Electrolytes

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Electrolytes

Electrolytes are substances that have a natural positive or negative electrical charge when dissolved in water. An adult's body is about 60% water, which means nearly every fluid and cell in your body contains electrolytes.

Electrolytes are essential for basic life functioning, such as maintaining electrical neutrality in cells and generating and conducting action potentials in the nerves and muscles.

These electrolytes can be imbalanced, leading to high or low levels. High or low levels of electrolytes disrupt normal bodily functions and can lead to life-threatening complications.

Electrolytes found in your body:

- Sodium
- Potassium
- Chloride
- Calcium
- Magnesium
- Phosphate
- Bicarbonate

Sodium

The most abundant in our body

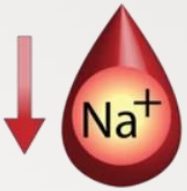
Normal sodium level : (135-145 mEq/L).

Sodium, an osmotically active cation, is one of the essential electrolytes in the extracellular fluid. It is responsible for maintaining the extracellular fluid volume and regulating the membrane potential of cells. Sodium is exchanged along with potassium across cell membranes as part of active transport.

Na ions[electricity] account for most the
osmolality in the extracellular fluid.
Wherever sodium goes water goes with it proportionally.

Sodium Disorders

Hyponatremia (<135 mEq/L), Symptoms begin when the Na+ level falls to <120 mmol/L.



Plasma sodium levels are directly affected by fluid balance.

Hyponatremia results from excess water intake that is not excreted, whereas

hypernatremia results from an excessive loss of free water.

Plasma Osmolality $= [Na^+] \times 2 + \frac{BUN}{2.8} + \frac{Glucose}{18}$

The osmolality of ECF is equal to the sum of the concentrations of all dissolved solutes.

Na+ and its anions account for nearly 90% of these solutes

Osmolality should be low in hyponatremia (true hyponatremia).

If not: **False Hyponatremia**

mainly high or normal osmolality due to increase of other molecules in blood like

1 - **hyperglycemia**

- In poorly controlled diabetes
- It will make dilution effect as H2O will be drawn into the intravascular space → H I G H serum osmolality

2 - **mannitol** usage in increased ICP cases

- Works in the same dilution effect → H I G H serum osmolality

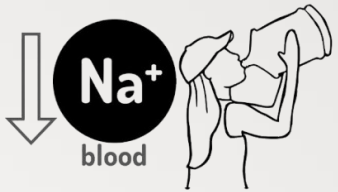
3 - **hyperlipidemia** and **hyperproteinemia**

- As in MM and recent IV IG therapy
- → N O R M A L serum osmolality
- Cause: lipids and proteins interfere with lab analysis of the serum (lab error)

4 - **TURP**

- sorbitol/mannitol + glycine are used in the procedure and do dilution effect

Hypotonic Hyponatremia is further classified according to volume status to hypovolemic/euvolemic/hypervolemic



Hypovolemic

- There's a depletion of both Na+ and water, but the sodium deficit exceeds the water deficit.
- Causes of Na+ loss:

***1) Extra-renal causes*:**

- ✓ **Low urine sodium (<10 mEq/L)**
- ✓ **Na+ loss can be from GIT or skin (**diarrhea, vomiting, diaphoresis, burn, nasogastric suction).
- ✓ Implies increased sodium retention by the kidneys to compensate for **extrarenal losses of sodium containing fluid.**

***2) Renal causes*:**

- ✓ **High urine sodium (>20 mEq/L)**
- ✓ Causes are either :
  - i. Diuretic therapy (thiazides)
  - ii. Adrenocortical failure

Euvolemic

- **There's no evidence of ECF expansion or contraction**
- **Caused by:**
  1. **Primary polydipsia**
  2. **SIADH**: Causes are numerous and include : CNS pathologies, malignancy (most commonly small cell lung cancer), surgery (postoperative hyponatremia)
  3. **Excessive electrolyte-free water infusion:** if a patient is given D5W (or other hypotonic solution) to replace fluids, or if water alone is consumed after intensive exertion (with profuse sweating)
  4. Hypothyroidism

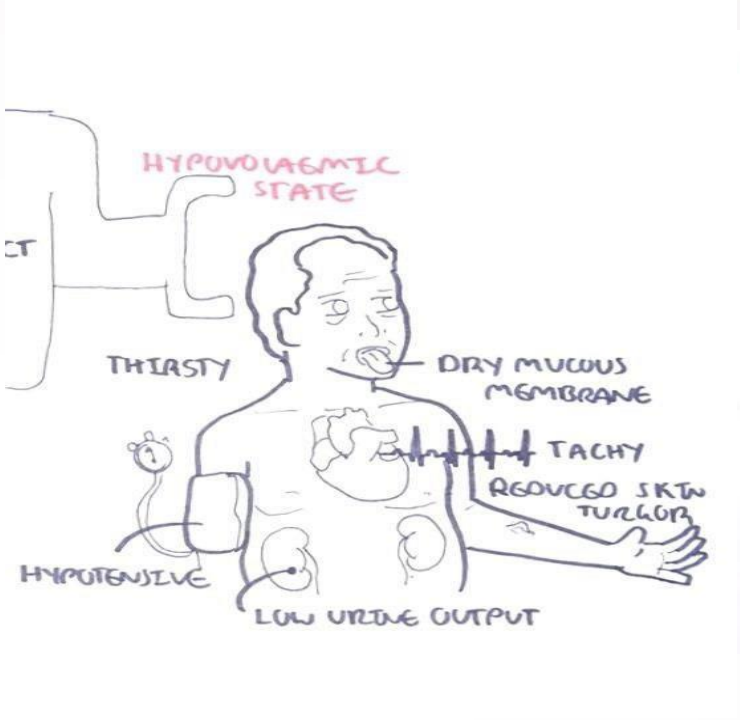
Hypervolemic

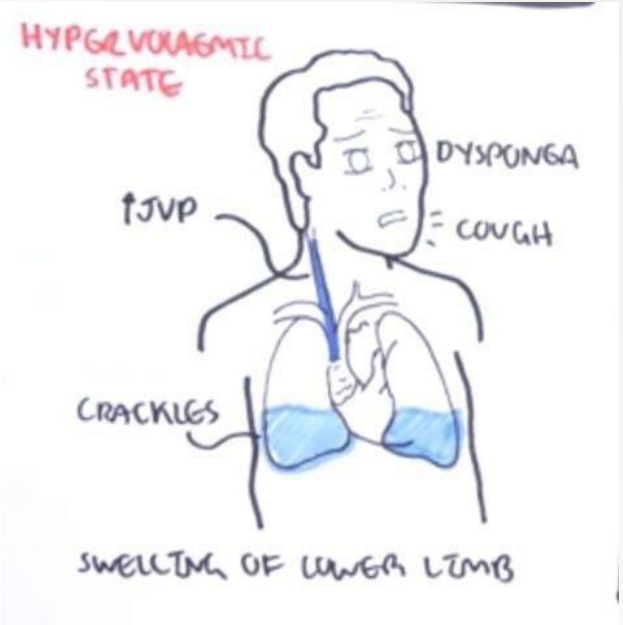
- This is due to water-retaining states that are with volume expansion
- The relative excess of water in relation to sodium results in hyponatremia

- Causes are:

1. CHF
2. Cirrhosis
3. Nephrotic syndrome
4. CKD; during free water intake

Signs and Symptoms





Hyponatremia isn't a disease but it's a manifestation of
Other disease

Clinical feature due to hyponatremia
seizures , confusion lethargy , coma weakness

Management

- Mild and asymptomatic hyponatremia (no treatment)
- Moderate and asymptomatic (strict fluid restriction 750ml to 1L then slow isotonic saline)
- Acute sever hyponatremia or rapid hyponatremia , management should be **quick to prevent cerebral edema and noncardiogenic pulmonary edema** (resuscitation then fluid restriction then slow hypertonic saline to increase serum sodium level)

Always don't forget to correct hyponatremia slowly (0.25–0.5 mEq/L per hour), to avoid central pontine myelinolysis.

The following correction rates have been suggested:
mild symptoms: 0.5 mEq/L/h or less;
moderate symptoms: 1 mEq/L/h or less;
severe symptoms: 1.5 mEq/L/h or less.

Hypernatremia

- Hypernatremia is defined as a sodium level greater than 145mEq/L (moderate, 146–159 mEq/L; severe, ≥160 Eq/L).

- Hypernatremia is caused by increase water loss or increase in sodium intake

- To diagnose patient with hypernatremia :

1. history (to ask patient if he had hyper aldosteronism or if he has been getting hypertonic saline and ask patient about diarrhea , vomiting and burn)
2. Urine osmolarity (if the urine osmolality is low so there is renal water loss and in high urine osmolality it will be extra renal water loss)
3. Sodium concentration in urine to differentiate between extra renal water loss (low sodium in urine) and sodium gain (high sodium in urine) .

- **Clinically:**
  seizures ,confusion, tremors .respiratory paralysis .
- **Management:**
  the most common cause is water loss so give them back water how much water you have to give ?

  free water deficit (L) = (serum sodium – normal sodium level)/140 * TBW

  Total body weight = weight(Kg)*constant (constant in male 0.6, in female 0.5)

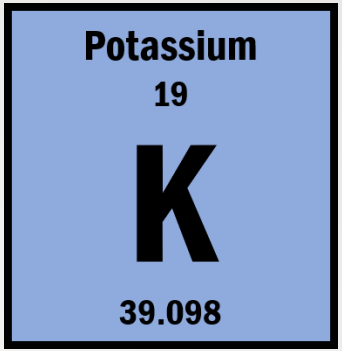
1. tell patient to drink water
2. If patient cant drink water give back water by NG or OG tube
3. Give patient IV FLUID like D5W or ½ NS , ¼ NS

- Slow lowering of serum sodium is very important less than 12 mEq/day TO AVOID SEZIURES . (brain edema) .

Potassium

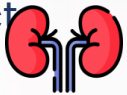
3.5-5 MEQ/L

Potassium



- Normal serum potassium: **3.5 – 5 mEq/L**.
- Most of our body's potassium **found Intracellular 98%.**
- It is one of the most vital electrolytes in the body, essential for maintaining proper cellular function, fluid balance, and electrical signaling especially in muscle and nerve cells.

- **Potassium Secretion**: most of the excretion of potassium occurs through **Kidneys** (80%) and the reminder occurs via GI tract.



- Aldosterone plays an important role in renal potassium secretion. **Extracellular potassium is the major determinant of aldosterone secretion from the adrenal gland.**

Hypokalemia

Mild: <3.5 mEq/L

Severe: <3 mEq/L

Hypokalemia

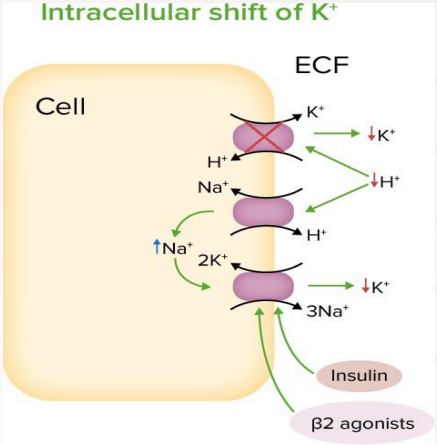
Causes

- **Renal Losses**:
  - Diuretics
  - Renal tubular or parenchymal disease
  - Primary or secondary hyperaldosteronism

- **Gastrointestinal losses**:
  - Vomiting
  - Diarrhea
  - N/G tube
  - Intestinal Malabsorption

- **Hypomagnesemia**
  - Promotes urinary K loss
  - Cannot correct k until Mg is corrected!!

- **K+ Redistribution into cells**
  - Insulin
  - Beta- Agonists : Albuterol, Dobutamine
  - Alkalosis



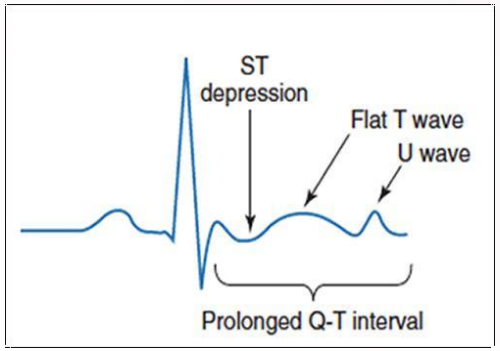
Hypokalemia

Signs/Symptoms

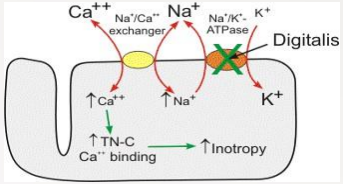
- **Muscle Weakness**
  - Fatigue.
  - Paralysis.

- **Arrhythmias**
  - Prolong normal cardiac conduction.
  - PACs, PVCs, Bradycardia.

- **ECG Changes**
  - **Flattened T-wave and** maybe inverted if severe.
  - U-wave appearance.



- Polyuria and polydipsia.
- Increase sensitivity to Digoxin toxicity.



Management

- **Treat** the **underlying cause** and stop any medication that may cause hypokalemia.
- Increase dietary k+.
- **Oral vs IV Potassium**
  - **Oral KCL** is the preferred safest method of replacement.
  - **IV** Potassium is irritant and painful. It can cause phlebitis or arrythmias if infused quickly. IV is used in **severe** cases of hypokalemia.

- **CORRECT HYPOMAGNESIMIA !!**

Hyperkalemia

Mild: >5.5 mEq/L

Severe: >6.5 mEq/L

Hyperkalemia

Causes

Transcellular shifting (from intracellular to extracellular)

- Acidosis
- Tissue/cell breakdown (chemotherapy, hemolysis, burns)
- GI bleeding
- Insulin deficiency—insulin stimulates the Na+-K+-ATPase and causes K+ to shift into cells.
- Rapid administration of β-blocker

Pseudohyperkalemia (spurious)

- This refers to an artificially elevated plasma K+ concentration due to K+ movement out of cells immediately before or after venipuncture. (Repeat the test to confirm)

- If the sample is not processed quickly, some red blood cells will hemolyze and cause spillage of K+ leading to a falsely elevated result.

Increased total-body potassium

- Renal failure (acute or chronic)
- Hypoaldosteronism states
- Drug-induced: ACE inhibitors, potassium-sparing diuretics (spironolactone)
- Blood transfusion—usually due to lysed cells

Clinical Presentation

The most severe symptom of hyperkalemia is impaired electrical conduction in the heart.

Cardiac symptoms are more likely to occur with increasing severity and acuity of hyperkalemia.

Muscular symptoms may be observed, and these include weakness and paralysis,

Muscular symptoms

- Muscle weakness
- Paralysis
- Decreased deep tendon reflexes
- Paraesthesia

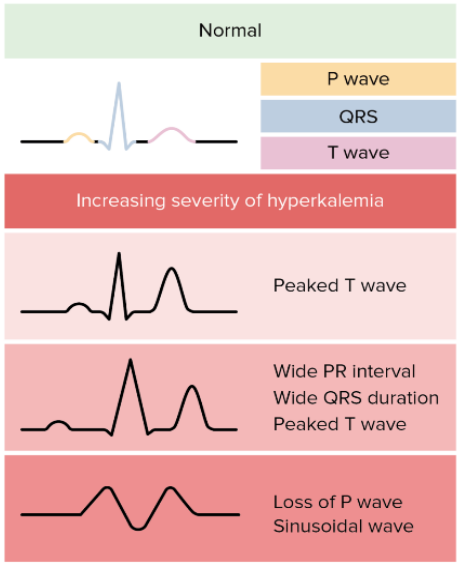
Cardiac Symptoms

Arrhythmias:

- Sinus bradycardia
- Ventricular tachycardia, ventricular fibrillation, and/or asystole if severe

ECG changes :

- Peaked T waves and short QT interval
- PR interval prolongation and QRS widening
- Loss of P waves QRS widens



Management

If Hyperkalemia is severe ?

ECG changes, arrhythmia, or severe muscle weakness/paralysis.
Serum K+ usually > 6.5 mEq/L.

Options for emergency treatment:

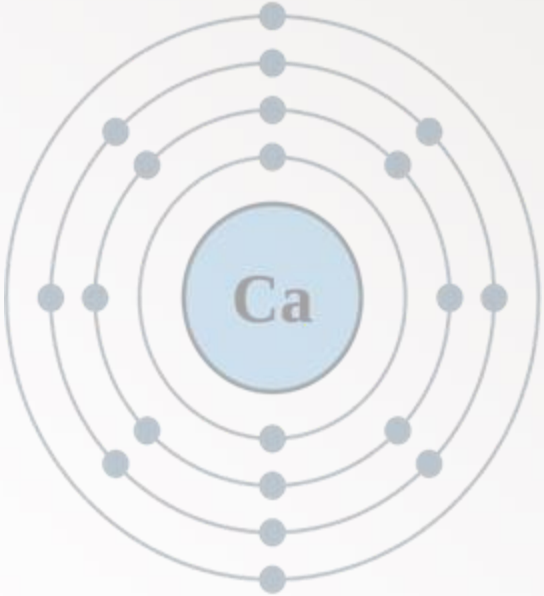
1. IV calcium gluconate to stabilize myocardium
2. 10 units of Insulin with dextrose I.V to shift K+ into cells
3. Sodium bicarbonate corrects acidosis

✓ Monitor serum K+ frequently
✓ Cardiac monitoring and/or repeat ECG while treating

➢ To Remove potassium from the body : Kayexalate, Hemodialysis

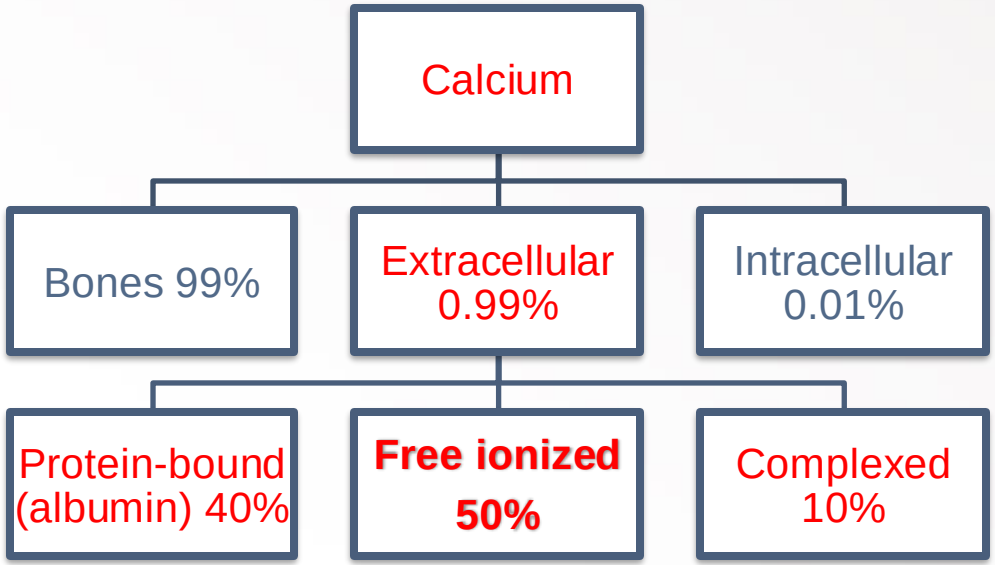
Calcium

8.5 - 10.5 mg/di.



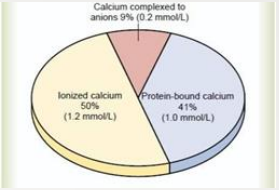
Calcium in our bodies

- The most abundant **metal** in our bodies.
- 99% in bones, 1% in serum and soft tissue .
- **Normal value** ***: 8.5 - 10.5 mg/di.***



- **Calcium in plasma or serum exists in <u>three forms</u>:**

1. <u>Protein-bound calcium (40%)</u> most calcium ions are bound to albumin, so the total calcium concentration fluctuates with the protein (albumin) concentration.



2. <u>Ionized or free calcium (50%)</u> is the physiologically **active form .**

3. <u>Complexed or chelated calcium(10%)</u> is bound to phosphate, bicarbonate, sulfate, citrate, and lactate.

Corrected Ca 2 + = measured Ca + 0.8 × [4 - measured albumin]

Hyperalbuminemia

-non diffusible calcium will bind w/ excess albumin, but free ionized Ca+ will stay the same ; hormone control.

→**Pseudo** hypercalcemia.

Hypoalbuminemia

-loss of total calcium with albumin loss, but free ionized Ca+ will stay the same ; hormone control.

→**Pseudo** hypocalcemia

Calcium Regulation

- **by Hormonal control, Albumin, and Blood's pH .**

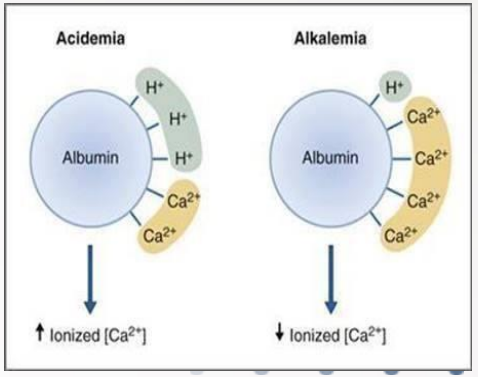
Hormonal Control Maintenance of calcium balance is a function of ↑PTH, ↓calcitonin , and ↑vitamin D , and their target organs bone,kidney, and gut.

Acidosis (low pH) (high H^+)

-high H^+ will repel Ca^+ from albumin$^-$ which will lead to high amount of ionized calcium in serum → **Hypercalcemia**

Alkalosis (high pH) (low H^+)

-albumin$^-$ will bind with ionized calcium leading to low amount of it in serum →**Hypocalcemia.**



Hypercalcemia

Causes:

1. **Hyperparathyroidism**

   (most common in outpatients)

   ↑ PTH → Osteoclastic bone resorption.

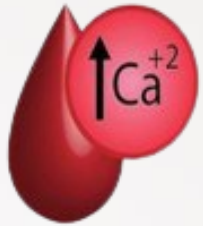
2. **Malignant tumor (PTH-rp)**

   (most common cause in inpatients) bony metastases, Multiple Myeloma

3. **Excess Vitamin D.**

4. **Medications→ Thiazide diuretics.**

Serum calcium **>10.5 mg/dl**



Hypercalcemia

Sign and Symptoms

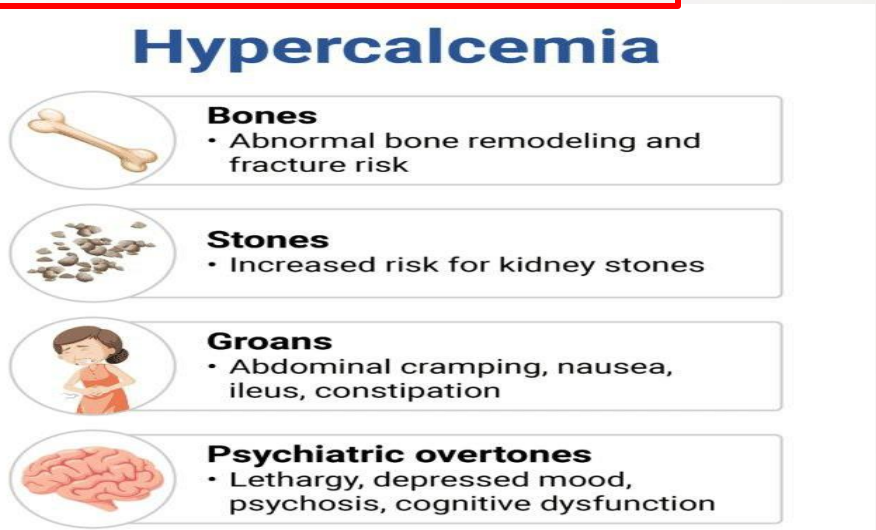
Fatigue, confusion, lethargy, coma.

#Muscle weakness, hyporeflexia.

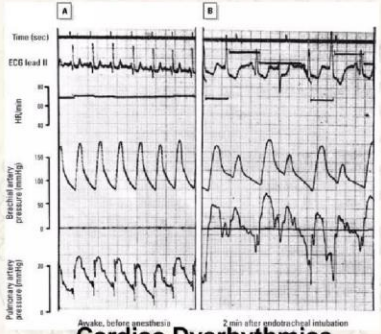
Bradycardia > cardiac arrest.

#Anorexia, nausea/vomiting, decreased bowel sounds, constipation.

Polyuria, renal calculi, renal failure.

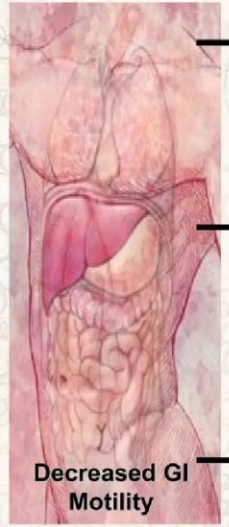


CLINICAL MANIFESTATIONS OF HYPERCALCEMIA



Cardiac Dysrhythmias

Mental status changes: lethargy, confusion, memory loss



Decreased GI Motility

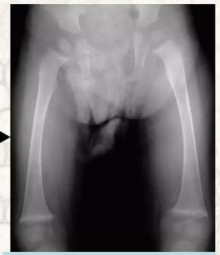
Constipation

Nausea

Vomiting

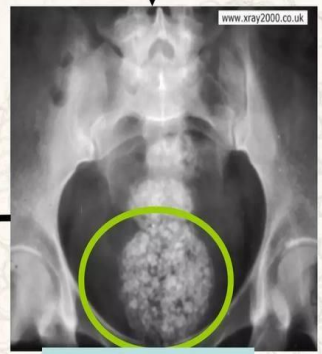
CLINICAL MANIFESTATIONS OF HYPERCALCEMIA

Immobilization

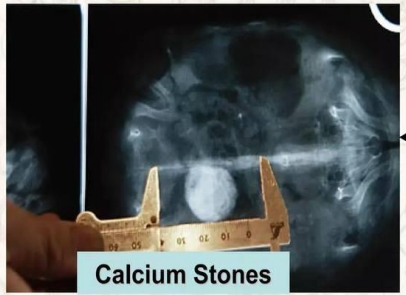


Bone Demineralization

Calcium accumulates in the ECF and passes through the kidneys



Ca Precipitation



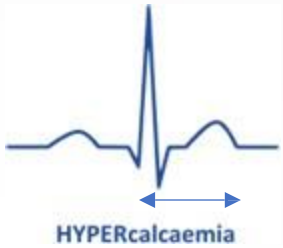
Calcium Stones

Hypercalcemia

Diagnosis

1. **High Ca^{+2} in blood >10.5 mg/dl**
2. **ECG → Bradycardia , AV block**

Short QT interval



3. Lab Tests (to know underlying cause).

Magnesium, albumin, and ionized calcium.

Amylase/ lipase / Serum PO43 / PTH.

What do we do?

Treatment

To lower Ca^{+2} levels.

1. Increase urinary retention.

   [IV Fluids; first step]

   [loop diuretics ; Furosemide]
2. Increase GI excretion.

   [glucocorticoids]
3. Prevent bone resorption

   [bisphosphonates & calcitonin]

Hypocalcemia

Causes of hypocalcemia :

1- **Hypoparathyroidism** (after neck surgery, autoimmune, infiltrative diseases)

2- Large volume blood transfusions (citrate in transfused blood can bind calcium)

3- Magnesium depletion (decreased PTH release) (patients on iv feeding for long time)

4- Acute pancreatitis (calcium binds to free fatty acids released by lipase)

5- Acute respiratory alkalosis (increases binding of calcium to albumin)

6- renal failure; acute and chronic

7- severe trauma (blood loss) (albumin loss)

8- crush injury (renal failure)

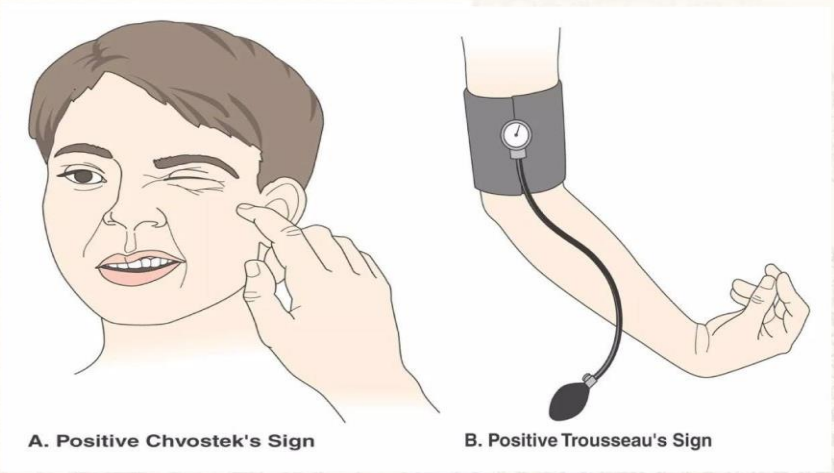
all soft tissue injury can cause hypocalcemia

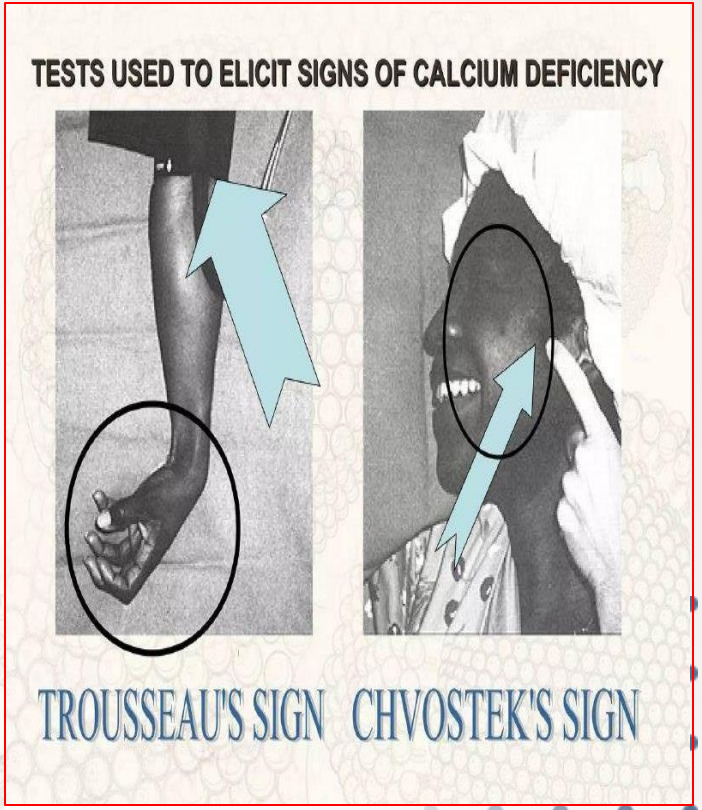
Serum calcium **<8.5 mg/dl**

Hypocalcemia

Signs and Symptoms

1. Tetany (involuntary muscle movements).
2. Chvostek's sign (facial nerve twitch).
3. Trousseau's sign (wrist spasms).
4. Muscle cramps / abdominal pain / perioral tingling / seizures





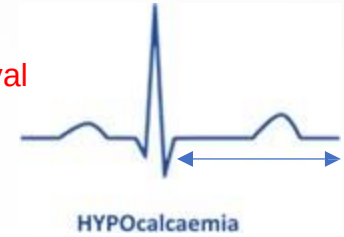
Hypocalcemia

Diagnosis

1. **low Ca^{+2} in blood <8.5 mg/dl**
2. **ECG → Prolonged ST segment**

   prolonged QT interval.

prolonged QT interval



3. Lab Tests (to know underlying cause).

Magnesium, albumin, and ionized calcium.

Amylase/ lipase / Serum PO43 / PTH.

Treatment

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. **It is also important to correct hypomagnesemia**. It is very difficult to correct the calcium level if the magnesium is not replaced first.

MAGNESIUM
(1.8 TO 2.5 MG/DL)

FUN FACTS

ONE OF THE FEW ELEMENTS THAT ARE ESSENTIAL FOR LIFE

THE SECOND MOST ABUNDANT METAL IN SEAWATER

THE AMOUNT OF MAGNESIUM IN OUR DIETS AFFECTS OUR SLEEP

Location in the body

-Most of the magnesium in the body (two-thirds) is in bones.
-The remainder (one-third) is intracellular.

Only 1% of magnesium is extracellular.

Magnesium absorption and balance—About 30% to 40% of dietary magnesium is absorbed in the GI tract, but this percentage increases when magnesium levels are low. The kidney has a great capacity to reabsorb magnesium and is the major regulator of magnesium balance.

Hypomagnesemia

Causes :-

1. GI causes Malabsorption, steatorrheic states (most common cause), Prolonged fasting

2. Alcoholism (common cause)—due to increased urinary excretion as well as frequently concomitant diarrhea, pancreatitis, and poor diet

3. Renal causes

4. DKA, burns, pancreatitis, lactation

Hypomagnesemia

Clinical features :-

1. Marked neuromuscular and CNS hyperirritability

2. Effect on calcium levels: coexisting hypocalcemia is common because of decreased release of PTH and bone resistance to PTH when Mg^{2+} is low

3. Effect on potassium levels Coexisting hypokalemia—in up to 50% of cases, usually due to etiology which leads to loss of both

4. ECG changes—prolonged QT interval, T-wave flattening, and ultimately, torsade de pointes

Hypomagnesemia

Treatment :-

1. For mild hypomagnesemia—oral Mg^{2+}
(e.g., magnesium oxide)

2. For severe hypomagnesemia—parenteral Mg^{2+}
(e.g., magnesium sulfate)

Hypermagnesemia

Causes :-

1. Renal failure (most common cause)

2. Early-stage burns, massive trauma or surgical stress,

3. Rhabdomyolysis

4. Iatrogenic—usually in the obstetric setting in women with preeclampsia or eclampsia being treated with magnesium sulfate

Hypermagnesemia

Clinical features :-

1. Nausea, weakness

2. Facial paresthesia

3. Progressive loss of deep tendon reflexes (classically the first sign)

4. ECG changes resemble those seen with hyperkalemia (increased P-R interval, widened QRS complex, and elevated T waves)

5. Death is usually caused by respiratory failure or cardiac arrest

Hypermagnesemia

Treatment :-

1. Withhold exogenously administered magnesium.
2. Prescribe IV calcium gluconate for emergent symptoms (cardioprotection as in hyperkalemia).
3. Administer saline and furosemide.
4. Order dialysis in renal failure patients.
5. Prepare to intubate if respiratory depression is severe.

Thank You