

Marta Serwanska-Swietek, MD, PhD

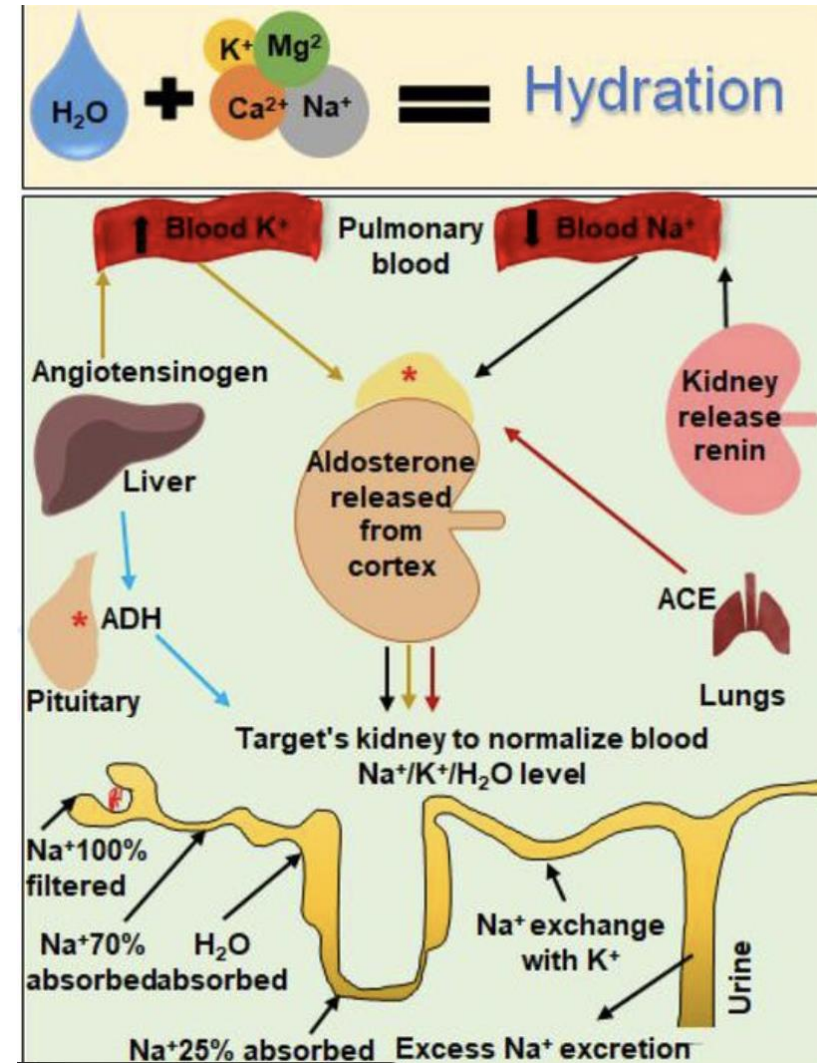
Department of Transplantation, Immunology,
Nephrology and Internal Medicine
Infant Jesus Clinical Hospital
Medical University of Warsaw

marta.serwanska-swietek@wum.edu.pl



Learning objectives:

- Recognize common electrolyte disorders.
- Interpret arterial blood gases.
- Identify acid–base disturbances.
- Explain renal mechanisms maintaining homeostasis.
- Recognize life-threatening abnormalities.
- Initiate appropriate management.
- Understand indications for urgent dialysis.



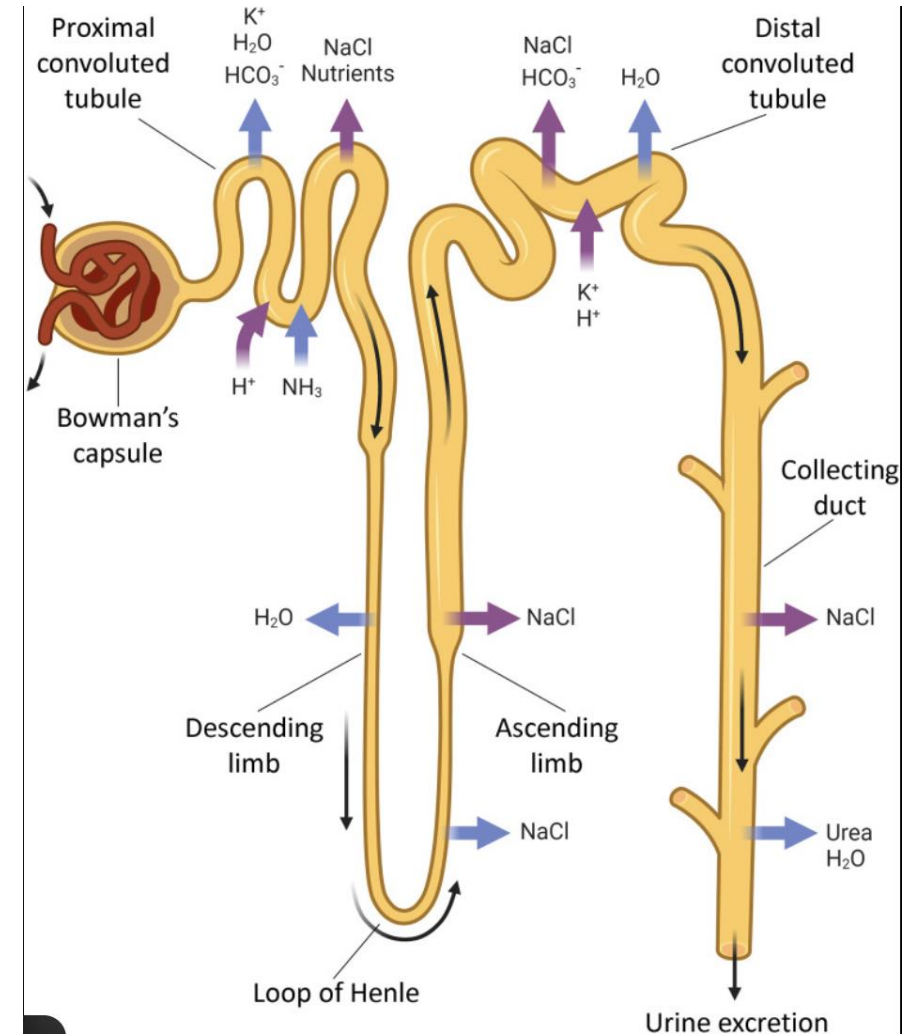
Kidney Regulation of Homeostasis

The kidneys regulate

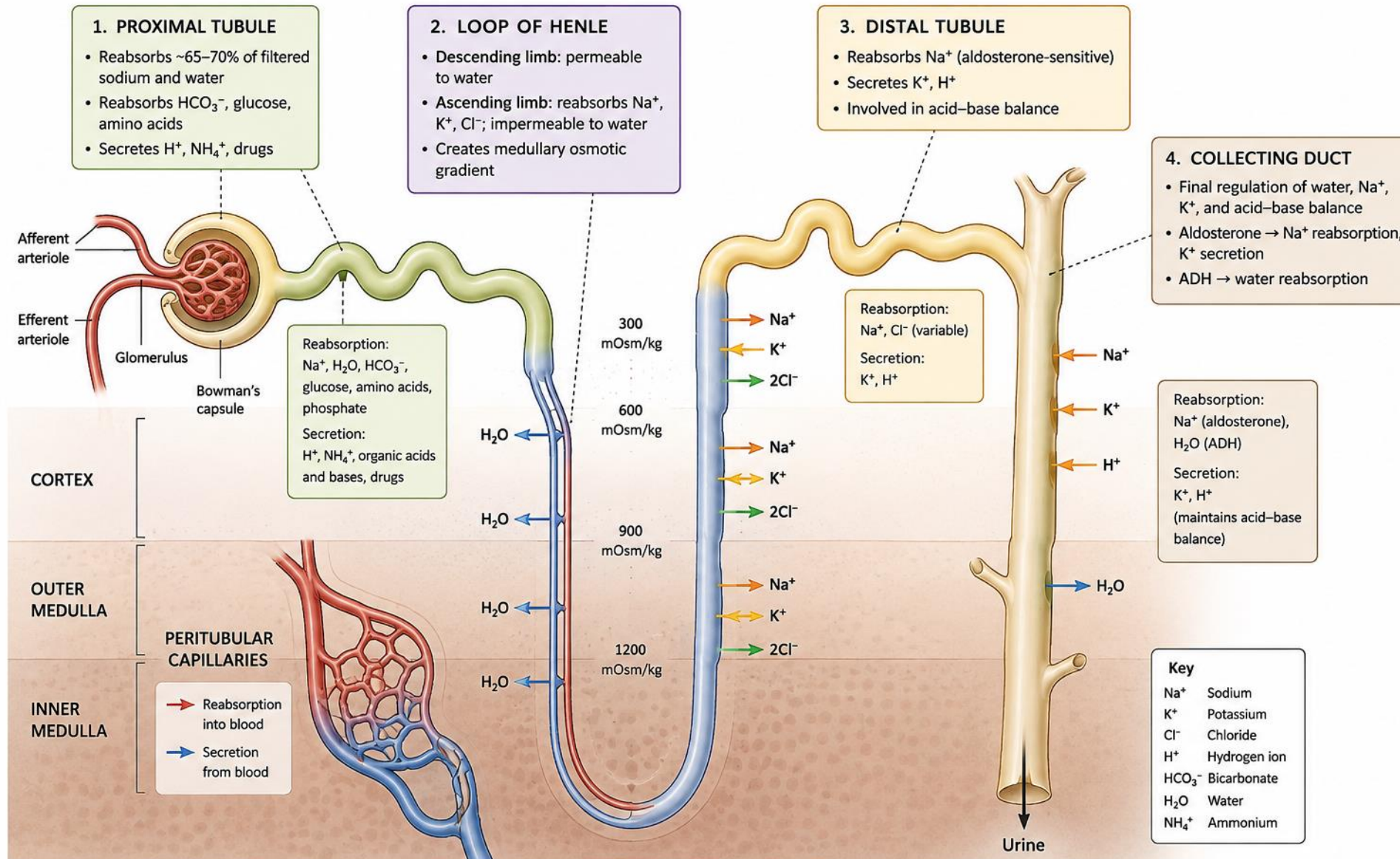
- Sodium and water balance
- Potassium homeostasis
- Calcium and phosphate metabolism
- Magnesium balance
- Hydrogen ion secretion
- Bicarbonate regeneration

The kidneys maintain extracellular fluid composition.

Renal failure commonly leads to electrolyte and acid–base disturbances.



The Nephron – Functional Units of the Kidney



HYPERKALEMIA

Elevated serum potassium

$K^+ > 5.5 \text{ mmol/L}$

CAUSES



CKD

Reduced K^+ excretion



AKI

Decreased K^+ excretion



RAAS inhibitors

(ACEi, ARB, aliskiren)

Decreased aldosterone
→ ↓ K^+ excretion



Potassium-sparing diuretics

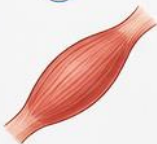
(spironolactone, eplerenone, amiloride, triamterene)
↓ K^+ secretion



H^+

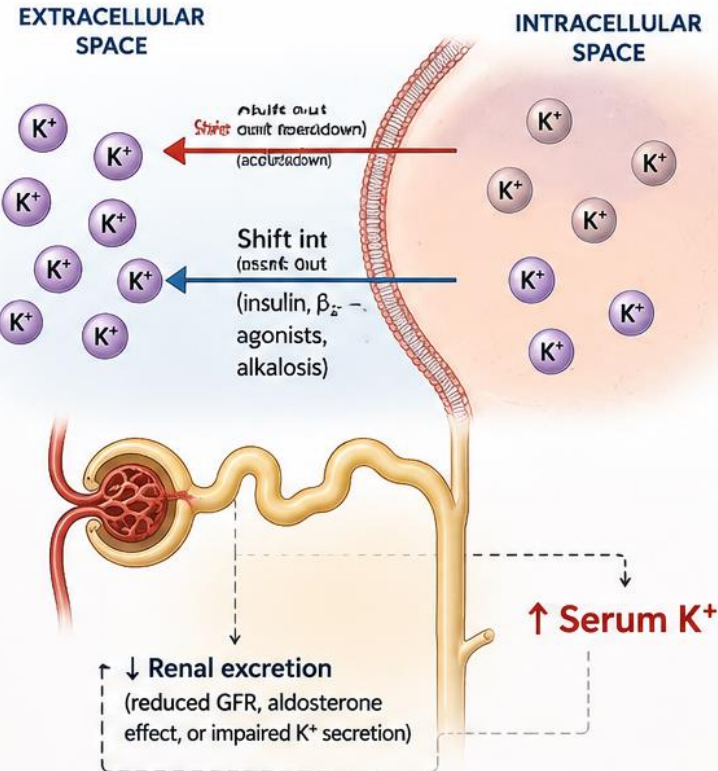
Metabolic acidosis

H^+ out, K^+ in → ↑ serum K^+



Tissue breakdown

(rhabdomyolysis, hemolysis, tumor lysis, burns)
Release of intracellular K^+



CLINICAL FEATURES



Muscle weakness
Fatigue



Paresthesias
(tingling, numbness)

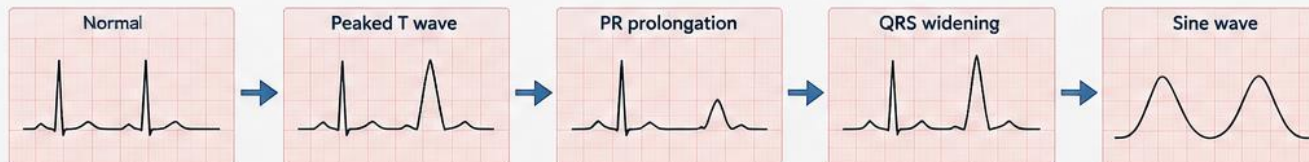


Cardiac arrhythmias

ECG changes:

- Peaked T waves
- PR prolongation
- QRS widening
- Sine wave (severe)

ECG CHANGES IN HYPERKALEMIA



SEVERE HYPERKALEMIA

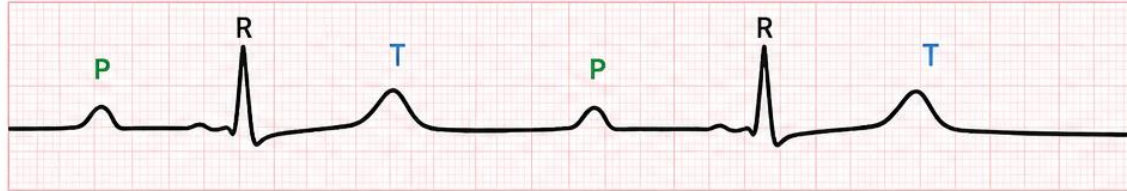
Risk of life-threatening arrhythmias

**URGENT TREATMENT
REQUIRED**



ECG Changes in Hyperkalemia

Normal ECG



- Normal P wave
- Normal PR interval
- Narrow QRS
- Normal T wave

Peaked T waves



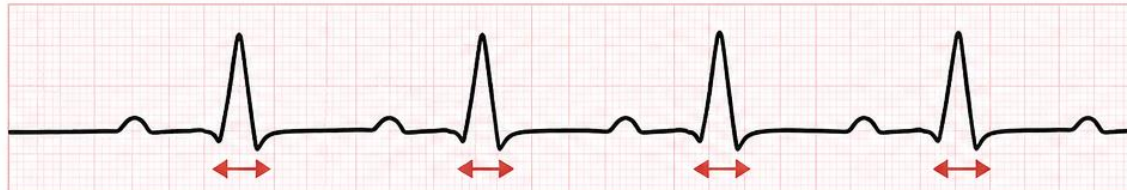
- Tall, narrow, peaked T waves

PR prolongation



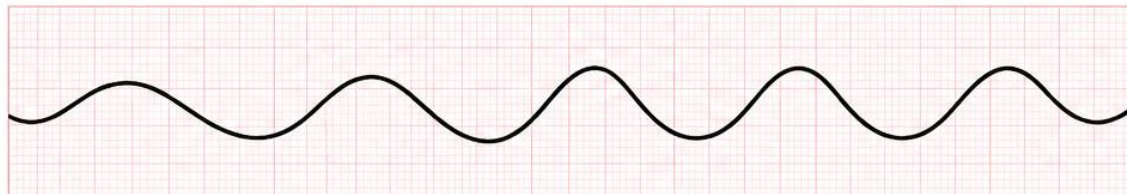
- Prolonged PR interval (> 200 ms)

QRS widening



- Widened QRS complex (> 120 ms)

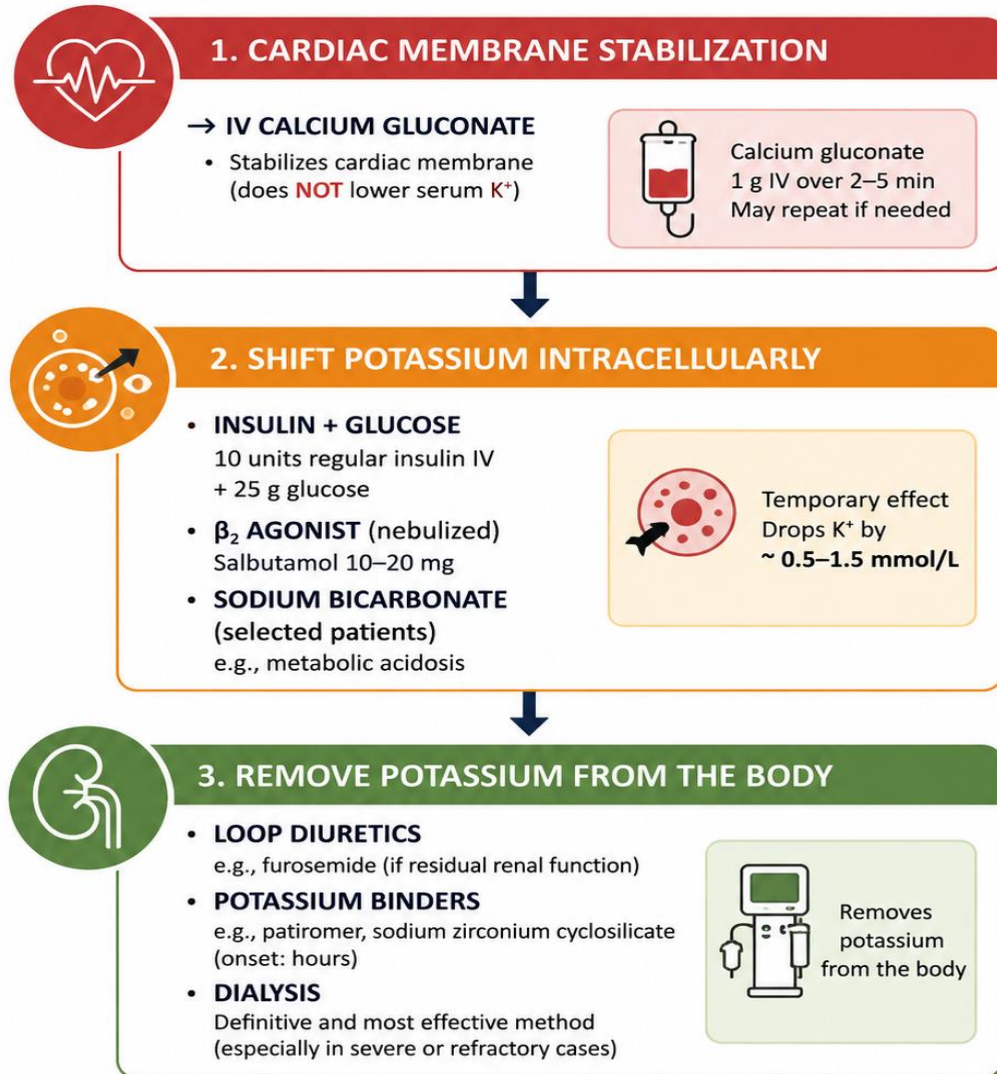
Sine wave (very severe hyperkalemia)



- Sine wave pattern
- No distinct P, QRS, or T
- Risk of ventricular fibrillation / asystole

HYPERKALEMIA MANAGEMENT

STEPWISE APPROACH



Always treat the cause, monitor ECG,
and recheck potassium levels frequently.

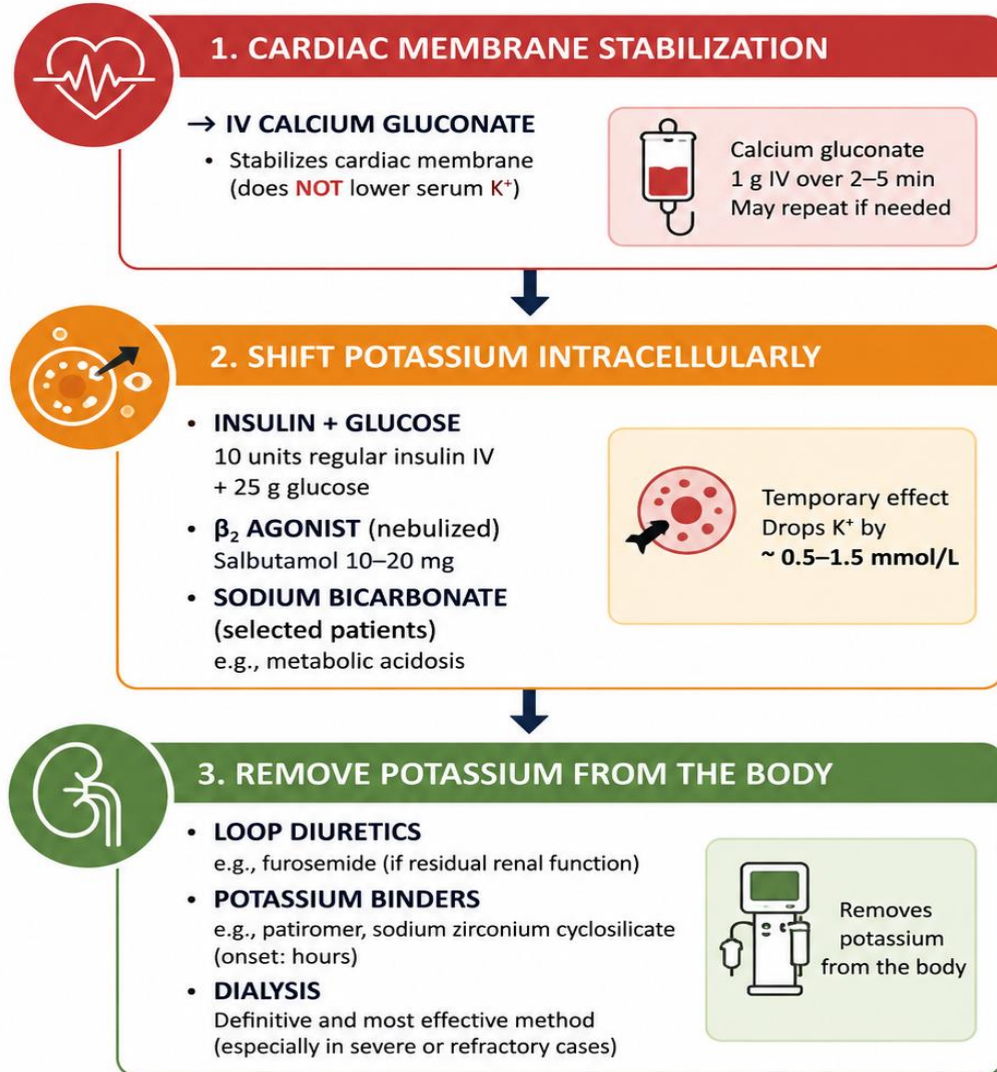
When dialysis?

AEIOU

especially refractory hyperkalemia

HYPERKALEMIA MANAGEMENT

STEPWISE APPROACH



Always treat the cause, monitor ECG, and recheck potassium levels frequently.



When dialysis?

AEIOU

especially refractory hyperkalemia

When is dialysis indicated? – AEIOU

A – Acidosis

- Severe metabolic acidosis
- **pH < 7.1** despite appropriate medical therapy
- Particularly if associated with hemodynamic instability

E – Electrolyte abnormalities

- **Refractory hyperkalemia**
- Usually $K^+ \geq 6.5$ mmol/L
- Or hyperkalemia with **ECG changes**
- Not responding to medical treatment

I – Intoxications

Dialyzable toxins, including:

- Lithium
- Methanol
- Ethylene glycol
- Salicylates
- Valproic acid (selected cases)

O – Overload

- Severe fluid overload
- Pulmonary edema
- Refractory hypertension
- Failure of diuretic therapy

U – Uremia

Clinical manifestations of uremia:

- Uremic encephalopathy
- Uremic pericarditis
- Uremic bleeding (platelet dysfunction)
- Persistent nausea/vomiting
- Severe pruritus with advanced kidney failure (selected cases)

Dialysis is initiated because of life-threatening complications—not because of a specific creatinine or eGFR value.

Urgent dialysis – what happens next?

1. Identify the emergency

Hyperkalemia • Pulmonary edema • Severe acidosis • Uremic complications • Intoxication



2. Stabilize the patient

Treat immediately while preparing dialysis

e.g. IV calcium + insulin/glucose in severe hyperkalemia



3. Obtain vascular access

Temporary double-lumen dialysis catheter

Usually internal jugular



4. Choose renal replacement therapy



5. Dialysis prescription depends on the indication

Duration • blood flow • dialysate composition • ultrafiltration



Reassess → repeat dialysis if necessary

Patient	Preferred approach
Hemodynamically stable	Intermittent hemodialysis (IHD)
Hemodynamically unstable / ICU	CRRT
Intermediate situation	SLED
Selected intoxications	Usually IHD

One dialysis session does not necessarily solve the problem.

The prescription and further treatment depend on the clinical response.

Hyponatremia

The most common electrolyte disorder in clinical practice



DEFINITION

Serum sodium (Na^+) < **135 mmol/L**



CLASSIFICATION



Hypovolemic



Euvolemic



Hypervolemic



CAUSES



SIADH



Heart failure



Cirrhosis



CKD



Diuretics



Other (e.g. low solute intake)



SYMPTOMS



Headache



Confusion



Seizures



Coma



NEVER CORRECT TOO RAPIDLY



Risk of osmotic demyelination syndrome (ODS)

Follow recommended correction limits!



Always **identify** the cause, **assess** volume status and correct sodium **safely** and appropriately.



Acid–Base Disorders

Disorder	pH	HCO ₃ ⁻	PaCO ₂
Metabolic acidosis	↓	↓	compensation ↓
Metabolic alkalosis	↑	↑	compensation ↑
Respiratory acidosis	↓	↑ (chronic)	↑
Respiratory alkalosis	↑	↓ (chronic)	↓

ROME

Respiratory Opposite

Metabolic Equal

METABOLIC ACIDOSIS

A primary decrease in serum bicarbonate (HCO_3^-) leading to a reduction in blood pH (< 7.35)



CAUSES



AKI



CKD



Diarrhea



Diabetic ketoacidosis



Lactic acidosis



Toxins



CALCULATE

ANION GAP

$$\text{AG} = \text{Na} - (\text{Cl} + \text{HCO}_3^-)$$

Normal:

8–12 mmol/L



HIGH ANION GAP

- Lactate
- Ketones
- Uremia
- Toxins



NORMAL ANION GAP

- Diarrhea
- Renal tubular acidosis



Think systematically:

Identify the cause → **Calculate** the anion gap → **Determine** the underlying disorder



METABOLIC ALKALOSIS

Increase in serum bicarbonate due to gain of base or loss of acid



DEFINITION

Elevated serum bicarbonate (HCO_3^-) **> 26 mmol/L**



COMMON CAUSES



Vomiting



Loop diuretics



Mineralocorticoid excess



TREATMENT



Volume replacement



Correct potassium



Treat underlying disease



Stop offending medications



Urine chloride helps identify the mechanism of metabolic alkalosis:

<20 mmol/L → chloride depletion / saline-responsive

>20 mmol/L → renal chloride loss or mineralocorticoid excess

Calcium, Phosphate Disorders

Key electrolyte imbalances and their clinical consequences



Ca^{2+}

CALCIUM DISORDERS

$\downarrow$
 Ca^{2+}

HYPOCALCEMIA

- Tetany
- Prolonged QT



$\uparrow$
 Ca^{2+}

HYPERCALCEMIA

- Stones
- Bones
- Abdominal groans
- Psychiatric overtones



PO_4^{3-}

HYPERPHOSPHATEMIA



- Chronic Kidney Disease (CKD)
Reduced phosphate excretion



- Secondary Hyperparathyroidism
 $\uparrow$ PTH due to phosphate retention and hypocalcemia



- Vascular Calcification
Increased risk of cardiovascular events

Remember

High phosphate binds calcium $\rightarrow$ low calcium and stimulates PTH release

Mg^{2+}

MAGNESIUM

$\downarrow$
 Mg^{2+}

HYPOMAGNESEMIA

$\rightarrow$ Arrhythmias



$\uparrow$
 Mg^{2+}

HYPERMAGNESEMIA

$\rightarrow$ Advanced CKD



Clinical features of hypermagnesemia

- Nausea, vomiting
- Flushing
- Hypotension
- Muscle weakness
- Decreased reflexes
- Respiratory depression, coma



KEY TAKEAWAYS



Check Ca^{2+} , PO_4^{3-} , Mg^{2+} regularly, especially in CKD.



Correct abnormalities and treat the underlying cause.



Control phosphate intake in CKD to prevent complications.



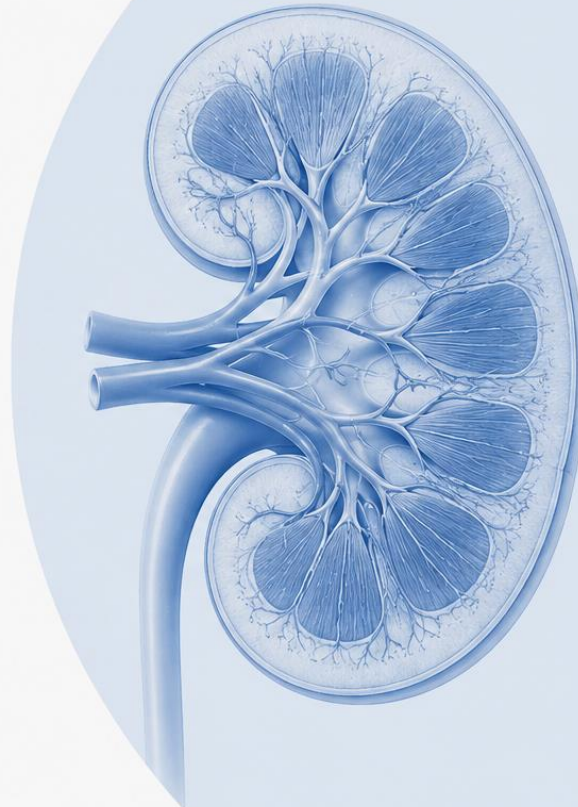
Early recognition prevents serious cardiovascular and skeletal events.

NORMAL RANGES (approx.)

Calcium (total)	2.15–2.55 mmol/L
Phosphate	0.81–1.45 mmol/L
Magnesium	0.70–1.00 mmol/L

Note: Management should be individualized based on clinical context and severity.

CASE 1



Clinical Case

Case

72-year-old man

CKD stage G5

K^+ 6.8 mmol/L

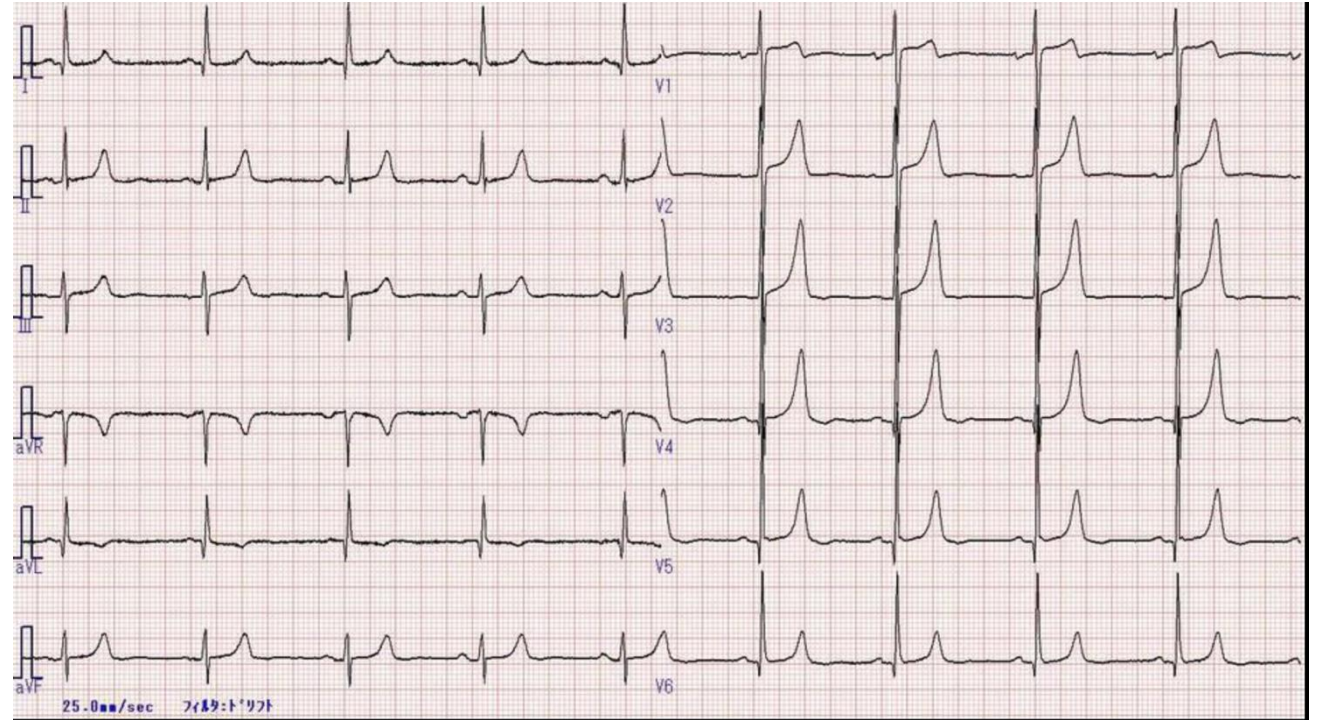
HCO_3^- 14 mmol/L

pH 7.18

Creatinine 7.2 mg/dL

ECG:

peaked T waves



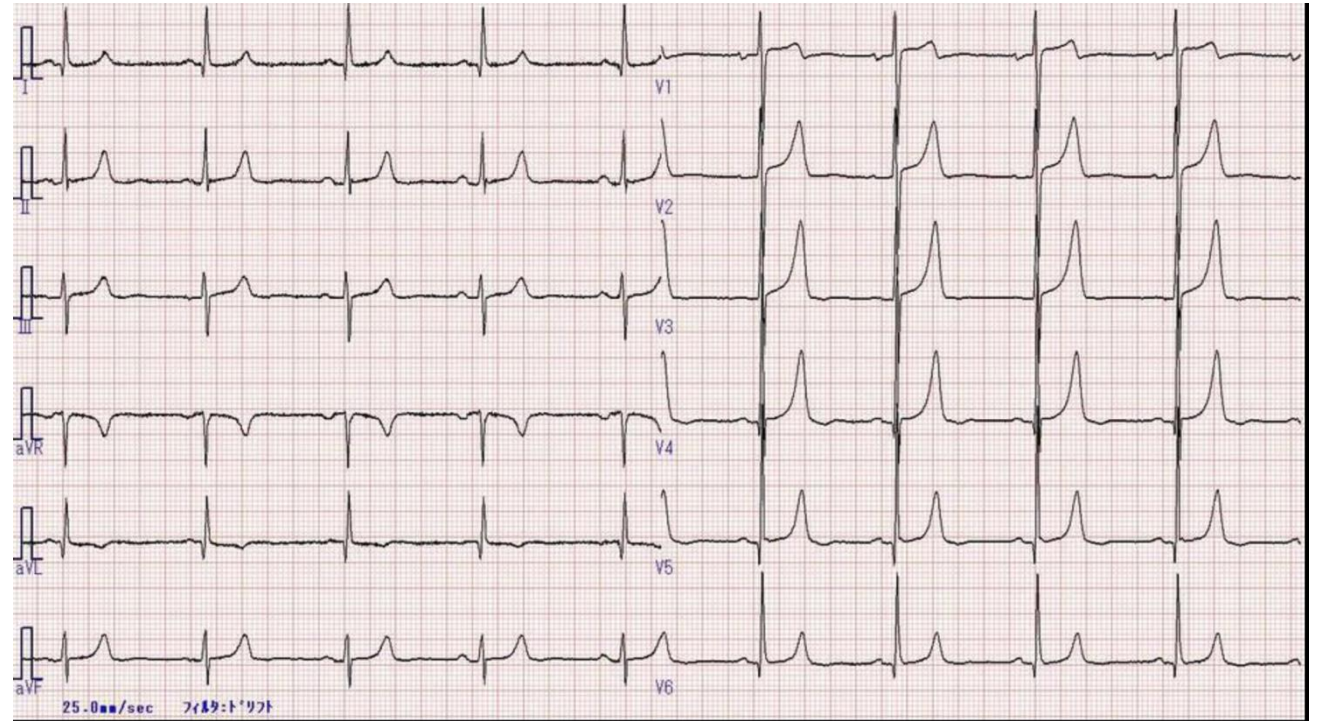
Clinical Case

Case

72-year-old man
CKD stage G5
 K^+ 6.8 mmol/L
 HCO_3^- 14 mmol/L
pH 7.18
Creatinine 7.2 mg/dL
ECG:
peaked T waves

Questions

1. What abnormalities are present?



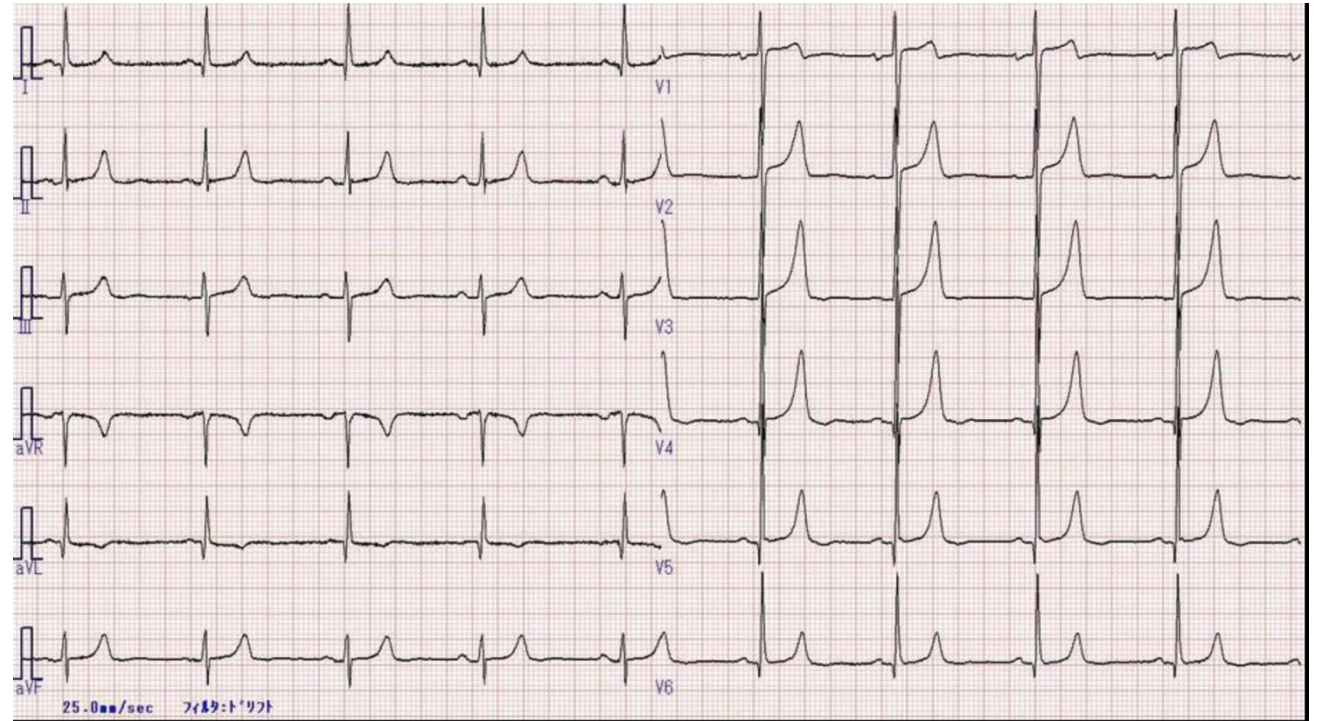
Clinical Case

Case

72-year-old man
CKD stage G5
 K^+ 6.8 mmol/L
 HCO_3^- 14 mmol/L
pH 7.18
Creatinine 7.2 mg/dL
ECG:
peaked T waves

Questions

1. What abnormalities are present?
2. Which treatment should be given first?



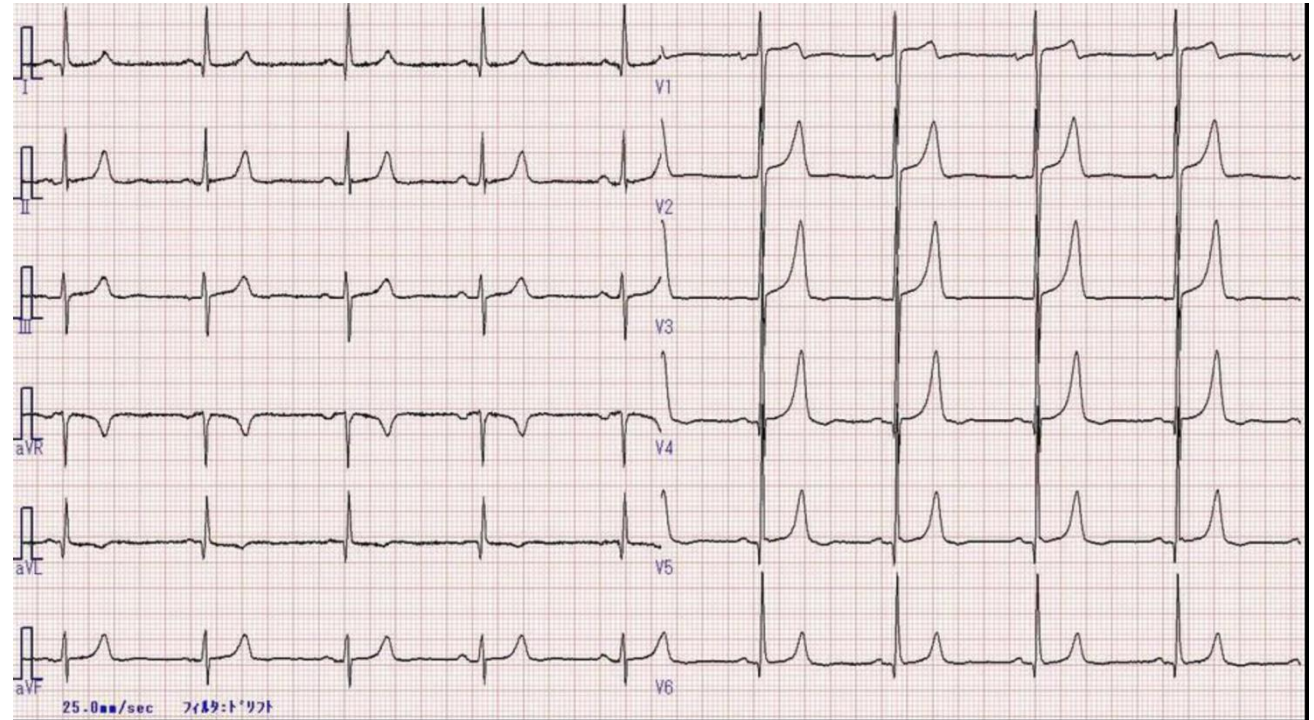
Clinical Case

Case

72-year-old man
CKD stage G5
 K^+ 6.8 mmol/L
 HCO_3^- 14 mmol/L
pH 7.18
Creatinine 7.2 mg/dL
ECG:
peaked T waves

Questions

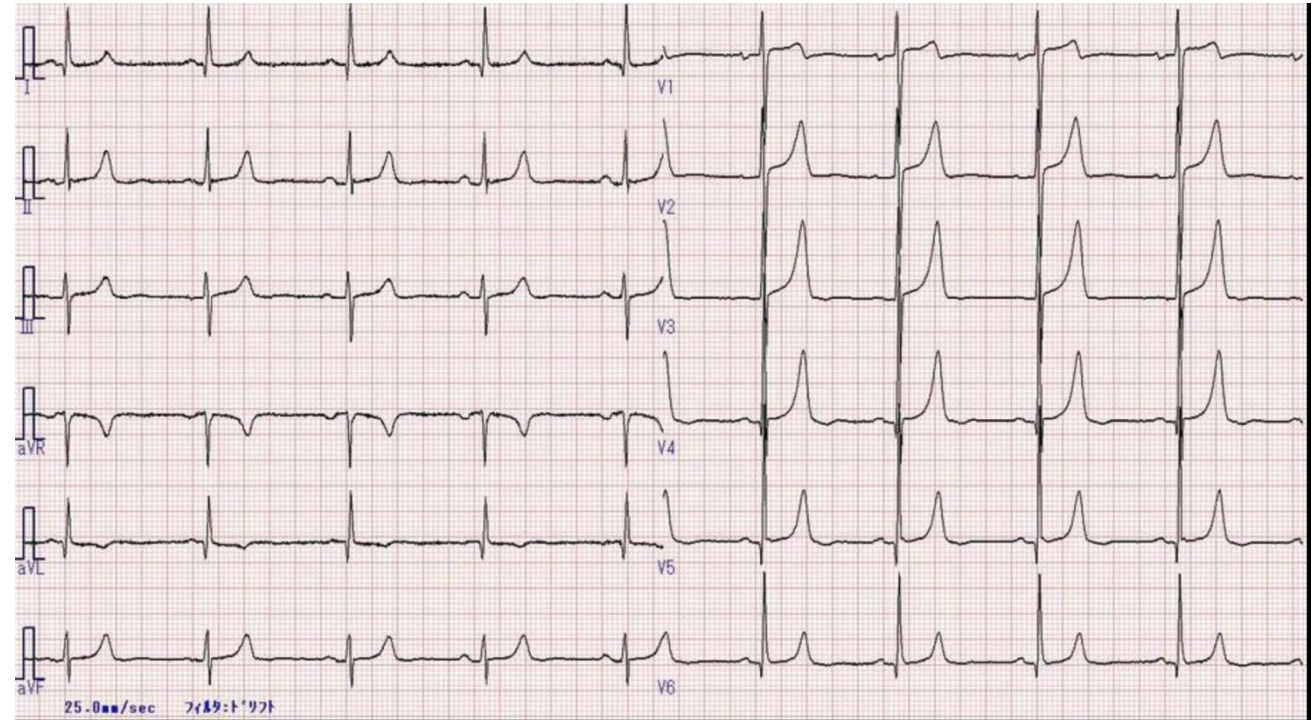
1. What abnormalities are present?
2. Which treatment should be given first?
3. Does this patient require dialysis?



Clinical Case

Case

72-year-old man
CKD stage G5
 K^+ 6.8 mmol/L
 HCO_3^- 14 mmol/L
pH 7.18
Creatinine 7.2 mg/dL
ECG:
peaked T waves



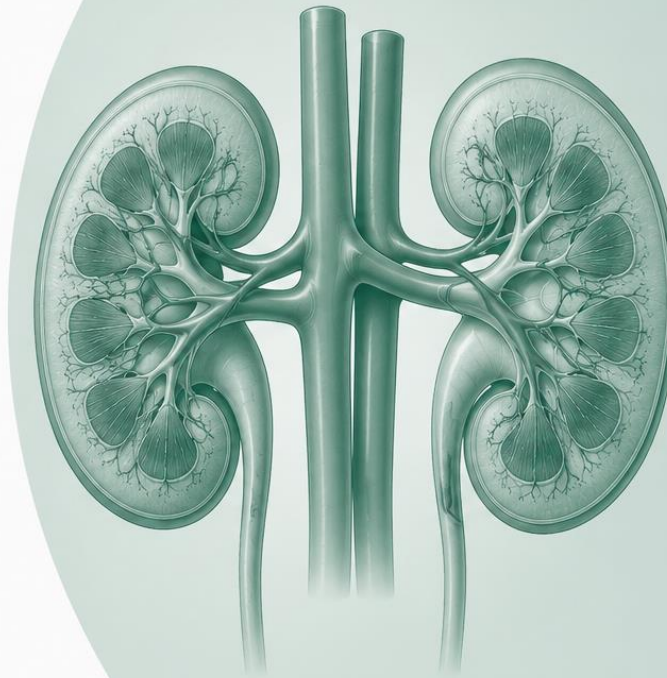
Questions

1. What abnormalities are present?
2. Which treatment should be given first?
3. Does this patient require dialysis?

IV calcium $\rightarrow$ insulin + glucose $\pm$ β_2 -agonist $\rightarrow$ definitive K^+ removal / dialysis

Think in three steps: protect the heart, shift potassium into the cells, and remove potassium from the body

CASE 2



1

CASE – HYPONATREMIA

HISTORY (SUBJECTIVE)



68-year-old woman

Admitted because of nausea,
headache, progressive weakness
and confusion.

1

CASE – HYPONATREMIA

HISTORY (SUBJECTIVE)



68-year-old woman

Admitted because of nausea, headache, progressive weakness and confusion.



HISTORY

- Symptoms started 2 days ago
- No vomiting or diarrhea
- Normal fluid intake
- No edema
- No history of heart failure, liver disease or kidney disease
- Recently started **sertraline**
- No diuretics



ADDITIONAL INFORMATION

- No alcohol abuse
- No endocrine disorders
- No recent surgery
- No trauma



Presenting symptoms:
nausea, headache, weakness, confusion

1

CASE – HYPONATREMIA

HISTORY (SUBJECTIVE)



68-year-old woman

Admitted because of nausea, headache, progressive weakness and confusion.



HISTORY

- Symptoms started 2 days ago
- No vomiting or diarrhea
- Normal fluid intake
- No edema
- No history of heart failure, liver disease or kidney disease
- Recently started **sertraline**
- No diuretics



ADDITIONAL INFORMATION

- No alcohol abuse
- No endocrine disorders
- No recent surgery
- No trauma



Presenting symptoms: nausea, headache, weakness, confusion

2

CASE – HYPONATREMIA

PHYSICAL EXAMINATION (OBJECTIVE)



BP
125/75
mmHg



HR
78
/min



SpO₂
98%



Temp
36.7°C



Patient is confused but responsive.



PHYSICAL EXAMINATION

- ✓ Moist mucous membranes
- ✓ No orthostatic hypotension
- ✓ No peripheral edema
- ✓ No signs of dehydration
- ✓ Lungs clear
- ✓ No signs of heart failure
- ✓ Abdomen soft, non-tender
- ✓ No neurologic focal deficits



VOLUME STATUS
EUVOLEMIC

1 CASE – HYPONATREMIA

HISTORY (SUBJECTIVE)



68-year-old woman

Admitted because of nausea, headache, progressive weakness and confusion.

HISTORY

- Symptoms started 2 days ago
- No vomiting or diarrhea
- Normal fluid intake
- No edema
- No history of heart failure, liver disease or kidney disease
- Recently started **sertraline**
- No diuretics

ADDITIONAL INFORMATION

- No alcohol abuse
- No endocrine disorders
- No recent surgery
- No trauma

 Presenting symptoms: nausea, headache, weakness, confusion

2 CASE – HYPONATREMIA

PHYSICAL EXAMINATION (OBJECTIVE)


BP
125/75
mmHg


HR
78
/min


SpO₂
98%


Temp
36.7°C



Patient is confused but responsive.

PHYSICAL EXAMINATION

- ✓ Moist mucous membranes
- ✓ No orthostatic hypotension
- ✓ No peripheral edema
- ✓ No signs of dehydration
- ✓ Lungs clear
- ✓ No signs of heart failure
- ✓ Abdomen soft, non-tender
- ✓ No neurologic focal deficits



VOLUME STATUS
EUVOLEMIC

3 CASE – HYPONATREMIA

LABORATORY RESULTS

TEST	RESULT
Na ⁺	118 mmol/L
K ⁺	4.1 mmol/L
Glucose	96 mg/dL
Creatinine	0.8 mg/dL
Serum osmolality	255 mOsm/kg
Urine osmolality	480 mOsm/kg
Urine Na ⁺	55 mmol/L
BUN	28 mg/dL
TSH	1.52 μ IU/mL (normal)
Morning cortisol	16.3 μ g/dL (normal)



KEY POINTS FROM RESULTS

- Hypotonic hyponatremia
- Inappropriately concentrated urine in the setting of hyponatremia
- Elevated urine sodium
- Normal thyroid and adrenal function



Interpret the results and determine the most likely cause.

1 CASE – HYPONATREMIA

HISTORY (SUBJECTIVE)



68-year-old woman

Admitted because of nausea, headache, progressive weakness and confusion.

HISTORY

- Symptoms started 2 days ago
- No vomiting or diarrhea
- Normal fluid intake
- No edema
- No history of heart failure, liver disease or kidney disease
- Recently started **sertraline**
- No diuretics

ADDITIONAL INFORMATION

- No alcohol abuse
- No endocrine disorders
- No recent surgery
- No trauma

 Presenting symptoms: nausea, headache, weakness, confusion

2 CASE – HYPONATREMIA

PHYSICAL EXAMINATION (OBJECTIVE)



BP
125/75
mmHg



HR
78
/min



SpO₂
98%



Temp
36.7°C



Patient is confused but responsive.



PHYSICAL EXAMINATION

- ✓ Moist mucous membranes
- ✓ No orthostatic hypotension
- ✓ No peripheral edema
- ✓ No signs of dehydration
- ✓ Lungs clear
- ✓ No signs of heart failure
- ✓ Abdomen soft, non-tender
- ✓ No neurologic focal deficits



VOLUME STATUS
EUVOLEMIC

3 CASE – HYPONATREMIA

LABORATORY RESULTS

TEST	RESULT
Na ⁺	118 mmol/L
K ⁺	4.1 mmol/L
Glucose	96 mg/dL
Creatinine	0.8 mg/dL
Serum osmolality	255 mOsm/kg
Urine osmolality	480 mOsm/kg
Urine Na ⁺	55 mmol/L
BUN	28 mg/dL
TSH	1.52 μ IU/mL (normal)
Morning cortisol	16.3 μ g/dL (normal)



KEY POINTS FROM RESULTS

- Hypotonic hyponatremia
- Inappropriately concentrated urine in the setting of hyponatremia
- Elevated urine sodium
- Normal thyroid and adrenal function



Interpret the results and determine the most likely cause.

SIADH =
Syndrome of
Inappropriate
Antidiuretic
Hormone
Secretion

CASE – HYPONATREMIA

Management and Safe Correction



68-year-old woman with nausea, headache, confusion. Na⁺ 118 mmol/L, hypotonic, euvolemic. Most likely diagnosis: SIADH (sertraline-induced)



Key principle:

Correct sodium safely – too rapid correction may cause osmotic demyelination syndrome (ODS).

1 ASSESS SEVERITY AND URGENCY

Severity of hyponatremia

Mild	Na ⁺ 130–134 mmol/L
Moderate	Na ⁺ 125–129 mmol/L
Severe	Na ⁺ < 125 mmol/L



Neurologic symptoms?

- Confusion
- Headache, nausea
- Weakness
- At risk: seizures, coma



This patient:

- ✓ Severe hyponatremia (Na⁺ 118 mmol/L)
- ✓ Neurologic symptoms present
- Requires urgent treatment

Immediate actions

- ✗ Stop the offending drug (sertraline)
- 🚰 Restrict free water (usually 800–1000 mL/day)
- 📊 Monitor: Na⁺ every 2–4 h initially, urine output, neurologic status

2 INITIAL TREATMENT – SYMPTOMATIC HYPONATREMIA



3% HYPERTONIC SALINE (IV)

Indicated in severe symptoms (this patient)

How to give

- Bolus strategy: 100 mL of 3% NaCl over 10 minutes
- Repeat up to 2–3 times if needed, until symptoms improve
- Reassess after each bolus
- Then switch to controlled correction

Goal of initial therapy



Increase Na⁺ by 4–6 mmol/L in the first 6 hours

to improve neurologic symptoms

After initial improvement

- Continue controlled correction (infusion if needed):
- 3% NaCl infusion 10–20 mL/h (adjust based on Na⁺ trend)
 - Or 0.9% NaCl if appropriate (not in SIADH)
 - Fluid restriction continues



Frequent reassessment

- Check Na⁺ every 2–4 hours
- Monitor urine output and urine osmolality
- Monitor neurologic status

3 SAFE CORRECTION TARGETS



Do not correct too rapidly!

Risk of osmotic demyelination syndrome (ODS) if correction is too fast.

Recommended rate of correction

First 24 hours	≤ 8 mmol/L
First 48 hours	≤ 14–16 mmol/L
High risk patients*	≤ 6 mmol/L in 24 h

* Chronic hyponatremia, malnutrition, alcoholism, liver disease, hypokalemia.



If overcorrection occurs

- Give D5W (5% dextrose in water)
- Desmopressin (DDAVP) 2–4 µg IV/SC
- Aim to re-lower Na⁺ to safe range

Once stabilized

- ✓ Continue fluid restriction
- ✓ Treat underlying cause (stop sertraline; consider alternative if needed)
- ✓ Consider pharmacologic therapy if persistent SIADH (e.g., urea, salt tablets + loop diuretic, or vasopressin antagonists)
- ✓ Follow-up: Na⁺ daily → then every 2–3 days



KEY MESSAGE

In symptomatic hyponatremia, correct sodium to relieve symptoms, but never too fast. Target: +4–6 mmol/L in the first 6 h, and ≤ 8 mmol/L in the first 24 h.

Simplified algorithm



Assess severity & symptoms



Stop cause, fluid restriction



Controlled 3% NaCl bolus if severe symptoms



Monitor correction

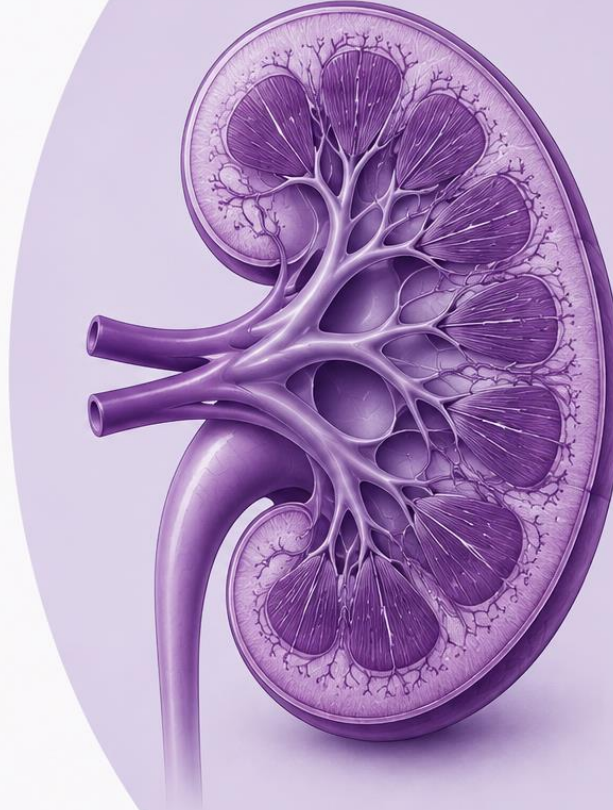


frequently



Treat cause, prevent relapse

CASE 3



1

CLINICAL CASE 3

HISTORY



A 62-year-old man presents to the emergency department with **fatigue, constipation, excessive thirst,** and **increased urination for the past two weeks.**



PAST MEDICAL HISTORY

- Hypertension
- Recurrent kidney stones



MEDICATIONS

- Ramipril
- Vitamin D supplement



PHYSICAL EXAMINATION

- BP: 135/80 mmHg
- HR: 88 bpm
- Mild dehydration
- No peripheral edema
- Normal neurological examination



LABORATORY RESULTS

Calcium (corrected)	3.15 mmol/L (12.6 mg/dL)
Phosphate	0.7 mmol/L
Creatinine	1.4 mg/dL
Albumin	4.1 g/dL
PTH	Elevated



INITIAL THOUGHTS

What are the most important abnormalities?
What is the most likely underlying diagnosis?

2

CLINICAL CASE 3

DIAGNOSIS & MANAGEMENT



DIAGNOSIS

Primary hyperparathyroidism causing PTH-dependent hypercalcemia.



INITIAL MANAGEMENT

- Intravenous **isotonic saline** to restore hydration.
- **Stop** calcium-containing medications if applicable.
- Consider **calcitonin** or **bisphosphonates** only in severe or symptomatic hypercalcemia.



DEFINITIVE TREATMENT

- **Parathyroidectomy** in eligible patients.



KEY TAKE-HOME MESSAGE

Think of hyperparathyroidism when hypercalcemia is accompanied by an **elevated PTH level**.



REMEMBER

Hypercalcemia + elevated PTH = think **Primary Hyperparathyroidism** until proven otherwise.



CLASSIC MANIFESTATIONS

S
STONES



Renal stones,
nephrocalcinosis

B
BONES



Bone pain,
osteoporosis,
fractures

A
ABDOMINAL
GROANS



Constipation,
nausea, abdominal
pain, peptic ulcer
disease, pancreatitis

P
PSYCHIATRIC
OVERTONES



Fatigue, depression,
memory problems,
confusion

Monthly Dialysis Review – What Would You Change?



CASE



58-year-old man
on maintenance HD
HD: 3 × 4 h/week

Monthly laboratory results:

Parameter	Result
Phosphate	10.0 mg/dL ↑
Calcium	8.7 mg/dL
PTH	620 pg/mL
Hemoglobin (Hb)	9.2 g/dL ↓
Ferritin	420 ng/mL
TSAT	27%



What should we change?

1



DIET

Reduce phosphate load

- Prefer fresh, minimally processed foods
- Avoid phosphate additives in processed foods
- Maintain adequate protein intake – do not simply restrict protein

2



BINDERS

Bind phosphate in the gut

- Phosphate binders **must be taken with meals**
- Review adherence and dose
- Choose calcium-based vs non-calcium binder according to Ca/PTH and clinical context

3



DIALYSIS

Remove more phosphate

- Check **dialysis adequacy** and missed/shortened sessions
- Consider **longer or more** frequent dialysis if phosphate remains uncontrolled

4



ANEMIA

Hb 9.2 g/dL

- Check iron status, inflammation and blood loss
- Ensure adequate iron availability
- Review and adjust **ESA therapy** if appropriate



Think beyond the laboratory value:

Diet + Binders + Dialysis + Anemia management

Goal: control phosphate, protect bones & vessels, improve outcomes