L2:Electrolytic Imbalance 2

(Potassium & Calcium)

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MEDICINE 433

objectives

1. Understand the basic physiologic principles of potassium hemostasis.
2. Know the application of physiologic and clinical principles in approaching hyperkalemia.
3. Know the application of physiologic and clinical principles in approaching hypokalemia.
4. Understand the basic principles of Calcium hemostasis.
5. Know the application of physiologic and clinical principles in approaching hypercalcemia .
5. Know the application of physiologic and clinical principles in approaching hypocalcemia .

Color index: Step up to medicine , slide , Female's note , Male's note , Davidson

Potassium hemostasis

1 small banana (85 g)	8.6 mmol of K
French fries (150 g)	17.7 mmol of K

Where does K come form?	○ Depending on diet * ○ High-fat diets usually contain low amounts of potassium ○ Average daily intake approximately 50 to 100 mmol
How do we loose K ?	○ Renal clearance - primary mechanism - Very efficient until GFR < 30 ml/min ○ Intestinal excretion - Only handles 10 % of the daily K load - Efficiency can be enhanced in renal failure but it is variable form one person to another (it is used therapeutically to decrease K level)
Where does K live in the body ?	- Total body K is approximately 50 mmol/kg body weight - K is the most abundant intracellular cation (100- 150 mmol/l) ➔ 98 % of total body K - Extracellular K concentration (3.4 – 5.5 mmol/L) ➔ 2% of total body K + (practically measured)
What keeps the IC K high ?	○ Insulin, Beta agonists enhance the pump function (↑ cells up take of K+) ○ Beta Blockers inhibit the pump function (↓ cells up take of K+)
What keeps EC K low?	○ The Na/K ATPase pump ○ Renal clearance : requires normal GFR and normal aldosterone axis ○ Intestinal excretion

Potassium hemostasis

What happens when we eat K ?	○ Oral K intake is initially absorbed in the intestine and enters portal circulation → increased ECF K stimulates insulin release → insulin facilitates K entry into intracellular compartment by stimulating cell membrane Na/K ATPase pump. The transient rise in serum K stimulates renal and intestinal clearance of extra K
What we need to Keep serum K in normal range ?	○ Normally functioning Na/K ATPase pump (keeps K inside- pushes NA out) ○ intact renal response
Why is K important ?	○ Maintains electrical gradient across cell membranes i.e. resting membrane potential essential for generation of action potential (resting membrane potential is -70) ○ Essential for intracellular metabolism e.g protein synthesis
What happens of K level is abnormal?	○ Skeletal muscle dysfunction: weakness and paralysis ○ Cardiac cell irritability: arrhythmia
Can you eat too much K ?	○ If GFR is normal, renal clearance of K has a huge adaptive capacity. Also aldosteron production and receptors should be normal. ○ K intake is restricted only if: - GFR is reduced - existing aldosterone axis dysfunction - Na/K ATPase is not efficient (blocked by drugs, Insulin ↓)

Hyperkalemia

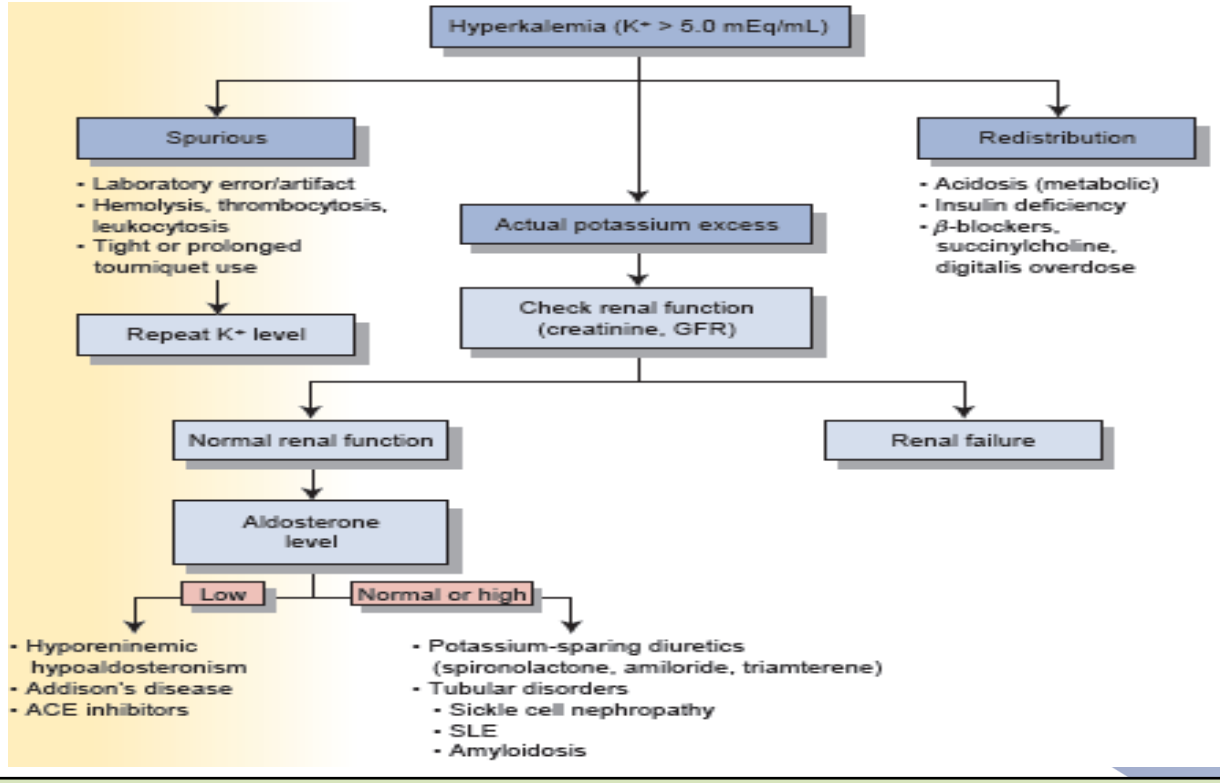
Causes	NA/K ATPase dysfunction - B blockers - Digoxin - ↓ Insulin - Acidosis (↓cell up take	Massive Cell breakdown - Rhabdomyolysis - Tumor lysis syndrome	Impaired Renal function -NSAIDs -ACE inhibitors	Aldosterone axis dysfunction - Adrenal deffiency - Aldosterone resistance - Addison disease
Signs & Symptoms	○ Arrhythmias → The most important effect of hyperkalemia is on the heart. Check an ECG immediately in a hyperkalemic patient. With increasing potassium, ECG changes progress through tall, peaked T waves, QRS widening, PR interval prolongation, loss of P waves, and finally a sine wave pattern.(because it will drop the cardiac threshold, so any action potential can stimulate it) ○ Muscle weakness and ○ Decreased deep tendon reflexes ○ Respiratory failure ○ Nausea/vomiting, intestinal colic, diarrhea			

Hypokalemia

Causes	↓ Oral intake - Malnutrition - eating disorders	Rapid transcellular shift - Insulin therapy - Periodic paralysis - Alkalosis	↑ Renal loss - Diuretics - high aldosterone (conn's disease, Cushing syndrome)	↑ Intestinal loss - Diarrhea - Laxative abuse
Signs & Symptoms	○ Arrhythmias—prolongs normal cardiac conduction ○ Muscular weakness, fatigue, paralysis, and muscle cramps ○ Decreased deep tendon reflexes ○ Paralytic ileus ○ Polyuria and polydipsia ○ Nausea/vomiting ○ Exacerbates digitalis toxicity ○ Flattening of T waves on EKG, U waves appear if severe.			

Hyperkalemia

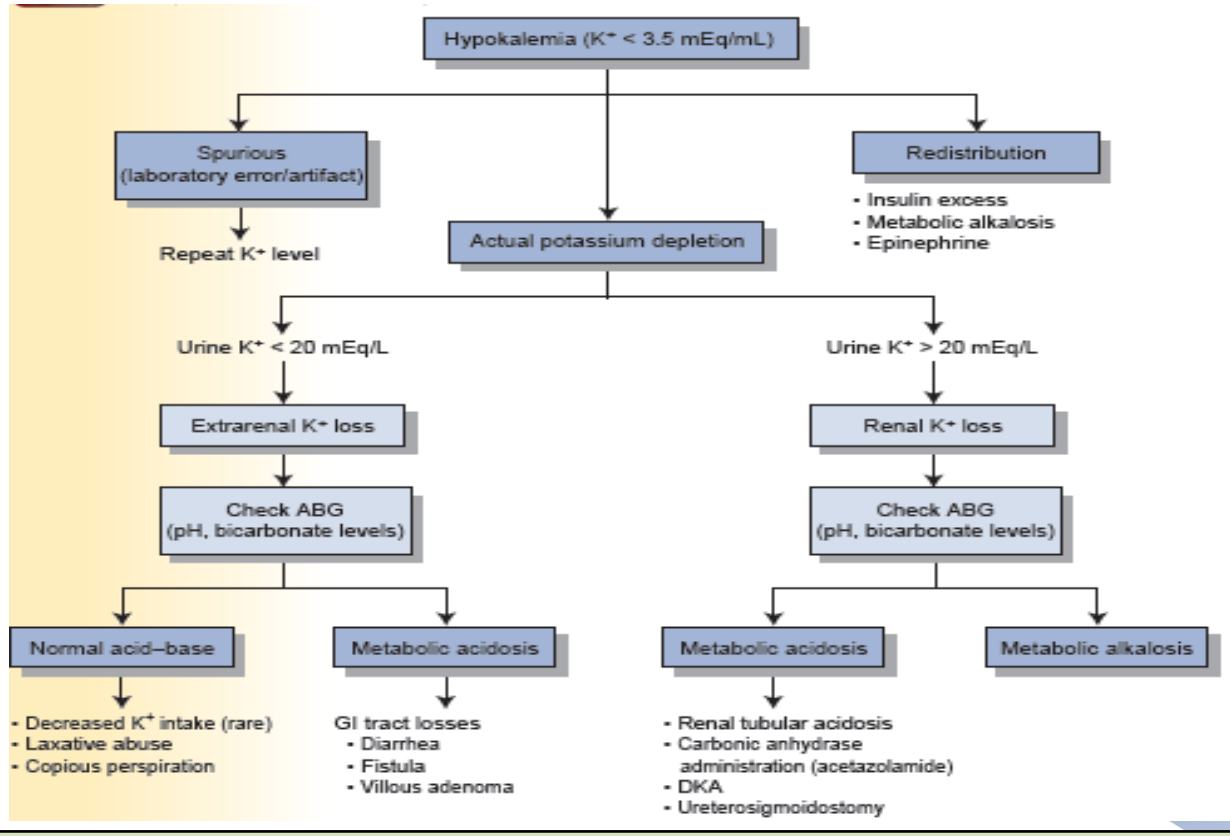
Asymptomatic K > 5.5
Symptomatic K >7.0

Diagnostic evaluation of hyperkalemia	
Management **How to lower K level ?**	○ Reduce Cardiac muscle irritability with Ca gluconate (only if EKG changes) ○ Push K into cells: Insulin , Beta agonists (higer doses) ○ Remove the K load ○ Through the kidney: diuretics (loop diuretics) , dialysis ○ Through the gut : Laxatives, K chelation (Ca resonium) *Note: calcium ion protect cell membrane but don't alter K level*

Hypokalemia

Asymptomatic K < 3.4
Symptomatic K < 2.5

Diagnostic evaluation of hypokalemia



Management

How to raise K level?

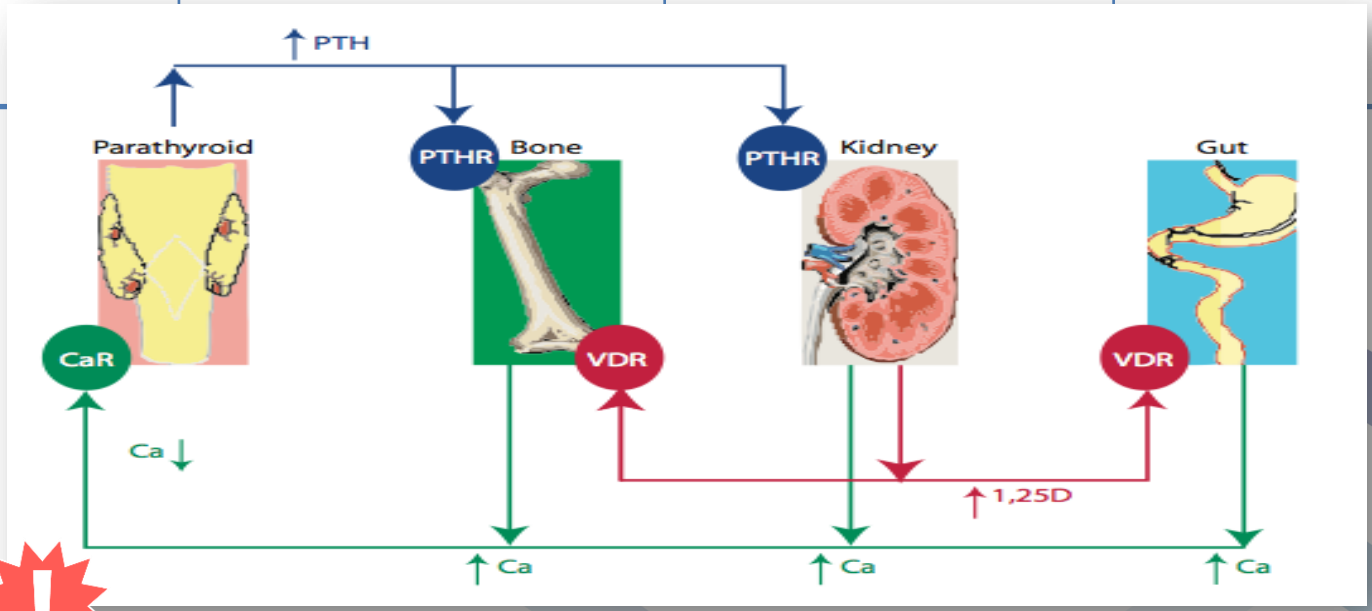
- Identify and treat the underlying cause.
- Replace lost K with K (PO or IV if rapid correction is urgently needed like in cardiac arrthymias)
- Mg level should be corrected if it's not
- Oral KCl is the preferred (safest) method of replacement
- IV KCl can be given if hypokalemia is severe (<2.5)

Calcium hemostasis

Where does Ca come from?	○ Diet : 1000 – 1500 mg /day in average ○ Total body Ca = 1000 g	
Where does Ca live ?	○ The vast majority of total body calcium (99%) is present in the skeleton ○ Non-bone calcium represents 1% of total body calcium ✓free ions (51%) (phusiologically active) ✓protein-bound complexes (40%) } Non Ionized ✓ionic complexes (9%) [calcium phosphate, calcium carbonate, and calcium oxalate]	
Why Ca is important ?	Bone Ca ✓skeletal strength ✓dynamic store	Non-Bone Ca ✓extra- and intracellular signaling ✓nerve impulse transmission ✓ muscle contraction
What keeps Ca in balance ?	Total intake Rate of intestinal absorption Intestinal excretion Renal reabsorption Renal excretion Bone turnover	All these parameters are controlled by: 1- PTH 2- Active Vitamin D 3-Serum Ionized Ca level

Calcium hemostasis

What are the hormones that affect Ca level?	○ PTH is a hyper-calcemic hormone - ↑ Release of Ca form bones (bone resorption) - ↑ Renal absorption of Ca	- Active Vitamin D is also hyper-calcemic - ↑ Intestinal absorption of Ca - ↑ Bone resorption	- Calcitonin - ↓ Plasma Ca and PO4



Hormonal mechanisms maintain narrow physiologic range of 10%.

Hypercalcemia

Hypertension is a common feature of hyperparathyroidism*

Causes	↑ Intestinal absorprtion - Increased intake - Increased Vit D	↑ Renal reabsorprtion - Hyperparathyroidism - Thiazide diuretics	↑ Bone resorption - Osteoclastic bone metastasis - Immobilization	↑ PTH - Primary hyperparathyroidism * - Multiple Endocrine Neoplasia - Lithium - Lung cancer ↑ Vit D - Intoxication
Signs & Symptoms	**Clinical Manifestations of Hypercalcemia** **Renal "stones"** Nephrolithiasis Nephrogenic diabetes insipidus Dehydration Nephrocalcinosis **Skeleton "bones"** Bone pain Arthritis Osteoporosis Osteitis fibrosa cystica in hyperparathyroidism (subperiosteal resorption, bone cysts) **Gastrointestinal "abdominal moans"** Nausea, vomiting Anorexia, weight loss Constipation Abdominal pain Pancreatitis Peptic ulcer disease			**Neuromuscular "psychic groans"** Impaired concentration and memory Confusion, stupor, coma Lethargy and fatigue Muscle weakness Corneal calcification (band keratopathy) **Cardiovascular** Hypertension Shortened QT interval on electrocardiogram Cardiac arrhythmias Vascular calcification **Other** Itching Keratitis, conjunctivitis

In hypercalcemia, ECG showes shortening of QT interval while with hypocalcemia ECG shows prolongation of QT interval*

Hypercalcemia con.

BUN = Blood urea nitrogen.
Cr = creatinine*

Investigations	1. Obtain the following: BUN, Cr*, magnesium, albumin, and ionized calcium. Amylase, lipase, and liver function tests may also be warranted). 2. Radioimmunoassay of PTH: elevated in primary hyperparathyroidism, low in occult malignancy 3. Radioimmunoassay of PTH-related protein: elevated in malignancy 4. Bone scan or bone survey to identify lytic lesions 5. Urinary cAMP: markedly elevated in primary hyperparathyroidism
Management	I. Increase urinary excretion. a. IV fluids (normal saline 0.9%)—first step in management b. Diuretics (furosemide)—further inhibit calcium reabsorption 2. Inhibit bone resorption in patients with osteoclastic disease (e.g., malignancy). a. Bisphosphonates (pamidronate) b. Calcitonin 3. Give glucocorticoids if vitamin D-related mechanisms (intoxication, granulomatous disorders) and multiple myeloma are the cause of the hypercalcemia. However, glucocorticoids are ineffective in most other forms of hypercalcemia. 4. Use hemodialysis for renal failure patients. 5. Phosphate is effective but incurs the risk of metastatic calcification.

Hypocalcemia

The most common cause is low serum albumin with normal ionized Ca concertation*

Causes*	↓ Intestinal absorprtion - Decreased intake -malabsorption -Small bowel resecton - Vit D defficiency	↓ Renal reabsorprtion - hypoparathyroidism -Loop diuretics -Tubular defects	Bone remodling -Hungry bone syndrome	↓ PTH - hypoparathyroidism ↓ Vit D - Renal failure
Hypoparathyroidism (most common cause) — usually due to surgery on the thyroid gland (with damage to nearby parathyroids).				
Signs & Symptoms	Neuropsychiatric - Seizure - Dementia - Extrapyramidal - Papillidema - Cataract	Neuromascular - Parasthesia - Spasm - Chvostek's sign* - Trousseau's sign ** - Rickets+osteomalacia	Cardiovascular - Prolonged QT interval - Heart failure - Hypotension	Autonomic - Biliary colic - Bronchospasm - Diaphoresis

*tapping a facial nerve leads to a contraction of facial muscle
**inflate BP cuff to a pressure higher that the patient's systolic BP for 3 mins (occludes blood flow in forearm) this elicits carpal spasms

Hypocalcemia

BUN = Blood urea nitrogen.
Cr = creatinine *

Investigations	1. Same laboratory tests as in hypercalcemia ,Obtain the following: BUN, Cr*, magnesium, albumin, and ionized calcium. Amylase, lipase, and liver function tests may also be warranted. 2. Serum PO4: high in renal insufficiency and in hypoparathyroidism, low in primary vitamin D deficiency 3. PTH • a. Low in hypoparathyroidism • b. Elevated in vitamin D deficiency • c. Very high in pseudohypoparathyroidism
Management	1. If symptomatic, provide emergency treatment with IV calcium gluconate. 2. For long-term management, use oral calcium supplements (calcium carbonate) and vitamin D. 3. For PTH deficiency a. Replacement therapy with vitamin D (or calcitriol) plus a high oral calcium intake b. Thiazide diuretics—lower urinary calcium and prevent urolithiasis 4. It is also important to correct hypomagnesemia by (magnesium chloride I.V) . It is very difficult to correct the calcium level if the magnesium is not replaced first.

MCQs

1. A 60 year-old male known case of peripheral arterial disease. He came to the hospital for following up. His blood pressure is high although he is taking his antihypertensive medications regularly. The investigations reveal renal artery stenosis. The physician ordered the electrolytes test. What do you expect he has

A-Hyperkalemia

B-Hypercalcemia

C-Hypokalemia

D-Hyponatremia

2. A 30 year-old female known case of pneumonia. The investigations showed low levels of potassium. The physician ordered to give IV potassium to correct hypokalemia. By mistake the nurse gave her double the dose which ordered by the doctor. What should the doctor do .Her ECG is normal?

A-give insulin

B-give Ca gluconate

C-Dialysis

D-give insulin + glucose

MCQs

3. A 56 year-old man with known case of hypertension since 10 years on diuretics. He came to the hospital for following up. His blood pressure is 130/80. Electrolytes test shows mild hyperkalemia. Which diuretic most likely he is using?

A-Furosemide
B-spironolactone
C-indapamide
D-metolazone

4. A 39 year-old male came to the clinic with kidney stone. He has history of weight loss and fatigue. The investigations showed hypercalcemia. Parathyroid hormone was low but PTH-related peptide was found (PTH-like molecule). Which neoplasm of the followings may be the cause?

A-Prostate adenocarcinoma
B-Transitional cell carcinoma of urinary bladder
C-Pheochromocytoma
D-Renal cell carcinoma

MCQs

5. A 46 year-old man comes to the emergency department with excruciating pain in his left flank radiating to the groin. He has some blood in his urine. What is the most appropriate next step in the management of this patient?

A-to give him NSAID
B-X-Ray
C-Sonography
D-Urinalysis

6. A patient is admitted with vomiting and diarrhea from gastroenteritis. His volume status is corrected with intravenous fluids and the diarrhea resolves. His pH is 7.40 and his serum bicarbonate has normalized. Despite vigorous oral and intravenous replacement, his potassium level fails to rise. What should you do?

A-Check magnesium level
B-Parathyroid hormone level
C-Intracellular pH level
D-Dialysis

MCQs

7. A woman with End stage renal disease and glucose 6-phosphate dehydrogenase deficiency skips dialysis for a few weeks then is crushed in a motor vehicle accident. She is taking dapsone and has recently eaten fava beans. What is the most urgent step?

A-initiate dialysis
B-Do ECG
C-Bicarbonate administration
D-Insulin administration

8. A 55 year-old female has history of thyroid cancer and had radical thyroidectomy. Which one of the following signs you may most likely see

A-carpopedal spasm
B-Murphy's sign
C-Pemberton's sign
D-Ape hand

Answers: 1-C 2- D 3-B 4-D 5-A 6-A 7-B 8-A

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Medicine is a science of uncertainty and an art of probability

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