
به نام آنکه جان را فکرت آموخت
چراغ دل به نور جان برافروخت

ELECTROLYTE DISORDER IN ICU

DR.M.Amerian

Shahrud University

- ***Potassium:***

- Potassium is the second most abundant cation in the body.

- Approximately 98% of total body potassium is found in the intracellular space, with approximately 2% in the extracellular space.

- The normal serum potassium concentration is 3.5–5.0 meq/L

- Potassium has many physiological functions, including:
- cellular metabolism
- glycogen and protein synthesis
- regulation of the electrical action potential across cell membranes, especially in the myocardium.

- The rate-limiting step for potassium entry into the cells is Na/KATPase that maintains a higher intracellular potassium level.
- Several factors affect the activity of this pump, including:
- insulin, glucagon, cathecholamines, aldosterone, acid-base status, plasma osmolality, and intracellular potassium levels.
- Critically ill patients in the ICU often have abnormalities in one or more of these mediators or conditions.

- ***Hypokalemia***:

- Hypokalemia is defined as a serum potassium concentration below 3.5 meq/L and considered severe if below 2.5 meq/L or if a patient is symptomatic.

- Because hypokalemia results in membrane hyperpolarization and impaired muscular contraction, its signs and symptoms generally involve changes in muscle and cardiovascular function

- Patients with mild hypokalemia (e.g., serum potassium approximately 3–3.4 meq/L) may be asymptomatic.
- Signs and symptoms of hypokalemia include nausea, vomiting, weakness, constipation, paralysis, respiratory compromise, and rhabdomyolysis

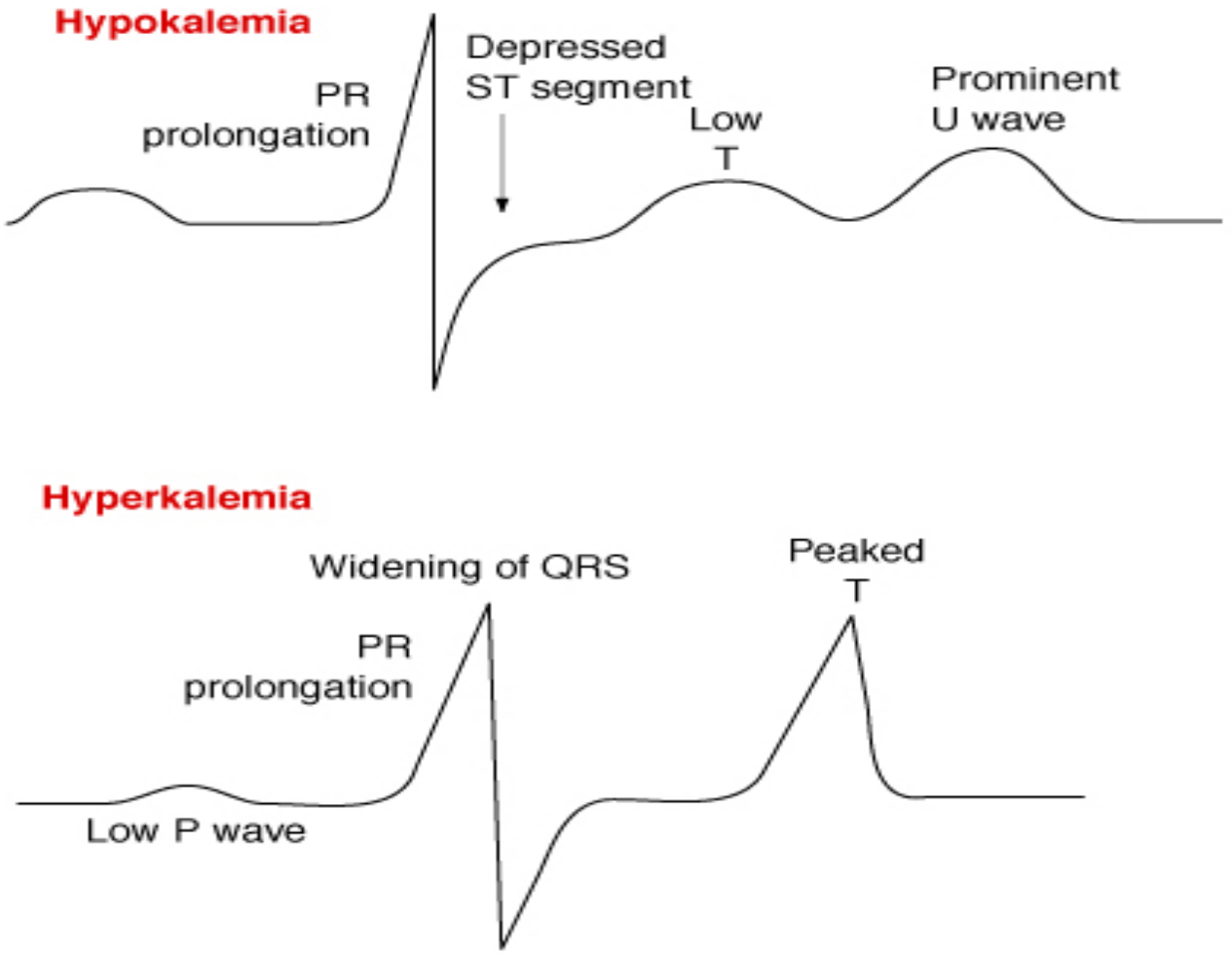

Figure 33-13 Electrocardiographic changes with hyperkalemia and hypokalemia.

- Hypokalemia can develop in ICU patients as a result :
- intracellular shifts of potassium, increased losses of potassium, or, less commonly, decreased ingestion or administration of potassium.
- Serum potassium levels do not correlate well with intracellular potassium levels and may not correlate with total body potassium

- It has been estimated that for every 0.3- meq/L decrease in serum potassium concentration, the total body potassium deficit is approximately 100 meq.
- Critically ill patients may have underlying conditions or receive medications that can cause hypokalemia.
- Metabolic alkalosis causes an intracellular shift of potassium.
- Other common causes of hypokalemia due to intracellular shifts of potassium include β-adrenergic agonists (e.g., albuterol), insulin, theophylline, and caffeine.

- Common causes of hypokalemia due to potassium losses include:

- potassium-wasting diuretics (loop and thiazide), sodium polystyrene sulfonate, corticosteroids (especially mineralocorticoids such as fludrocortisone), aminoglycosides, amphotericin B, magnesium depletion, renal replacement therapies (e.g., hemodialysis, continuous renal replacement therapy [CRRT]), and GI losses (e.g., diarrhea, nasogastric suctioning)

- *Treatment Hypokalemia*:
- Goals of therapy for hypokalemia include avoidance or resolution of symptoms, returning the serum potassium concentration to the normal range (i.e., 3.5–5 meq/L), and avoiding hyperkalemia.
- I.V. potassium supplementation is reserved for the treatment of severe hypokalemia, symptomatic hypokalemia, or when the GI tract cannot be used, and ICU patients may often have one or more of these conditions. The most commonly used oral or i.v. supplement is potassium chloride.
- Oral potassium supplements are available as chloride, bicarbonate, citrate, gluconate, and phosphate salts.

IV POTASSIUM

- Iv route carries high risk for hyperkalemia
- Reserved only for severe symptomatic hypokalemia or for the patients who can't take oral feeds.
- Always monitor IV therapy with cont EC monitoring and frequent K measurements
- Avoid IV till U/O is established
- **Don't Give**
- **> 10-20 mEqL/hr**
- **> 40 mEq/Litre**
- **>240 mEq/day**

- DKA and non ketotic hyperosmolar hypergylcemia are the commenest indication for IV potassium therapy.
- 100 mEq of K+ mixed in 1 litre of isotonic saline at rate of 100 ml/hour (25 macro or 100 micro drops) will deliver 10 mEq KCl per hour.
- **IV potassium max rate of infusion: Central line 60 mEq/L and peripheral line 40 mEq/L.**
- > 40 mEq/L can cause thrombophlebitis
- Avg rise in S. K+ level ins 0.25 mEq/L when 20 mEq/l given in one hour.
- As soon as cardiac rhythm returns to normal or respiratory muscle strength is restored to normal; IV potassium drip is to be tapered and switch to oral potassium therapy.

ORAL K+ SALTS:

- Oral salts are safer as having minimal risk of hyperkalemia
- Mild to mod hypo K+ (3 to 3.5 mEq/L): avg dose is 20 mEq 3 to 4 times a day along with treatment of underlying disorder
- Potassium chloride solution contains 20 mEq per 15 ml solution.
- KCl Tab contains 8 mEq per tab.
- May cause frequent GI Irritation; so advised to take solution with proper dilution with water and after food
- Oesophageal or small bowel erosion or stricture are uncommon side effects.

محصولات تجاری پتاسیم کلرید در ایران

.Kwality Pharmaceuticals Ltd

- پتاسیم کلراید قرص پیوسته رهش خوراکی ۶۰۰ mg (قرص پیوسته رهش پتاسیم کلراید)

.Astellas Pharma S.P.A

- پتاسیم کلراید ریتارد قرص پیوسته رهش خوراکی ۶۰۰ mg (قرص پیوسته رهش پتاسیم کلراید)

Amino Ag

- پلاس کالیم ریتارد قرص پیوسته رهش خوراکی ۶۰۰ mg (قرص پیوسته رهش پتاسیم کلراید)

Apotex

- آپو-کا قرص پیوسته رهش خوراکی ۶۰۰ mg (قرص پیوسته رهش پتاسیم کلراید)

Euro-Pharm International Canada Inc

- یورو کی قرص پیوسته رهش خوراکی ۶۰۰ mg (قرص پیوسته رهش پتاسیم کلراید)

.Novartis Pharmaceuticals Australia Pty Ltd

- اسلو کی قرص پیوسته رهش خوراکی ۶۰۰ mg (قرص پیوسته رهش پتاسیم کلراید)

- An oral potassium dosage of 20 meq daily has been suggested to prevent hypokalemia in patients receiving chronic diuretic therapy.
- Patients in the ICU who receive higher or more frequent doses of potassium-wasting diuretic may require i.v. therapy with higher doses.
- An alternative therapy to potassium supplementation in correcting or preventing hypokalemia is the use of potassium-sparing diuretics (e.g., spironolactone, triamterene, amiloride), which are especially useful in patients receiving drugs known to deplete potassium.

- Magnesium is important in the regulation of intracellular potassium.
- Hypomagnesemia may result in refractory hypokalemia, likely due to accelerated renal potassium loss or impairment of Na/K ATPase activity.
- Serum potassium levels should be monitored frequently (every 1–6 hours) in patients with severe hypokalemia if symptoms are present or if aggressive i.v. treatment is ongoing. Monitr after i.v. repletion in ICU patients with mild to moderate hypokalemia (within 2–8 hours) may be appropriate, in addition to routine monitoring (e.g., every 24–48 hours)

- ***Hyperkalemia***:

- Hyperkalemia (serum potassium concentration of >5.5 meq/L) can become life threatening when the serum potassium concentration exceeds 6.5 meq/L.

- Signs and symptoms of hyperkalemia include :

- muscle twitching, cramping, weakness, ascending paralysis, ECG changes (e.g., tall peaked T-waves, prolonged PR-interval, widened QRS complex, shortened QT-interval) and arrhythmias (e.g., bradyarrhythmias, ventricular fibrillation, asystole).

- True hyperkalemia can develop due to:
- extracellular shifts of potassium, increased potassium ingestion, or impaired potassium elimination
- . Pseudohyperkalemia can be an artifact of hemolysis from a traumatic blood draw or when the blood sample is contaminated with infused potassium.
- Hyperkalemia is most commonly seen in the setting of renal insufficiency, and acute renal failure is frequently seen in ICU patients.

- Potassium-sparing diuretics, angiotensin-converting-enzyme inhibitors, nonsteroidal antiinflammatory drugs, and hypoaldosteronism can impair potassium excretion and cause hyperkalemia.
- Metabolic acidosis results in an extracellular shift of potassium, without changes in total body potassium. Correction of the underlying metabolic acidosis redistributes potassium into the intracellular space and corrects the hyperkalemia.
- Other causes of extracellular shifts of potassium include succinylcholine, β-adrenergic blockers, digoxin overdose, and muscular injury (e.g., trauma, rhabdomyolysis).

- The goals of the hyperkalemia therapy are to antagonize the cardiac effects of potassium, reverse symptoms (if present), and return serum potassium to normal while avoiding overcorrection.
- All sources of exogenous potassium should be discontinued, and potassium-sparing diuretics and other medications that can cause hyperkalemia should be stopped, or the dosages decreased, if feasible.

Treatments for Hyperkalemia[19,20,23]

Treatment	Dose	Route	Time to Onset	Duration of Effect	Mechanism of Action and Effects
Calcium gluconate[a,b]	1–2 g (4.56–9.12 meq cacium)	I.V. over 5–10 min	1–2 min	10–30 min	Antagonizes cardiac conduction abnormalities
Sodium bicarbonate[a]	50–100 meq	I.V. over 2–5 min	30 min	2–6 hr	Increases serum pH; redistributes potassium into cells
Insulin (regular)[a] (with dextrose)	5–10 units	I.V. with 50 mL of 50% dextrose injection	15–45 min	2–6 hr	Redistributes potassium into cells
50% dextrose	50 mL (25 g)	I.V. over 5 min	30 min	2–6 hr	Increases insulin release; redistributes potassium into cells; prevents hypoglycemia when insulin is given
10% dextrose	1000 mL (100 g)	I.V. over 1–2 hr	30 min	2–6 hr	Increases insulin release; redistributes potassium into cells; prevents hypoglycemia when given with insulin
Furosemide	20–40 mg	I.V.	5–15 min	4–6 hr	Increases renal potassium loss
Sodium polystyrene sulfonate[c]	15–60 g	Oral or rectal	1 hr	4–6 hr	Resin exchanges sodium for potassium; increases fecal potassium elimination
Albuterol	10–20 mg	Nebulized over 10 min	30 min	1–2 hr	Stimulates sodium–potassium pump; redistributes potassium into cells
Hemodialysis	2–4 hr	NA[d]	Immediate	Variable	Removes potassium from plasma

[a]First-line therapies in hyperkalemic emergencies.

[b]Repeat dose in five minutes if abnormal electrocardiogram persists. Calcium chloride may also be used, but calcium gluconate is preferred over calcium chloride for peripheral venous administration because it causes less venous irritation. Calcium chloride (1000 mg = 13.6 meq calcium) provides three times more calcium than calcium gluconate (1 g = 4.56 meq calcium).

[c]Can be used to treat acute hyperkalemia, but the effects may not be seen for several hours. Removes 0.5–1 meq of potassium per 1 g of sodium polystyrene sulfonate.

[d]NA = not applicable.

- Sodium polystyrene sulfonate, a cation-exchange resin, binds potassium in the GI tract and eliminates it from the body.
- Necrosis of the GI tract has been reported in patients who received sodium polystyrene sulfonate in sorbitol. Sorbitol, used as a diluent to counteract constipation with sodium polystyrene sulfonate, is believed to be an important factor in inducing necrosis.
- Sodium polystyrene sulfonate in sorbitol should be used with extreme caution, or avoided, especially in uremic patients under stress or with critical illness patients with hypotension or decreased GI perfusion,and patients with GI obstruction or decreased GI motility , heart failure or severe hepatic disease

- Serum potassium should be monitored frequently throughout treatment (every 1–6 hours) in symptomatic patients with severe hyperkalemia, keeping in mind that many therapies for acute hyperkalemia will only redistribute potassium and not remove it from the body. Continued monitoring of serum potassium levels in patients with hyperkalemia after resolution of symptoms is recommended after therapeutic interventions every 4–12 hours until serum potassium levels return to normal.

- Routine monitoring of serum potassium levels (every 24–48 hours) is also recommended in adult patients in the ICU.

- Calcium:
- Calcium functions in bone metabolism, blood coagulation, platelet adhesion, neuromuscular activity, endocrine and exocrine secretory functions, and electrophysiology of the heart and smooth muscles.
- Serum calcium concentration is regulated by parathyroid hormone, vitamin D, and calcitonin.
- The normal range for total serum calcium concentration is 8.6–10.2 mg/d

- About 99% of total body calcium is found in bones, with less than 1% in the serum. Approximately 40– 50% of calcium in the blood is bound to plasma proteins, primarily albumin. Hypoalbuminemia, which is frequently seen in critically ill patients, can therefore cause a decrease in total serum calcium levels.

- For each 1-g/dL decrease in serum albumin concentration (below 4 g/dL), total serum calcium concentration decreases by approximately 0.8 mg/dL.

- Corrected serum calcium conc. = serum calcium conc. + (0.8 × [4 – serum albumin conc.])

- Ionized serum calcium is closely regulated by the endocrine system and is a better indicator of the functional status of calcium metabolism than total calcium levels.
- Because of the poor correlation between ionized calcium and total calcium levels, especially in ICU patients with hypoalbuminemia or acid–base imbalance, direct measurement of ionized calcium concentration in critically ill ICU patients is recommended.
- The normal range for ionized serum calcium concentration is 1.12–1.30 mmol/L.
- Metabolic alkalosis increases calcium binding to plasma proteins, reducing the serum level of ionized calcium. Conversely, metabolic acidosis decreases calcium binding to plasma proteins, increasing the serum concentration of ionized calcium.

Hypocalcemia:

Hypocalcemia (total serum calcium concentration of <8.6 mg/dL or ionized calcium concentration of <1.1mmol/l is primarily due to hypoalbuminemia.

Other causes include: hypomagnesemia, hyperphosphatemia, sepsis, pancreatitis, renal insufficiency, hypoparathyroidism, and administration of blood preserved with citrate.

The hallmark sign of severe acute hypocalcemia is tetany. Other neuromuscular, CNS, and cardiovascular symptoms may be present, even with mild to moderate hypocalcemia.

- I.V. calcium gluconate and calcium chloride are used to treat hypocalcemia when rapid correction of serum calcium levels is required.
- Calcium gluconate should be used as the preferred salt for routine calcium maintenance and supplementation; calcium chloride infusion may cause tissue necrosis if extravasation occurs. Calcium should not be infused in the same i.v. catheter as solutions containing phosphate because of the risk of calcium– phosphate precipitation.
- Asymptomatic hypocalcemia due to hypoalbuminemia requires no therapy.

- Severe hypocalcemia (total serum calcium concentration of<7.5mg/dl or ionized calcium concentration of <0.9mmol/l or acute symptomatic hypocalcemia requires prompt correction with i.v. calcium administration.
- Initially, 1000 mg of calcium chloride (13.6 meq of calcium) or 2 g of calcium gluconate (13.7 meq of calcium) may be given over 10 minutes to control symptoms .
- Since an i.v. bolus dose of calcium may only be effective for about two hours or less severe acute hypocalcemia may not be corrected with intermittent i.v. bolus doses.
- Instead, a continuous infusion of i.v. calcium may be required, with close monitoring of serum calcium levels at least every six hours during the infusion.
- The infusion rate should not exceed 0.8–1.5 meq/min because of the potential risk for cardiac arrhythmias associated with rapid calcium infusion

- Hypocalcemia due to citrated blood transfusion can be treated by administering 1.35 meq of calcium for each 100 mL of blood transfused.
- In addition to the monitoring described above, routine monitoring of serum calcium levels (total or ionized) in adult patients in the ICU is recommended (every 24–48 hours).
- Oral calcium supplements may be used once serum calcium levels are corrected by administration of i.v. calcium.

- Hypercalcemia:
- Hypercalcemia is defined as a total serum calcium concentration of >10.2 mg/dL and can be characterized as mild to moderate (total serum calcium concentration of 10.3–12.9 mg/dL) or severe (total serum calcium concentration of ≥13 mg/dL).
- The primary causes of hypercalcemia are malignancy (e.g., breast cancer, lung cancer, multiple myeloma, non-Hodgkin's lymphoma) and primary hyperparathyroidism.

Certain medications (e.g., thiazide diuretics, lithium), vitamin A toxicity, vitamin D toxicity

milk-alkali syndrome, adrenal insufficiency, immobilization, Paget's disease, rhabdomyolysis, and tuberculosis can also cause hypercalcemia

- Severe hypercalcemia may have cardiac manifestations, such as bradycardia or arrhythmias with ECG changes.
- Severe hypercalcemia or hypercalcemic crisis with a total serum calcium concentration of >13 mg/dL requires emergency treatment because it can lead to acute renal failure, obtundation, ventricular arrhythmias, coma, and death.

- Treatment Hypercalcemia:

- should begin promptly, starting with i.v. hydration using 0.9% sodium chloride infusion at 200–300 mL/hr to reverse the intravascular volume contraction caused by hypercalcemia. After adequate hydration is achieved, furosemide 40–100 mg i.v. every one to four hours can be used to enhance renal calcium elimination and avoid fluid overload from the saline hydration, but caution should be taken to avoid further intravascular volume depletion.

- Saline hydration and furosemide can reduce serum calcium levels by about 2–3 mg/dL within the first 48 hours of treatment.

- Hemodialysis may be necessary in lifethreatening hypercalcemia or in patients with impaired renal function

- Bisphosphonates (e.g., etidronate, pamidronate, zoledronic acid) are potent inhibitors of bone resorption via action on osteoblast and osteoclast precursors and are frequently used for the treatment of hypercalcemia of malignancy.
- Etidronate disodium is effective when given intravenously at a dosage of 7.5 mg/kg/day over 2 hours for three to seven days.
- Pamidronate disodium is given as a single i.v. dose of 60–90 mg infused over 2–24 hours. The usual dose of zoledronic acid is a single i.v. dose of 4–8 mg infused over 15 minutes.
- serum calcium levels usually begins to decline within two days of the first bisphosphonate dose.

- ***Magnesium*** :

- Magnesium is the second most abundant intracellular cation. It is found primarily in bone, muscle, and soft tissue, with about 1% of the total body content in the extracellular fluid.

- The normal serum magnesium concentration ranges from 1.5 to 2.4 mg/dL.

- Magnesium serves as an important cofactor for numerous enzymes and in many biochemical reactions and is a required cofactor for reactions involving ATP.

- Magnesium is absorbed throughout the small intestine, with the majority of absorption occurring in the ileum and jejunum.
- Magnesium homeostasis is primarily handled by the kidneys, but GI function, parathyroid hormone, and plasma magnesium concentrations also play a major role.
- Several other factors can affect magnesium homeostasis, including:
- the patient's clinical condition, medications (e.g., loop diuretics, amphotericin B), and
- alcohol use.

- ***Hypomagnesemia:***

- Hypomagnesemia (serum magnesium concentration of <1.5mg/dl is frequently observed in critically ill patients and has been associated with increased mortality.

- Severe hypomagnesemia (serum magnesium concentration of <1mg/dl can result in ECG changes, arrhythmias (including torsades de pointes), seizures, coma, and even death. Hypomagnesemia may cause concomitant refractory hypokalemia and hypocalcemia.

- hypokalemia is likely a result of impaired activity of the Na/KATPase and hypocalcemia is likely due to impaired parathyroid release or activity.

- Causes of hypomagnesemia include :
- excessive GI losses, renal losses, surgery, trauma, infection or sepsis, burns, transfusion of blood preserved with citrate, starvation, malnutrition, alcoholism, and certain medications (e.g., thiazide and loop diuretics, aminoglycosides, amphotericin B, cisplatin, cyclosporine).
- Use of cardiac glycosides (e.g., digoxin) has also been associated with hypomagnesemia, possibly by enhancing magnesium excretion, and hypomagnesemia may potentiate digoxin toxicity (e.g., dysrhythmias)

- Only about 1% of magnesium stores are found in the extracellular space, and serum magnesium levels may not correlate with intracellular concentrations or total body magnesium levels.
- Because hypomagnesemia is observed frequently in critically ill patients and has been associated with increased mortality,the serum magnesium concentrations in critically ill patients in the ICU should be kept at 1.5 mg/dL or higher.
- Patients who have recently had an acute myocardial infarction may require higher concentrations≥1.7mg/dl to prevent cardiac arrhythmias.

- The goals of therapy should be to avoid or resolve symptoms, return the serum magnesium concentration to 1.5–2.4 mg/dL, and avoid hypermagnesemia.
- Oral magnesium supplements are available; however, problems with administration, slow onset of action, and GI intolerance may limit their utility.
- The i.v. route of administration is preferred, especially in critically ill patients with severe symptomatic hypomagnesemia. Few i.v. dosing regimens for the treatment and repletion of hypomagnesemia have been suggested.

- Magnesium distributes into tissues slowly, but renal elimination is rapid, with up to 50% of an i.v. dose of magnesium excreted in the urine.

- Therefore, infusion time is critical, and additional supplementation may be required after the initial dose, with total repletion taking several days.

- For patients with mild to moderate hypomagnesemia, 8–32 meq of magnesium should be given (up to 1.0 meq/kg).
- Severe hypomagnesemia should be treated with 32–64 meq of magnesium sium (up to 1.5 meq/kg).
- Doses of<6g of magnesium sulfate should be infused over 8–12 hours, with higher doses infused over 24 hours.

- We recommend administering 50% or less of the suggested empirical magnesium dose in patients with renal insufficiency to decrease the risk of hypermagnesemia.
- For i.v. administration, the magnesium sulfate concentration should be diluted to 20%
- (20 g/100 mL) or less before administration.
- Because a renal threshold for magnesium exists, with up to 50% of an i.v. dose being eliminated in the urine, i.v. magnesium sulfate should be administered at a maximum rate of
- 1 g/hr (8 meq of magnesium per hour) in asymptomatic patients with hypomagnesemia, and the total dose should not exceed 12 g (100 meq of magnesium) over 12 hours.

- Serum magnesium levels should be monitored at least once daily during magnesium repletion, in addition to routine monitoring (every 24–48 hours)
- Because of the slow equilibration of magnesium between serum and intracellular spaces and tissues (e.g., bone, red blood cells, muscle),the serum magnesium levels could appear artificially high if measured too soon after a dose is given.

Empirical Treatment of Hypomagnesemia[42,45,166-173]

Severity	Serum Magnesium Concentration (mg/dL)	I.V. Magnesium Replacement Dose[a,b]
Mild to moderate	1.0–1.5	8–32 meq magnesium (1–4 g magnesium sulfate), up to 1.0 meq/kg
Severe	<1.0	32–64 meq magnesium (4–8 g magnesium sulfate), up to 1.5 meq/kg

[a]In patients with normal renal function; patients with renal insufficiency should receive ≤50% of the initial empirical dose. Maximum rate of infusion = 8 meq magnesium per hour (1 g magnesium sulfate per hour), up to 100 meq magnesium (approximately 12 g magnesium sulfate) over 12 hours if asymptomatic; up to 32 meq magnesium (4 g magnesium sulfate) over 4–5 minutes in severe symptomatic hypomagnesemia. 1 g magnesium sulfate = 8.1 meq magnesium.

[b]The authors suggest using adjusted body weight (AdjBW) in patients who are significantly obese (weight of >130% of ideal body weight [IBW] or have a body mass index of ≥30 kg/m^2): AdjBW (men) = ([wt (kg) – IBW (kg)] × 0.3) + IBW; AdjBW (women) = ([wt (kg) – IBW (kg)] × 0.25) + IBW.

- ***Hypermagnesemia***:
- Hypermagnesemia is defined as a serum magnesium concentration of>2.7mg/dl .
- Generally, patients tolerate mild hypermagnesemia (serum magnesium concentration of 2.5–4 mg/dL) and are usually asymptomatic.
- Patients with moderate hypermagnesemia (4–12.5 mg/dL) may exhibit signs and symptoms, including nausea, vomiting, loss of deep tendon reflexes, hypotension, bradycardia, and ECG changes (e.g., increased PR interval, increased QRS interval duration).

- Severe hypermagnesemia (>12.5–32 mg/dL) can result in respiratory paralysis, refractory hypotension, atrioventricular block, cardiac arrest, and death.
- The most common causes of hypermagnesemia are renal insufficiency and iatrogenic cause.
- Magnesium-containing antacids and other medications containing magnesium should be avoided in patients with renal insufficiency. Serum magnesium levels should be monitored at least once daily during treatment.

- Treatment: The primary therapy for hypermagnesemia is to discontinue exogenous magnesium administration.
- I.V. calcium should be administered to patients with severe symptomatic hypermagnesemia to reverse the cardiovascular and neuromuscular effects.
- 1–3 g of i.v. calcium gluconate (4.56–13.7 meq of calcium) should be infused over 3–10 minutes.
- Patients with asymptomatic hypermagnesemia may be treated with magnesium restriction, loop diuretics, or hemodialysis.

- Serum magnesium levels should be monitored at least once daily during treatment.
- More frequent monitoring may be required in symptomatic patients when more aggressive therapy is used (e.g., loop diuretics, hemodialysis).
- Serum magnesium concentration should be maintained in the normal range (1.5–2.4 mg/dL) and hypomagnesemia avoided during treatment.


خسته نباشید