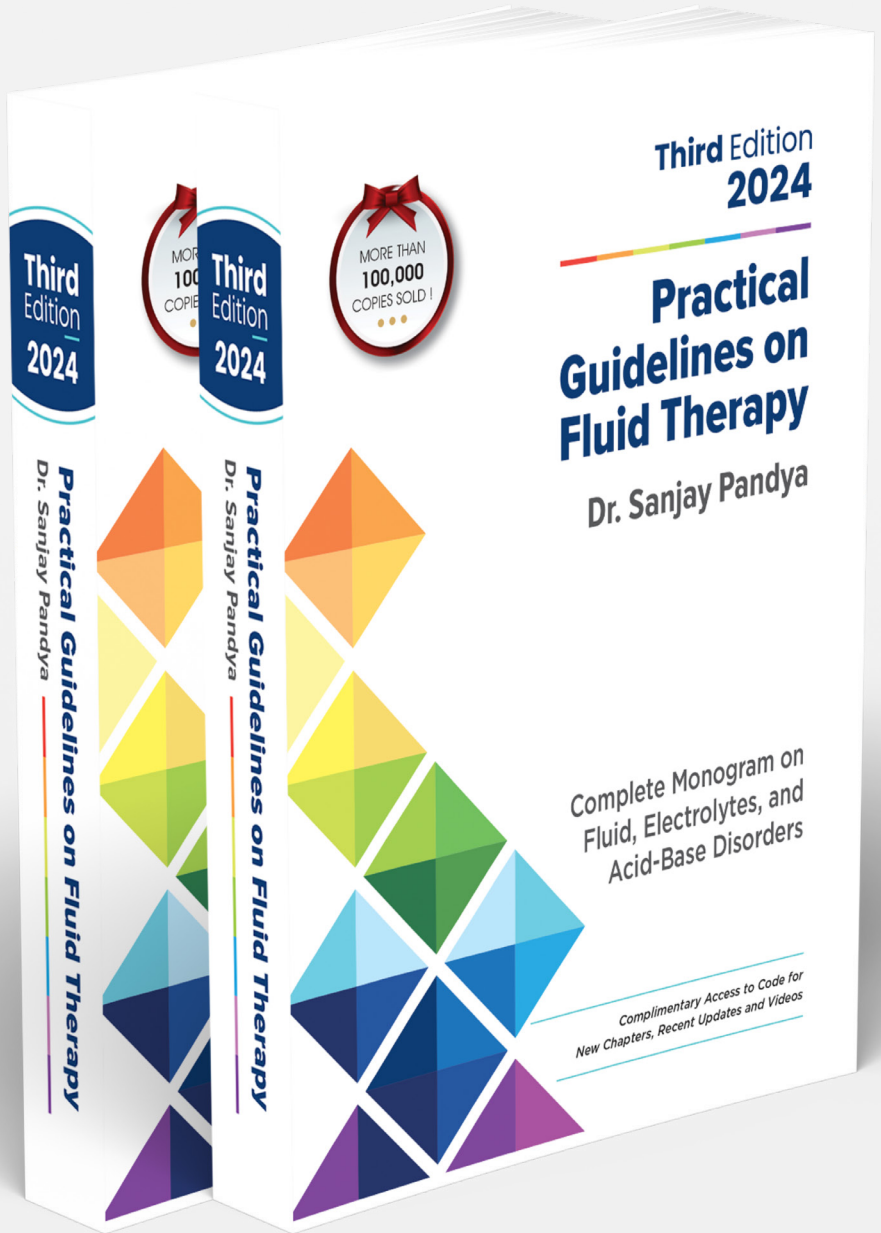
Fluid therapy.org

Chapter 29:
Hypermagnesemia



To get a copy of the book, visit: **www.fluidtherapy.org**

29 Hypermagnesemia

Hypermagnesemia is an uncommon electrolyte disorder defined as serum magnesium concentration above 2.6 mg/dL (1.1 mmol/L, or 2.14 mEq/L). As a normal kidney can effectively excrete magnesium load, hypermagnesemia is rarely seen in clinical practice if renal function is normal.

ETIOLOGY

Hypermagnesemia is frequently iatrogenic and almost always occurs either due to impaired excretion of magnesium in acute kidney injury (AKI) or chronic kidney disease (CKD) or the administration of magnesium in a large amount [1–4].

Common causes are:

- Renal failure: AKI or CKD patients receiving magnesium-containing antacids, laxatives, or IV fluids [3, 5, 6].
- Excessive magnesium intake: Treatment of preeclampsia or eclampsia with IV magnesium sulfate, aggressive treatment of hypomagnesemia with IV magnesium, intake of large amounts of magnesium salts as cathartics or antacids, and use of rectal magnesium sulfate enemas.
- Compartment shift or leak: Diabetic ketoacidosis untreated, tumor lysis syndrome, acute rhabdomyolysis, hemolysis, and severe burns.
- Miscellaneous causes: Milk-alkali syndrome and impaired renal excretion of magnesium due to primary hyperparathyroidism, adrenal insufficiency, and hypothyroidism.

CLINICAL FEATURES [7, 8]

Symptoms of hypermagnesemia are chiefly neuromuscular, cardiac, and related to hypocalcemia, which varies as per the magnesium level and, in severe form, can be serious and potentially fatal, as summarized in Table 29.1.

Neuromuscular manifestations: Hypermagnesemia inhibits acetylcholine release from the neuromuscular endplate, causing the blockage of neuromuscular transmission leading to a neuromuscular symptom. It includes muscular weakness, lethargy, loss of deep tendon jerks, muscular paresis leading to respiratory depression, respiratory failure, and quadriparesis. In addition, due to smooth-muscle paralysis, hypermagnesemia may present as paralytic ileus or urinary retention.

REFERENCES

1. Jahnen-Dechent W, Ketteler M. Magnesium basics. Clin Kidney J. 2012;5(Suppl 1):i3–i14.
2. Alaini A, Roldan CA, Servilla K, et al. Near death by milk of magnesia. BMJ Case Rep. 2017;2017:bcr2016218260.
3. Wakai E, Ikemura K, Sugimoto H, et al. Risk factors for the development of hypermagnesemia in patients prescribed magnesium oxide: a retrospective cohort study. J Pharm Health Care Sci. 2019;5:4.
4. Mori H, Suzuki H, Hirai Y, et al. Clinical features of hypermagnesemia in patients with functional constipation taking daily magnesium oxide. J Clin Biochem Nutr. 2019;65(1):76–81.
5. Wyskida K, Witkowicz J, Chudek J, et al. Daily magnesium intake and hypermagnesemia in hemodialysis patients with chronic kidney disease. J Ren Nutr 2012;22(1):19–26.
6. Khairi T, Amer S, Spitalewitz S, et al. Severe Symptomatic Hypermagnesemia Associated with Over-the-Counter Laxatives in a Patient with Renal Failure and Sigmoid Volvulus. Case Rep Nephrol. 2014;2014:560746.
7. Topf JM, Murray PT. Hypomagnesemia and hypermagnesemia. Rev Endocr Metab Disord. 2003;4(2):195–206.
8. Kraft MD, Btaiche IF, Sacks GS, et al. Treatment of electrolyte disorders in adult patients in the intensive care unit. Am J Health Syst Pharm. 2005;62(16):1663–82.
9. Mordes JP, Swartz R, Arky RA. Extreme hypermagnesemia as a cause of refractory hypotension. Ann Intern Med. 1975;83(5):657–658.
10. McDonnell NJ, Muchatuta NA, Paech MJ. Acute magnesium toxicity in an obstetric patient undergoing general anaesthesia for caesarean delivery. Int J Obstet Anesth. 2010;19(2):226–31.
11. Ishida Y, Tabuchi A. Severe Hypermagnesemia with Normal Renal Function Can Improve with Symptomatic Treatment. Case Rep Emerg Med. 2020;2020:2918249.