Chapter 31
Approach to Hypermagnesemia

31.1 Introduction

Magnesium is a vital electrolyte integral to over 300 enzymatic reactions essential for normal physiological functions. These include ATP metabolism, nucleic acid synthesis, muscle contraction, and nerve function. Its crucial role in cellular energy production and stabilization of cellular processes underscores the importance of maintaining appropriate magnesium levels. Despite its significance, magnesium imbalance, particularly hypermagnesemia, is relatively rare but can have severe clinical implications, especially in critically ill patients.

Hypermagnesemia is characterized by elevated serum magnesium levels exceeding 2.5 mg/dL. This condition can lead to profound cardiovascular, neuromuscular, and respiratory complications. Effective management in the intensive care unit (ICU) necessitates prompt identification, assessment of severity, and implementation of appropriate interventions to mitigate adverse outcomes [1, 2] [Ref: Algorithm 31.1].

31.2 Magnesium Homeostasis and Regulation

31.2.1 Renal and Intestinal Regulation

Magnesium balance is tightly regulated through intestinal absorption and renal excretion. The kidneys play a pivotal role in maintaining magnesium homeostasis by filtering and reabsorbing magnesium primarily in the thick ascending loop of Henle and the distal convoluted tubule. Approximately 70% of filtered magnesium is reabsorbed in the loop of Henle, with another 10% reabsorbed in the distal tubule. Any impairment in renal function can significantly affect this balance, leading to elevated serum magnesium levels [3].

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31.3 Etiology and Pathophysiology

31.3.1 Renal Failure

Renal failure is the predominant cause of hypermagnesemia. Since the kidneys are responsible for excreting the majority of absorbed magnesium, impaired renal function can lead to accumulation. Patients with chronic kidney disease (CKD) or acute kidney injury (AKI) are particularly susceptible due to decreased glomerular filtration rate (GFR) and impaired tubular reabsorption processes.

31.3.2 Iatrogenic Causes

Excessive intake of magnesium-containing medications is a significant iatrogenic contributor. Antacids and laxatives containing magnesium hydroxide or magnesium citrate can lead to elevated serum levels, especially in patients with compromised renal function. Intravenous magnesium therapy, often used in obstetric care for conditions like severe preeclampsia with premonitory signs and eclampsia, can also result in hypermagnesemia if not carefully monitored.

31.3.3 Clinical Manifestations

Hypermagnesemia presents with a spectrum of clinical manifestations that correlate with serum magnesium levels:

- Neuromuscular Symptoms: Lethargy, muscle weakness, hyporeflexia, and, in severe cases, flaccid paralysis.
- Cardiovascular Symptoms: Hypotension due to vasodilation, bradycardia, and arrhythmias. High levels can lead to complete heart block or cardiac arrest.
- Respiratory Symptoms: Depressed respiratory drive and apnea in extreme cases.
- Effect on Calcium Homeostasis: Elevated magnesium levels can suppress PTH secretion, leading to hypocalcemia. This hypocalcemia exacerbates neuromuscular irritability and cardiac dysfunction, adding complexity to the clinical picture [3].

Understanding these manifestations is crucial for timely diagnosis and intervention.

31.4 Diagnostic Approach

Clinical Evaluation

1. History and Risk Factor Assessment:
   - Dietary and Medication History: Evaluate intake of magnesium-rich foods, supplements, antacids, and laxatives.
   - Renal Function: Assess for history of CKD or AKI.
   - Medication Use: Review any recent intravenous magnesium administration.
2. Physical Examination:
   - Vital Signs: Check for hypotension and bradycardia.
   - Neuromuscular Assessment: Test deep tendon reflexes, muscle strength, and consciousness level.
   - Respiratory Evaluation: Monitor for signs of respiratory depression.

Laboratory Tests

1. Serum Magnesium Levels:
   - Interpretation: Levels exceeding 2.5 mg/dL confirm hypermagnesemia, but serum levels may not accurately reflect total body magnesium stores.
2. Urinary Magnesium Excretion Tests:
   - Purpose: Differentiate between renal and nonrenal causes.
   - Interpretation: Low urinary magnesium suggests impaired renal excretion, while high levels indicate excessive intake or increased absorption.
3. Electrolyte Panel:
   - Calcium and Potassium Levels: Check for hypocalcemia due to suppressed PTH secretion and other electrolyte imbalances.
4. Renal Function Tests:
   - Serum Creatinine and BUN: Elevated levels indicate reduced renal clearance.

Electrocardiogram (ECG)

- ECG Changes: Look for prolonged PR intervals, widened QRS complexes, peaked T waves, and arrhythmias. These changes help assess the cardiac impact of elevated magnesium levels.

31.5 Management Based on Severity

31.5.1 Mild Hypermagnesemia (2.5–4.0 mg/dL)

Interventions
- Discontinue Magnesium Sources: Immediately halt all magnesium-containing medications and dietary supplements.
- Promote Renal Excretion:
- Intravenous Fluids: Administer isotonic saline (0.9% NaCl) to enhance glomerular filtration and magnesium excretion.
- Monitoring:
- Serum Magnesium Levels: Check levels every 24 hours until normalization.
- Renal Function Tests: Monitor to ensure adequate excretion.

31.5.2 Moderate Hypermagnesemia (4.1–6.0 mg/dL)

Interventions
- Discontinue Magnesium Sources: Stop all exogenous magnesium intake.
- Enhance Excretion:
- Loop Diuretics: Administer furosemide (20–40 mg IV) to inhibit magnesium reabsorption in the loop of Henle.
- Monitoring:
- Cardiac Monitoring: Continuous ECG to detect arrhythmias.
- Neurological Assessment: Regular checks for changes in reflexes and muscle strength.

31.5.3 Severe Hypermagnesemia (>6.0 mg/dL)

Interventions
- Immediate Actions:
- Discontinue Magnesium Sources: Eliminate all sources promptly.
- Intravenous Calcium Gluconate: Administer 1–2 g IV over 5–10 min.

- Mechanism: Acts as a physiological antagonist to magnesium, stabilizing cardiac membranes and restoring neuromuscular function.
- Aggressive Management:
- Hydration: Provide isotonic saline to promote diuresis.
- Diuretics: Use higher doses of furosemide (40–80 mg IV) to facilitate magnesium excretion.

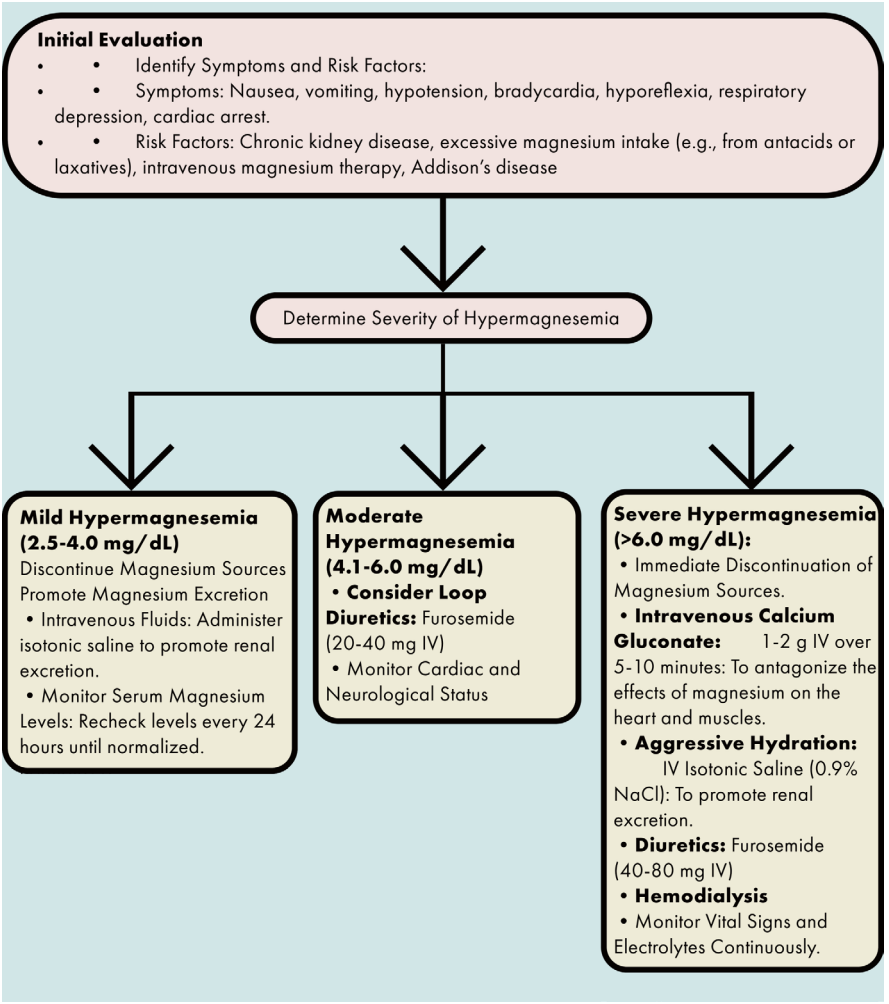
31.5.4 Renal Replacement Therapy

Hemodialysis
- Indications: Renal failure patients or refractory cases.
- Effectiveness: Rapidly reduces serum magnesium levels.
- Monitoring:
- Vital Signs: Continuous monitoring of blood pressure, heart rate, and respiratory status.
- Electrolytes: Frequent checks to manage potential shifts and prevent hypocalcemia.

31.6 Conclusion

Hypermagnesemia is a serious electrolyte disturbance that demands prompt recognition and management, especially in the ICU setting. Understanding the physiological importance of magnesium and the mechanisms regulating its balance is essential. Clinicians must be vigilant for signs of hypermagnesemia in patients with renal dysfunction or those receiving magnesium-containing treatments.

Algorithm 31.1: Approach to hypermagnesemia



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