia by impairing urinary concentrating ability, which produces nephrogenic DI.
• stimulates renal ammonia production, an effect that is clinically significant if hepatic failure is present, because the liver cannot metabolize the ammonia. Consequently, hypokalemia may worsen hepatic encephalopathy.
• Chronic hypokalemia may cause kidney damage, including interstitial nephritis and renal cysts.
Most causes of hypokalemia are readily apparent from the history.

Child's diet, GI losses, and medications.

The presence of hypertension suggests excess mineralocorticoid effects or levels.

The combination of hypokalemia and metabolic acidosis is characteristic of diarrhea and distal and proximal RTA.

A concurrent metabolic alkalosis is characteristic of emesis or nasogastric losses, aldosterone excess, use of diuretics, and Bartter and Gitelman syndrome.
FIG. 62.4 Diagnostic algorithm to evaluate persistent hypokalemia. **AME, licorice, Carbexcylolone Chronic grapefruit juice intake Liddle syndrome (PA-I) MR activating mutation (PA-II) Exogenous mineralocorticoid Excess ACTH / Glucocorticoids**
FIG. 68.4 Diagnostic algorithm to evaluate persistent hypokalemia. *Spurious
• If a clear etiology is not apparent, the measurement of urinary K+ distinguishes between renal and extrarenal losses.
• The kidneys should conserve K+ in the presence of extrarenal losses.
• Urinary K+ losses can be assessed with a 24 hr urine collection, spot K+ :creatinine ratio, fractional excretion of K+ , or calculation of the transtubular K + gradient (TTKG), which is the most widely used approach in children.
• The urine osmolality must be greater than the serum osmolality for the result of this calculation to be valid.
• A TTKG >4 in the presence of hypokalemia suggests excessive urinary losses of K+. 
TTKC = \frac{[K]_{\text{urine}}}{[K]_{\text{plasma}}} \times \frac{\text{plasma osmolality}}{\text{urine osmolality}}
Treatment
Factors that influence the treatment of hypokalemia include:

- the K+ level,
- clinical symptoms,
- renal function,
- the presence of transcellular shifts of K+,
- ongoing losses,
- patient's ability to tolerate oral K+
• Severe, symptomatic hypokalemia requires aggressive treatment.
• Supplementation is more cautious if renal function is decreased because of kidney's limited ability to excrete excessive K+.
• The plasma potassium level does not always provide an accurate estimation of the total body K+ deficit because there may be shifts of K+ from the ICS to the plasma.
Clinically, such shifts occur most often with metabolic acidosis and the insulin deficiency of DKA;

the plasma [K+] measurement underestimates the degree of total body K+ depletion.

When these problems are corrected, K+ moves into the ICS, so more K+ supplementation is required to correct the hypokalemia.
• the presence of a transcellular shift of K+ into the cells indicates that the total body K+ depletion is less severe.

• In an isolated transcellular shift, as in hypokalemic periodic paralysis, K+ supplementation should be used cautiously.
• Because of the **risk of hyperkalemia**, intravenous K+ should be used very cautiously.

• **Oral K+** is safer, but not as rapid in urgent situations. Liquid preparations are bitter tasting;

• Oral dosing is variable depending on the clinical situation.
• A typical starting dose is 1-2 mEq/kg/day,
• with a maximum of 60 mEq/day in divided doses.
• The dose of IV potassium is 0.5-1.0 mEq/kg, usually given over 1 hr.
• The adult maximum dose is 40 mEq.
• *Potassium chloride* is the usual choice for supplementation

• Patients with *acidosis* and hypokalemia can receive *potassium acetate or potassium citrate*.

• If *hypophosphatemia* is present, can be replaced with *potassium phosphate*
• It is sometimes possible to decrease ongoing K+ losses.
• For patients with excessive urinary losses, potassium-sparing diuretics are effective,
• but they need to be used cautiously in patients with renal insufficiency
• If hypokalemia, metabolic alkalosis, and volume depletion are present (with gastric losses), restoration of **intravascular volume** with adequate NaCl will decrease urinary K+ losses.

• **Correction of concurrent hypomagnesemia** is important because it may cause hypokalemia.

• **Disease-specific therapy** is effective in many of the genetic tubular disorders.
Thanks for your attention