Hypomagnesemia treatment in critically ill patients

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Introduction - Magnesium

- The fourth most abundant cation in the human body

  The second most abundant intracellular cation

- Healthy adult have a content of about 25 g (1000 mmol) Mg
- Stores:

  60% in bones, 20% in muscles, 20% in soft tissues, 0.5% in RBCs,

  0.3% in serum

- Healthy individuals need to ingest about 0.15–0.2 mmol/kg/day for normal Mg status.

Hypomagnesemia in critically ill patients, Hansen and Bruserud Journal of Intensive Care. 2018

- Normal serum concentration of Mg is 1.6 to 1.9 mEq/L

- Epidemiology studies :

  Hypomagnesemia was recorded (50%–60%) as more common among critically ill patients and they seem to develop during their intensive care unit (ICU) stay.

An assessment of serum magnesium levels in critically ill patients: A prospective observational study. Int J Crit Illn Inj Sci. 2023

Review > Shock. 2017 Mar;47(3):288-295. doi: 10.1097/SHK.0000000000000769.

Does Hypomagnesemia Impact on the Outcome of Patients Admitted to the Intensive Care Unit? A Systematic Review and Meta-Analysis

Pan Jiang [1], Qiurong Lv, Tianwen Lai, Feng Xu

Affiliations + expand

PMID: 27753792 DOI: 10.1097/SHK.0000000

A comprehensive search for clinical trials was performed, and 10 studies comprising 1,122 cases and 630 controls. hypomagnesemia appears associated with greater risk of mortality, sepsis, mechanical ventilation, and the length of ICU stay in patients admitted to ICU

Abstract

Hypomagnesemia is commonly seen but frequ
unit (ICU). However, the strength and consiste
critically ill patients remain controversial. In th
analysis to evaluate the association of serum magnesium level with prognosis of critically ill patients upon admission to the ICU. A comprehensive search for clinical trials was performed, and 10 studies comprising 1,122 cases and 630 controls were finally selected for analysis. The patients with hypomagnesemia had higher mortality rate (risk ratio [RR] 1.76; 95% confidence interval [CI] 1.54-2.00; P <0.00001), more frequently had sepsis (RR 2.04; 95% CI 1.21-3.42; P = 0.0007) and more frequent need for ventilatory support (RR 1.36; 95% CI 1.21 to 1.53; P <0.00001). Length of ICU stay was also higher in the hypomagnesemia group (RR 1.85; 95% CI 0.43- 3.26; P = 0.01). Collectively, our data indicated that hypomagnesemia appears associated with greater risk of mortality, sepsis, mechanical ventilation, and the length of ICU stay in patients admitted to ICU. The role of magnesium therapy for improving outcomes in critically ill patients is needed to further study.

› Int J Crit Illn Inj Sci. 2023 Jul-Sep;13(3):111-117. doi: 10.4103/ijciis.ijciis_11_23. Epub 2023 Sep 21.

An assessment of serum magnesium levels in critically ill patients: A prospective observational study

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Affiliations + expand

PMID: 38023578 PMCID: P

Results: One hundred participants were enrolled, of which 40% were between the age group of 46 and 65 years and 71% were males. Among all participants with hypomagnesemia, 52% were diabetic, 19% had a history of alcohol use disorder, and 27% had normal calcium and potassium levels. Hypomagnesemia significantly correlated with a longer duration of ICU stay among participants.

Conclusion: A significant correlation was observed between hypomagnesemia and increased ICU length of stay and mortality but not the duration of mechanical ventilation. Monitoring and appropriate supplementation of serum magnesium is recommended to limit further comorbidity and mortality in the critical care setting.

vaccines

MDPI

Review

Role of Magnesium in the Intensive Care Unit and Immunomodulation: A Literature Review

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Abstract: Both the role and the importance of magnesium in clinical practice have grown considerably in recent years. Emerging evidence suggests an association between loss of magnesium homeostasis and increased mortality in the critical care setting. The underlying mechanism is still unclear, but an increasing number of in vivo and in vitro studies on magnesium's immunomodulating capabilities may shed some light on the matter. This review aims to discuss the evidence behind magnesium homeostasis in critically ill patients, and its link with intensive care unit mortality via a likely magnesium-induced dysregulation of the immune response. The underlying pathogenetic mechanisms, and their implications for clinical outcomes, are discussed. The available evidence strongly supports the crucial role of magnesium in immune system regulation and inflammatory response. The loss of magnesium homeostasis has been associated with an elevated risk of bacterial infections, exacerbated sepsis progression, and detrimental effects on the cardiac, respiratory, neurological, and renal systems, ultimately leading to increased mortality. However, magnesium supplementation has been shown to be beneficial in these conditions, highlighting the importance of maintaining adequate magnesium levels in the intensive care setting.

Keywords: magnesium; critical care; immunomodulation; infections

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Hypomagnesemia associated with an elevated risk of bacterial infections, exacerbated sepsis progression, and detrimental effects on the cardiac, respiratory, neurological, and renal systems, ultimately leading to increased mortality.

Magnesium supplementation has been shown to be beneficial in these conditions, highlighting the importance of maintaining adequate magnesium levels in the intensive care setting.

The impact of correcting magnesium levels on the risk of developing acute respiratory failure requiring mechanical ventilation remains unknown, and warrants further investigation.

Magnesium plays an important role in protecting the kidney from AKI.

reducing the risk of progression to AKI requiring dialysis.

The current literature suggests there are benefits of maintaining normal magnesemia in critically ill patients, such as reducing overall mortality, modulating the inflammatory response, and protecting the kidney.

Role of Magnesium in the Intensive Care Unit and Immunomodulation: A Literature Review. Vaccines (Basel). 2023 Jun

Table 1 Differential diagnosis of Mg deficiency in the ICU setting

Gastrointestinal disorders
Prolonged nasogastric suction
Malabsorption syndromes
Extensive bowel resection
Acute and chronic diarrhea
Intestinal and biliary fistulae
Protein-calorie malnutrition (parenteral nutrition, anorexia, refeeding syndrome)
Acute hemorrhagic pancreatitis
Primary intestinal hypomagnesemia (neonatal)
Renal loss
Chronic parenteral fluid therapy
Osmotic diuresis (glucose, mannitol, urea)
Hypercalcemia
Alcohol
Drugs (see Table 2)
Metabolic acidosis (starvation, ketoacidosis, alcoholism)
Renal diseases
Chronic pyelonephritis, interstitial nephritis, and glomerulonephritis
Diuretic phase of acute tubular necrosis
Postobstructive nephropathy
Renal tubular acidosis
Post-renal transplantation
Primary renal hypomagnesemia

Mg deficiency in critically ill patients is mainly caused by gastrointestinal and/or renal disorders and may lead to secondary hypokalemia and hypocalcemia, and severe neuromuscular and cardiovascular clinical manifestations.

Hypomagnesemia in critically ill patients, Hansen and Bruserud Journal of Intensive Care. 2018

Table 2 Drugs associated with Mg deficiency and hypomagnesemia

Drugs	Mechanisms causing Mg deficiency
	Renal loss
Diuretics	
Loop	Increased renal Mg excretion by affecting the transepithelial voltage and inhibiting passive absorption.
Thiazides	Enhance Mg entry into the cells in the distal convoluted tubule.
Antimicrobial	
Amphotericin B Aminoglycosides Capreomycin Pentamidine	Renal urinary Mg wasting caused by nephrotoxins may be part of tubular necrosis and acute renal failure. Notably, impairment in Mg reabsorption in the loop of Henle and distal tubules may occur before the onset and may persist after the resolution of renal damage.
Chemotherapy	
Cisplatin	Renal urinary Mg wasting caused by nephrotoxins may be part of tubular necrosis and acute renal failure. Cisplatin treatment is also associated with lowered intestinal absorption
Immunosuppressive	
Calcineurin inhibitors	Urinary Mg wasting due to a downregulation of the Mg^{2+} transport proteins (TRPM6) in the loop of Henle and distal convoluted tubules.
Epidermal growth factor receptor inhibitors	
Cetuximab Panitumumab Matuzumab	Urinary Mg wasting due to a downregulation of the TRPM6 in the loop of Henle and distal convoluted tubules.
	Gastrointestinal loss
Proton-pump inhibitor	Impairing the intestinal Mg absorption by inhibiting Mg transporters (TRPM6 and TRPM7).
	Miscellaneous
Foscarnet	A general potent chelator of divalent cations which therefore has the potential to reduce ionized levels of Mg.
Cardiac glycosides	Mg deficiency is associated with cardiac glycosides. The exact mechanisms are not known.

Hypomagnesemia in critically ill patients, Hansen and Bruserud Journal of Intensive Care. 2018

Evaluation of Hypomagnesemia:

$$FEMg = \frac{UMg \times PCr}{(0.7 \times PMg) \times UCr} \times 100 \text{ percent}$$

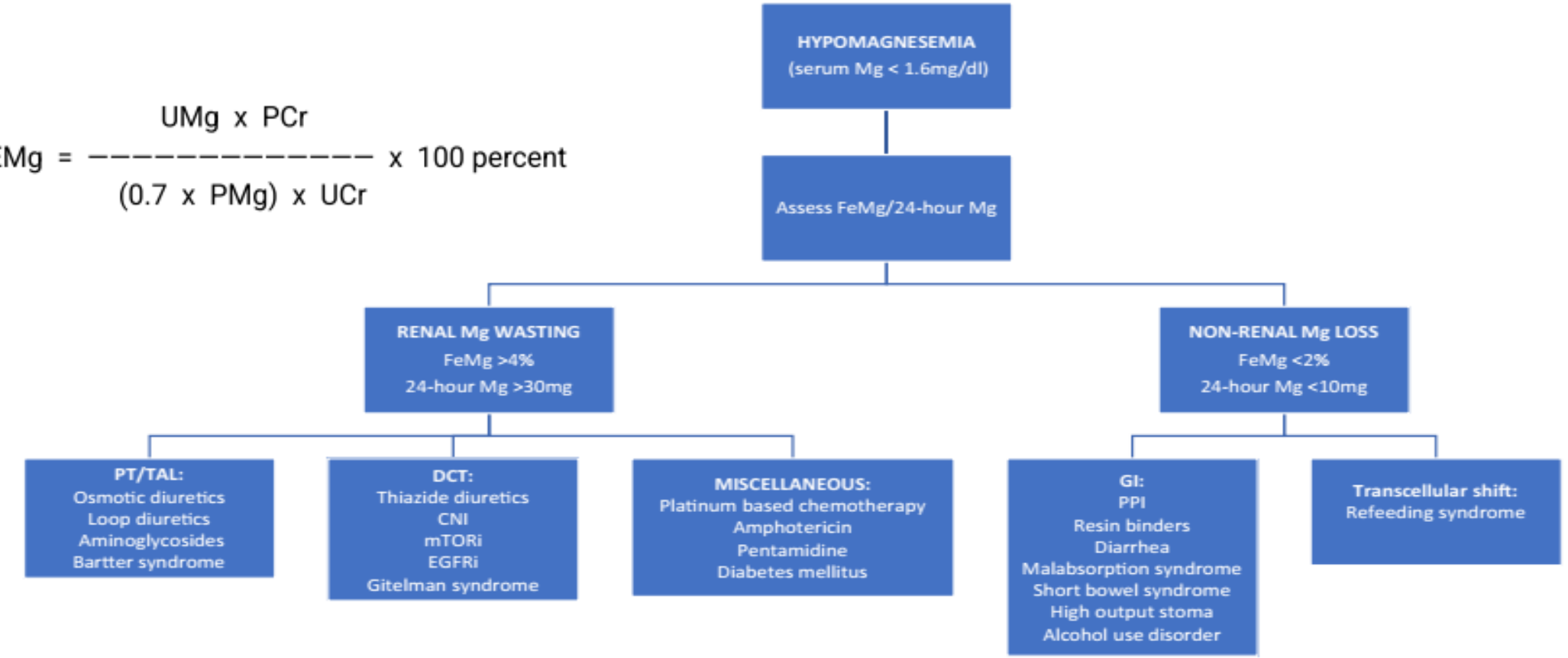

Figure 4. Algorithm for the evaluation of hypomagnesemia. Abbreviations: CNI, calcineurin inhibitor; DCT, distal convoluted tubule; EGFRi, epidermal growth factor receptor inhibitor; FeMg, fractional excretion of magnesium; GI, gastrointestinal; mTORi, mammalian target of rapamycin inhibitor; PPI, proton pump inhibitor; PT, proximal tubule; TAL, thick ascending limb of the loop of Henle.

Magnesium Disorders: Core Curriculum 2024. AJKD

Table 3 Clinical and biochemical effects of moderate to severe Mg deficiency and hypomagnesemia

- Biochemical
  - Hypokalemia
  - Renal K wasting
  - Decreased intracellular K
  - Hypocalcemia
  - Impaired parathyroid hormone secretion
  - Renal and skeletal resistance to parathyroid hormone
  - Resistance to vitamin D
- Neuromuscular
  - Tetany
  - Spontaneous carpal-pedal spasm
  - Seizures
  - Vertigo, ataxia, nystagmus, athetoid, and choreiform movements
  - Muscular weakness, tremor, fasciculation, and wasting
  - Psychiatric: depression, psychosis
- Cardiovascular
  - Dysrhythmias
  - Ventricular tachycardia (torsade de pointes
  - Atrial fibrillation
  - Supraventricular tachycardia
  - Hypertension
  - Vasospasm
  - Electrocardiographic changes
    - Prolonged QT interval
    - Prolonged PR interval
    - Wide QRS
    - Peaked T waves
    - ST depression
- Others
  - Acute myocardial infarction
  - Acute cerebral ischemia
  - Asthma exacerbation
  - Preeclampsia

Hypomagnesemia in critically ill patients, Hansen and Bruserud Journal of Intensive Care. 2018

Magnesium therapy:

- Serum Mg not necessarily reflects the total body Mg status.
- The total body deficit in patients with hypomagnesemia ranges from 0.5 to 1.0 mmol/kg (1-2 meq/kg).
- Patients at risk of magnesium deficiency with

  symptoms consistent with hypomagnesaemia

  refractory hypokalemia or unexplained hypocalcemia

  Considered for treatment even with NL serum Mg

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Hypomagnesaemia treatment. uptodate.2025
Magnesium Disorders: Core Curriculum 2024. AJKD

Magnesium load test in Normomagnesemic magnesium depletion

- One suggested method (uncertain utility):

  reduced excretion (<80 % over 24h) of an infused magnesium load (2.4 mg/kg over 4h).

- Positive test : malnutrition, cirrhosis, diarrhea, or long-term diuretic use

  +/_ signs or symptoms referable to magnesium depletion.

Administer magnesium

in unexplained hypocalcemia and/or hypokalemia.

Hypomagnesaemia treatment. uptodate.2025

How to treat:

- An assessment of the severity of symptoms
- Determination of the etiology

therapeutic approach to hypomagnesemia

- Plasma magnesium is >1 mg/dL: Mostly asymptomatic

Severity of symptoms:

- Mild hypomagnesemia with no or only mild symptoms can be treated with oral supplement.
- Parenteral Mg supplementation is indicated if :

  Mg concentration is < 1 mg/dL

  Patient presents with significant symptoms (tetany, arrhythmias, seizures)

Hypomagnesemia in critically ill patients, Hansen and Bruserud Journal of Intensive Care. 2018

- For critically ill patients with mild to moderate hypomagnesemia, empirically derived “rules of thumb” suggest that the administration of 1 g (8 mEq) of intravenous Mg will increase the serum Mg concentration by 0.15 mEq/L within 18 to 30 h.

Hypomagnesemia in critically ill patients, Hansen and Bruserud Journal of Intensive Care. 2018

Patients with Severe symptoms (tetany, arrhythmias, or seizures):

- Hemodynamically unstable (torsade de pointes arrhythmias or hypomagnesemic hypokalemia):

  1- 2 g of magnesium sulfate (8-16 mEq [4- 8 mmol]) given initially over 2 -15 min

  remains hemodynamically unstable after initial bolus: repeat bolus/ continues infusion

- Hemodynamically stable severe symptomatic hypomagnesemia (less than or equal to 1 mg/dL [0.4 mmol/L or 0.8 mEq/L]):

  1-2 g of magnesium sulfate in 50 to 100 mL of DW5 % can be given initially over 5-

  60 min/ followed by an infusion

Continues Infusion regimen for nonemergency repletion:

- 4-8 gr magnesium sulfate (32- 64 mEq [16-32 mmol]) given slowly over 12- 24h. This dose can be repeated as necessary to maintain the plasma magnesium concentration above 1 mg/dL (0.4 mmol/L or 0.8 mEq/L).
- In the normomagnesemic patient with hypocalcemia, it has been suggested to repeat this dose daily for three to five days.
- Severe hypomagnesemia may require treatment with doses until 1.5 mEq/kg.
- The slow distribution of Mg in tissues and the rapidly renal excretion makes the infusion time crucial.

Need for Mg infusion:

The plasma magnesium equilibrates slowly with intracellular sources,

With IV magnesium infusion, an abrupt temporary elevation in the plasma magnesium concentration partially inhibit the stimulus to magnesium reabsorption in the loop of Henle. Thus, up to 50% of the infused magnesium will be excreted in the urine.

Therefore adequate repletion requires sustained correction of the hypomagnesemia.

Patients with kidney function impairment:

- Patients with creatinine clearance less than 30 mL/min/1.73m are at risk for severe Hypermagnesemia if large doses of magnesium.
- Thus, we reduce the IV magnesium dose in such patients by 50% or more and closely monitoring magnesium concentrations.

Pregnant women with eclampsia

They routinely tolerate loading doses of 4-6 g (1gr= 8mEq= 4 mmol) administered over 20-30 min.

In the MAGPIE study , 32 mEq (4 g) Mg was initially given, followed by 8 mEq (1 g) per hour in women with preeclampsia.

Hypomagnesemia in critically ill patients, Hansen and Bruserud Journal of Intensive Care. 2018

Table 5 Treatment with Mg in specific clinical settings

Diagnose	Suggested Mg doses	Comments
Hemodynamically stable patients with severe symptomatic hypomagnesemia	1–2 g [8–16 mEq] (4–8 mmol) $MgSO_4$ given initially over 5–60 min followed by an infusion 4–8 g [32–64 mEq] (16–32 mmol) given slowly over 12–24 h.	–
Torsades de pointes	2 g [16 mEq] (8 mmol) over 2–15 min followed by a continuous infusion.	The rate of Mg infusion depends on the clinical situation. Rapid infusion is associated with hypotension and asystole.
Preeclampsia	4 g [32 mEq] (16 mmol) over 10–15 min followed by 1 g [8 mEq] (8 mmol) every following hours.	Evidence is conflicting and no consensus about the optimal Mg regimen exists. Suggested loading doses vary from 4 to 6 g (32–48 mEq; 16–24 mmol) and maintenance doses of 1–3 g (8–24 mEq; 4–12 mmol)/h.

Hypomagnesemia in critically ill patients, Hansen and Bruserud Journal of Intensive Care. 2018

Monitoring and follow up

- Plasma Mg concentration may be transiently elevated for a few hours after administration of an IV dose so the magnesium concentration should be measured 6 to 12 hours after each dose of IV magnesium.
- Repeat doses are given based upon the follow-up measurement.
- In nonemergency situations, administration IV Mg at a rate not exceeding 1 g/hour, resulting in more magnesium retention.

Points

- The evidence of using Mg as a routine in other critical conditions such as asthma or CABG is still insufficient.
- High levels of Mg (> 4–5 mg/dL) may give muscle weakness, reduced respiration, and in worst case cardiac arrest.
- In case of intolerable intoxication; intravenous calcium (100–200 mg over 5–10 min) should be administrated as it antagonizes the neuromuscular and cardiovascular effects of Mg.

Hypomagnesemia in critically ill patients, Hansen and Bruserud Journal of Intensive Care. 2018

Magnesium sulfate:

- IV use: Most widely available intravenous form of magnesium

1 g contains 4 mmol or 8mEq of magnesium

this is administered as a 20% formulation (also 50%)

- IM use: A 50% intramuscular formulation

available for pregnant women with eclampsia

- SC use : Subcutaneous delivery of magnesium sulfate has been used in intractable chronic hypomagnesemia.

- Topical use: topical application of magnesium to increase plasma magnesium has also been described but requires further rigorous evaluation

Magnesium Disorders: Core Curriculum 2024. AJKD

Attention to factors that promote magnesium loss:

- PPI
- Potassium binders: sodium polystyrene sulfonate, patiromer
- Medications associated with diarrhea and diuretics
- Transplant patients on Calcineurin inhibitors or cancer patients on EGFR inhibitors

If the culprit medications cannot be stopped and must be continued concurrently with treatment for hypomagnesemia

Magnesium Disorders: Core Curriculum 2024. AJKD

Patients with no or minimal symptoms:

- If available and tolerable, oral replacement should be given to the hypomagnesemic patient with no or minimal symptoms.
- Many hospitalized patients with hypomagnesemia are given IV rather than oral magnesium supplementation, even if symptoms are minimal or absent because of side effect of oral supplement like gastrointestinal discomfort and diarrhea.

Hypomagnesaemia treatment. uptodate.2025

Intravenous repletion in stable hospitalized patients

Dose dependent to severity of hypomagnesemia:

- Severe (<1 mg/dL),
- Moderate (1-1.5 mg/dL),
- Mild (1.6-1.9 mg/dL combined with suspicion of magnesium depletion)

- Plasma magnesium is less than 1 mg/dL (0.4 mmol/L or 0.8 mEq/L):

give 4-8 gr (32-64 mEq [16-32 mmol]) of magnesium sulfate over 12-24h

- Plasma magnesium is 1-1.5 mg/dL (0.4-0.6 mmol/L or 0.8-1.2 mEq/L):

give 2-4 gr (16-32 mEq [8-16 mmol]) of magnesium sulfate over 4-12 h.

- Plasma magnesium is 1.6-1.9 mg/dL (0.7-0.8 mmol/L or 1.4 -1.6 mEq/L):

give 1-2 gr (8 to 16 mEq [4 to 8 mmol]) of magnesium sulfate over 1-2 h.

Hypomagnesaemia treatment. uptodate.2025

Monitoring and Follow up:

- Plasma magnesium concentration should be measured daily or more frequently if indicated.
- Repeat doses are given based upon the follow-up measurement.
- It is advisable in patients with normal kidney function to continue magnesium repletion for at least one to two days after the serum magnesium concentration normalizes.

Hypomagnesaemia treatment. uptodate.2025

Oral preparation:

- A number of oral magnesium salts, differs in the content of elemental magnesium and bioavailability.
- Organic salts are more bioavailable compared with inorganic salts
- A typical daily dose in a patient with normal kidney function: 240 to 1000 mg (20 to 80 mEq [10 to 40 mmol]) of elemental magnesium in divided doses.

Inorganic Magnesium Salts	Organic Magnesium Salts	Combinations/Different Formulations
Carbonate	Acetate	Citrate + hydrogen-L-glutamate
Chloride	Aspartate	Dicitrate
Oxide	Citrate	Glycinate lysinate chelate
Sulfate	Gluconate	Oxide + glycerophosphate
	Lactate	Pyrrolidone carboxylic acid
	Pidolate	Trimagnesium dicitrate
		U-aspartate-hydrochloride-trihydrate

- Sustained-release preparations:

(Magnesium chloride and Magnesium L-lactate)

Slowly absorbed and minimize renal excretion

Use of lower doses

Minimizes the associated diarrhea

- Six to eight tablets (30 to 56 mEq) should be taken daily in divided doses for severe magnesium depletion. Two to four tablets (10 to 28 mEq [5 to 14 mmol]) for mild hypomagnesemia.

- Rapid-release preparation:

Magnesium oxide: 800 to 1600 mg (20 to 40 mmol [40 to 80 mEq]) daily in divided doses may be used for moderate to severe hypomagnesemia. Diarrhea frequently occurs

Magnesium citrate is more bioavailable compared with magnesium oxide based on measurements of plasma and urine magnesium.

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Table 1. Oral Formulations of Magnesium Supplements, Magnesium Content, and Typical Starting Dose

Magnesium Formulation	Elemental Magnesium/ Dose, mg	Typical Dosing
Magnesium oxide, 400 mg	241.3	1 tablet 2-3 times daily
Magnesium carbonate	54 mg/5 mL	5-10 mL 3 times daily
Magnesium chloride, 520 mg	62.17	1-2 tablets 3 times daily
Magnesium glycinate, 665 mg	120	1 tablet 2-3 times daily
Magnesium glycerophosphate, 425 mg	50	2 tablets 2-3 times daily
Magnesium lactate, 84 mg elemental magnesium	84	1 tablet 3 times daily
Magnesium phosphate, 710 mg	27.73	1 tablet 3 times daily
Magnesium aspartate hydrochloride, 685 mg	133	1 tablet 3 times daily
Magnesium gluconate, 500 mg	28.5	2 tablets 3 times daily
Magnesium citrate, 625 mg	100	1 capsule 3 times daily

A reasonable target dose is to administer 300 mg of elemental magnesium in divided doses and titrate as tolerated. In general, although the amount absorbed increases, bioavailability decreases with increasing doses. However, the risk of diarrhea increases with increased amounts of elemental magnesium.

Magnesium Disorders: Core Curriculum 2024. AJKD

Side effect:

Diarrhea is a major limiting factor to replacement with oral magnesium.

- Magnesium is an effective osmotic laxative
- The severity of diarrhea increases linearly with the magnesium content of stool. Some formulations such as magnesium hydroxide are primarily laxatives and should not be used at all to treat hypomagnesemia.
- Interaction:

  Oral magnesium salts reduce absorption of oral bisphosphonates

Magnesium Disorders: Core Curriculum 2024. AJKD

Guideline for the Management of Hypomagnesaemia in Adults.NHS.2024

Patients with impaired kidney function

- Patients with reduced kidney function may require magnesium repletion if they have severe hypomagnesemia.
- It is extremely unusual for a patient with no kidney function (on dialysis) to have severe magnesium depletion in the absence of an extra renal loss such as diarrhea.

  Treating the diarrhea may be sufficient to correct the hypomagnesemia.
- Here are no published data to guide therapy

Hypomagnesaemia treatment. uptodate.2025

Patient with GFR 15-30 mL/min/1.73 m and severe hypomagnesemia :

Symptomatic:

- 2-4 gr of IV magnesium sulfate given slowly over 4 to 12 hours.

Asymptomatic:

- One-half the dose of the selected oral preparation that is recommended for the patient with normal kidney function.

The plasma magnesium should be checked prior to subsequent doses and daily if doses are given less frequently.

Hypomagnesaemia treatment. uptodate.2025

Adjunct treatment:

- Potassium-sparing diuretic :

  Amiloride and Triamterene (In renal magnesium wasting)

  Increasing Mg reabsorption in the distal nephron

  Efficacy is limited

- Inulin supplementation

  Increase gut absorption of magnesium.

Magnesium Disorders: Core Curriculum 2024. AJKD

- SGLT2 inhibitors:

Correct hypomagnesemia and potentially be more effective than magnesium repletion.

Decrease the fractional excretion of magnesium

Might create a favorable electrical gradient for magnesium reabsorption in the PCT by decreasing the electrogenic absorption of sodium.

Magnesium Disorders: Core Curriculum 2024. AJKD

Gloucestershire Hospitals NHS
NHS Foundation Trust

Intravenous administration

Compatible infusion fluids:

- sodium chloride 0.9%
- glucose 5%

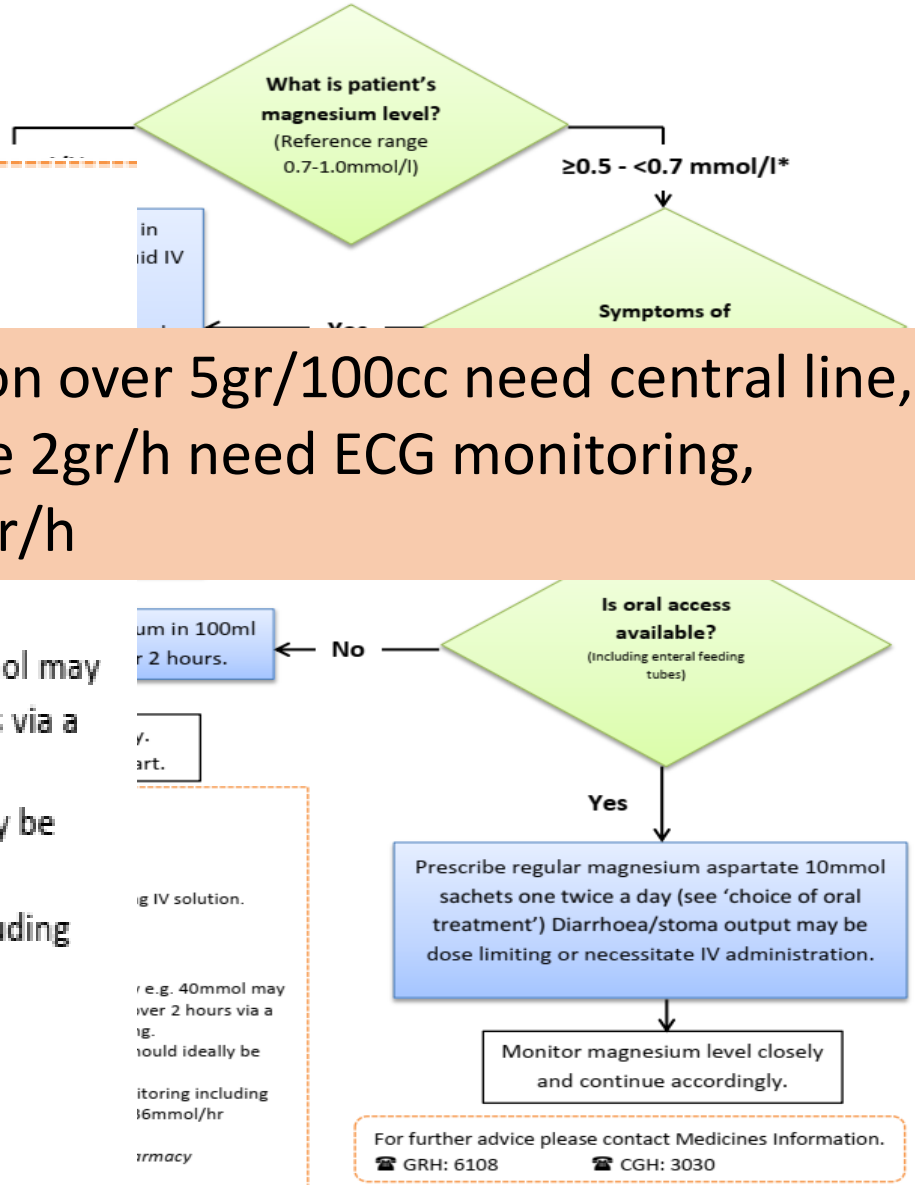
Magnesium sulphate 50% is use

- 1g (4mmol) in 2ml amps
- 5g (20mmol) in 10ml amp

Notes

- Higher doses may be given if necessary e.g. 40mmol may be given in 100ml of compatible fluid over 2 hours via a central line with appropriate monitoring.
- Concentrations over 20mmol/100ml should ideally be given via a central line – see Medusa
- With rates above 8mmol/hr close monitoring including ECG is recommended. Maximum rate 36mmol/hr
- See Medusa for further details.
- *For TPN patients: please liaise with pharmacy manufacturing or dietetics.*

Concentration over 5gr/100cc need central line,
infusion rate 2gr/h need ECG monitoring,
Max rate 9 gr/h



Conversion relationships:
1 mmol = 2 mEq = 24 mg elemental magnesium = 240 mg magnesium sulfate
Or
8meq=1 gr MgSo4

Review date: October 2024

THANK YOU