Hypokalemia

Dr.V.Poovazhagi MD,DCH,PhD

HOD and Professor

Department of Pediatric Intensive Care

Institute of Child Health and Hospital for Children, Chennai

Hypokalemia

- Prevalence and definition
- Physiology
- Etiology
- Clinical features
- Diagnosis
- Evaluation as 3 steps
- Treatment
- Scenario

Why is it important ?

- 20% of hospitalised children have hypokalemia 4-5% clinically significant*
- 80% of those on diuretics develop hypokalemia
- Increased mortality and morbidity if not recognised and treated in time

* Potassium Homeostasis, Oxidative Stress, and Human Disease.*Udensi UK, Tchounwou PB Int J Clin Exp Physiol. 2017; 4(3):111-122.*

Hypokalemia

- Increase mortality by 10 fold
- Life threatening arrythmias
- Respiratory failure and arrest
- Precipitates encephalopathy
- Risk of toxicity with drugs
- Rhabdomyolysis

Endocr connect.2018 Apr; 7(4): R135–R146.Published online 2018 Mar 14.

Why is potassium important ?

- Intracellular cation
- Important for Na K Pump
- Na pumped out and K pumped in –K gradient important for potential difference across cell membrane
- Excitability of neuromuscular system
- K imbalances lead to conduction issues and arrythmias and death

Potassium balance

- Maintained by intake
- Renal and GI excretion

Hypokalemia

- Defined as serum potassium <3.5 mEq/L
- Mild if between 3-3.5mEq/L
- Moderate if between 2.5-3mEq/L
- Severe if <2.5mEq/L
- Critical if <2mEq/L

Etiology

- Due to decreased intake- malnutrition, anorexia,
- Due to transcellular shift- alkalosis, insulin , beta 2 agonists , hypokalemic periodic paralysis
- Drugs
- Renal Loss

GI loss

- Vomiting / diarrhea
- Chronic laxative abuse
- Bowel diversion
- Clay ingestion
- Villous adenoma of the colon

Drugs

- Thiazides
  - Loop diuretics
  - Osmotic diuretics
  - Laxatives
  - Amphotericin B
  - Antipseudomonal penicillins (carbenicillin)
  - Penicillin in high doses

Renal loss

- Renal tubular acidosis
- Fanconi syndrome
- Adrenal steroid excess (Cushing's syndrome)
- Primary hyperaldosteronism
- Renin-secreting tumors
- Glucocorticoid-remediable congenital adrenal hyperplasia
- Hypomagnesemia

Clinical features

- Muscle weakness and paralysis- hypotonia,absent reflexes, abdominal distension, hypoactive bowel sounds and resp. paralysis
- Cardiac arrhythmias and ECG changes –
- Impaired tubular absorption and renal abnormalities- polyuria and polydipsia.
- ECG

Investigations

Serum Electrolytes, Urinary Potassium

ECG Initially : flattening of t wave
depression of ST Segment
development of prominent u waves

Severe hypokalemia : increased amplitude of p

Plasma K^+, meq/L 4.0 3.0 2.0 1.0

ECG.....

- ST- depression, PR- prolongation
- Sinus bradycardia
- Premature atrial and ventricular beats
- Paroxysmal atrial or junctional tachycardia
- Atrioventricular block
- Ventricular tachycardia or fibrillation

History

- GI loss- vomiting, diarrhea,aspiration drain
- Decreased intake of potassium
- Drugs like B2agonist, diuretics,insulin.
- F/H/O hypokalemic periodic paralysis
- Native medicines like Oduvanthazhai

Clinical clues

- History
  - Vomiting/Awd/drugs
- Examination
  - Failure to thrive
  - Lethargy
  - Dehydration
  - Drains/ nebulisation/

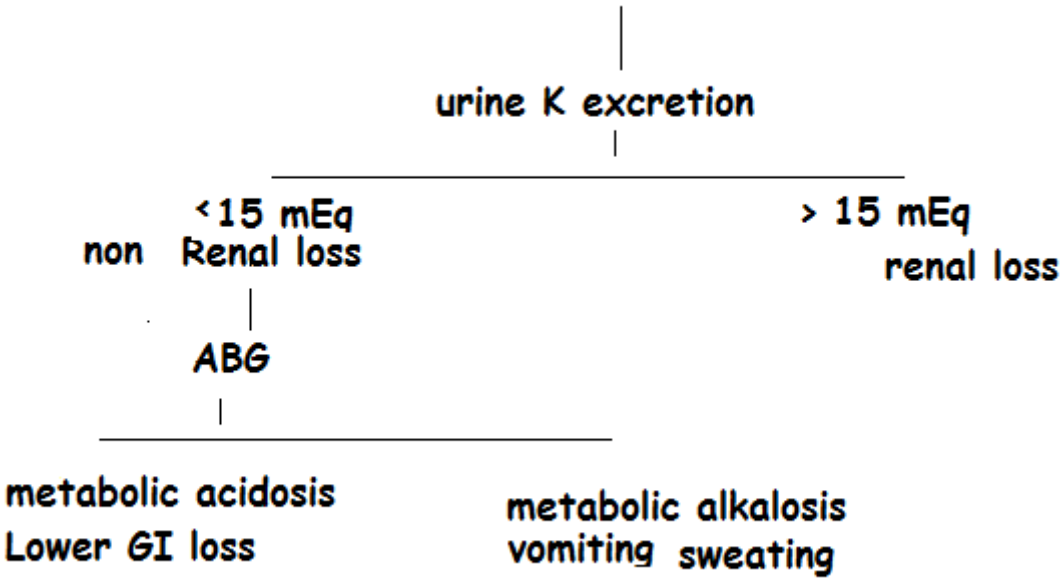
Examination

- Brady cardia or abnormal pulses
- Shallow respiration
- Abdominal distension/illeus
- Reduced muscle tone
- Hypo or areflexia
- Polyuria

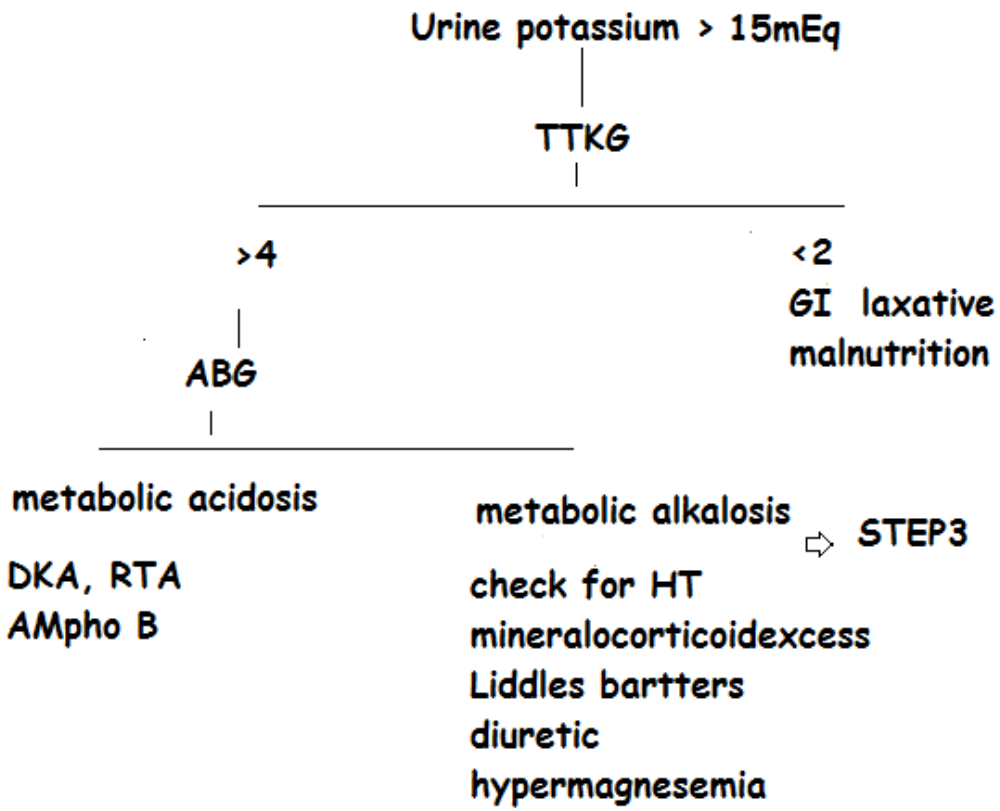
What if no clue?

- Urine potassium
- TTKG
- ABG
- Aldosterone,renin
- Cortisol
- Urine chlorides
- Ca Cr ratio

Hypokalemia Step 1



Step 2 urine K >15 mEq



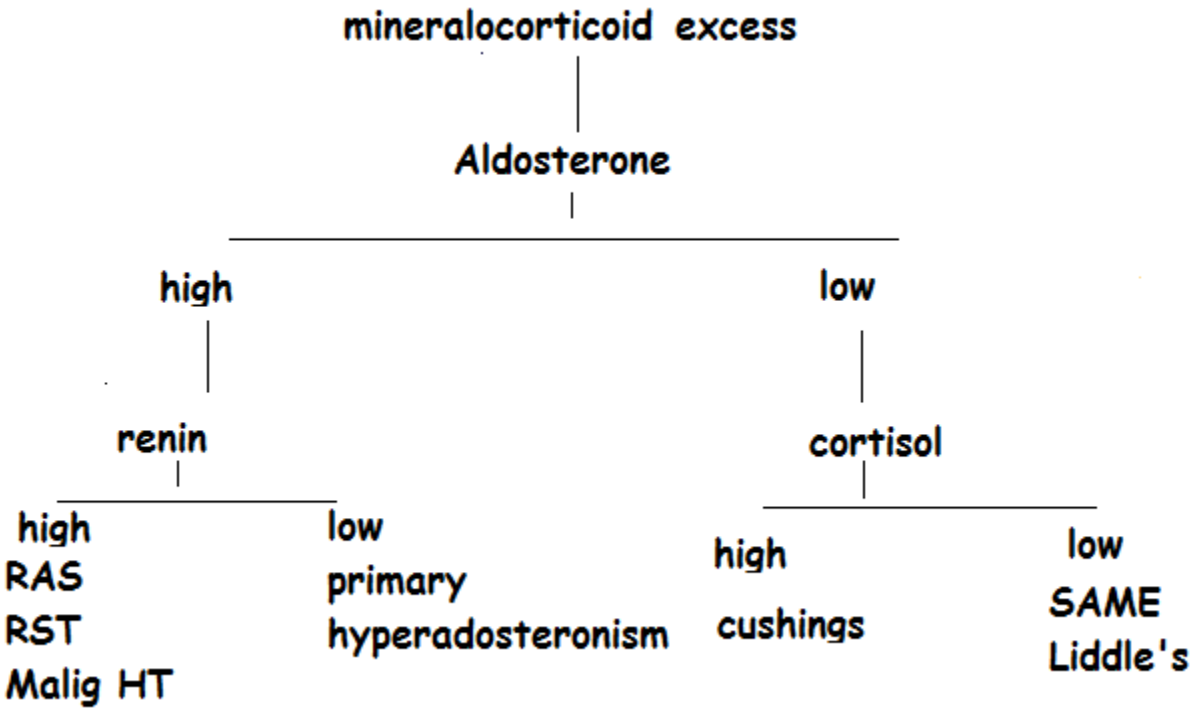
TTKG

- Urine potassium/serum potassium X serum osmolality/urine osmolality
- Pre requisite

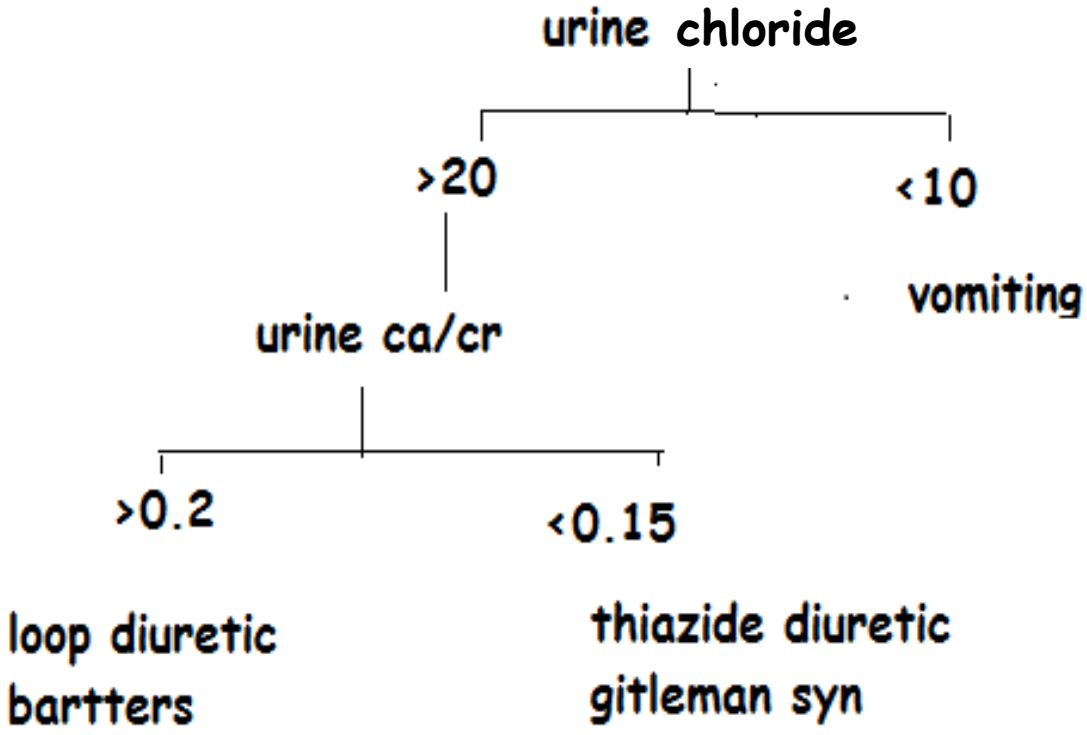
Urine sodium >20mEq/

Urine osmolality > serum osmolality

Step 3 Hypertension



Step 3 No hypertension



Other biochemical tests

- Serum renin aldosterone
- Drug screen
- Thyroid function test
- Pitutary /adrenal imaging
- Renal artery doppler

Potassium rich foods

- Banana, kiwi, mango, orange, sweet potato, tomato, papaya, coconut water, fruit juices, pickles, dry fruits, coffee, chocolates, spinach

Treatment

- Potassium replacement if:

serum potassium <3.0 mmol/L or

serum potassium <3.5 mmol/L with symptoms/signs/ECG changes

- If serum potassium is 3.0 mmol/L - 3.4 mmol/L in a well child, it is reasonable to either:

  monitor electrolytes,

  increase maintenance potassium dose, or

  replace potassium depending on the clinical situation

In children with stable hemodynamics and no ECG changes, aim for a gradual correction over 24-48 hours.

Dosing route

- Oral/enteral is the preferred route
  - Oral K is well absorbed from the GIT
  - Best taken with or soon after food

- Consider intravenous replacement if:
  - Child is unable to tolerate oral medication,
  - Serum potassium <2.5 mmol/L, or
  - ECG changes present

Treatment

- Syp pot chlor
  15ml = 20 mEq
  1-2 mEq/kg/dose (maxi 20mEq/dose)
  up to 5 mEq/kg/day

- K < 2.5mEq needs IV correction
  Inj KCL 1ml- 2 mEq

IV correction

- Peripheral line up to 40mEq/L
- 0.2-0.5 mEq/kg- infusion over 1-2 hours under ECG monitoring (max 20mEq/hr)
- Never bolus Inj KCL
- Ensure urine output
- Recheck after 1 hr of completion of correction
- Rapid Iv dosing may cause cardiac arrest

IV K correction

- Dilution in 5% dextrose
- Wt X 0.5 mEq /2 =ml of kcl

- Dilute in 50 ml if <10kg
- 100ml if 10-20kg
- 150 ml if >20Kg run over 1 hour

- IV KCL is life saving yet a dangerous drug
- Check the dose prior to infusion
- Proper labelling is important
- Always monitor ECG
- Use adequate dilution in 5% dextrose
- Always infuse over 1-2 hours
- Recheck K after 1hr of completion of therapy.

Magnesium and potassium

- More than 50% of clinically significant hypokalemia has concomitant magnesium deficiency
- In children receiving loop or thiazide diuretic
- Concomitant magnesium deficiency aggravates hypokalemia.
- Hypokalemia associated with magnesium deficiency is often refractory to treatment with K^+

Summary

- Confirm hypokalemia by clinical features and ECG
- If history is s/o cause- treat
- If no clue- urine K to know renal or no renal
- Proceed to do ABG if acidosis RTA
- If alkalotic check BP
- If hypertensive do renin aldosterone and cortisol
- If normotensive urine chlorides and calcium/chloride ratio
- Be cautious during rapid correction.

References

- Anthony JV Noah W, Potassium Disorders: Hypokalemia and Hyperkalemia Am Fam Physician . 2015 Sep 15;92(6):487-95.
- Udensi UK, Tchounwou PB Potassium Homeostasis, Oxidative Stress, and Human Disease. Int J Clin Exp Physiol. 2017; 4(3):111-122.
- Ashurst J, Sergent SR, Wagner BJ, Kim J. Evidence-based management of potassium disorders in the emergency department [digest]. Emerg Med Pract. 2016 Nov 22;18(11 Suppl Points & Pearls):S1-S2. PMID: 28745843.
- Pepin J, Shields C. Advances in diagnosis and management of hypokalemic and hyperkalemic emergencies. Emerg Med Pract. 2012 Feb;14(2):1-17; quiz 17-8. PMID: 22413702
- Elliott TL, Braun M. Electrolytes: Potassium Disorders. FP Essent. 2017 Aug;459:21-28. PMID: 28806047.

Thank You