Magnesium Mg^{++}

EMC 360 lecture 09

K pp. 88 - 98

C pp. 44-45 ; c pp 39-40

" Magnesium can be used to treat every disease known except hypermagnesemia "

- **C.M. Slovis**, MD

Objectives

Upon completion of this lecture the learner should be able to:

- Discuss the functions, physiology, and pathophysiology of magnesium .
- Discuss the unique properties of magnesium that account for its beneficial effects as an anti arrhythmic and as a smooth muscle relaxant.
- Discuss the clinical settings or risks for magnesium emergencies .
- Identify abnormal signs,symptoms, and clinical features of magnesium emergencies .

Priorities - review

- When fluids and electrolytes are altered, they should be corrected in the following order:
  - Volume
  - pH
  - Potassium, ***magnesium***, and calcium
  - Sodium and chloride

Magnesium physiology

- Distribution :
  - ECF: 1% (potassium-like)
  - ICF : the second most common intracellular cation
  - 1/3 is protein bound (calcium-like)
- Serum levels
  - 1.5 - 2.5 mEq/L [not useful]

Magnesium physiology

- Function :
  - Potassium-like
    - Muscular
    - Neuromuscular
    - Cardiac conduction
  - Sodium-like
    - Neurological
  - Cellular
    - Enzymatic
    - Cell wall function
- Excretion
  - Renal

Magnesium : Electrophysiologic Effects

Multiple beneficial electrophysiologic effects. These include:

- Increasing resting membrane negativity (phase 4)
- Calcium channel blockade (phase 2)
- Direct myocardial stabilization (not yet elucidated)

Magnesium : Electrophysiologic Effects

Thus:

- Magnesium stimulates the Na-K ATP-ase pump to make the cell more negative
- Decreases the relative refractory period by speeding repolarization and by decreasing the duration of phase 2

Magnesium for Torsades

Mechanism of Action

- Reverses underlying etiology
- Decreases Q-T interval
- Non Arrhythmogenic
- Rapidly effective
- Much safer and quicker than non-magnesium therapies for Torsades:

Mechanism for Digitalis OD

- Mg^{++} fixes a poisoned Na-K pump (digitalis toxicity) : leak of K out of the cell and Na into the cell

Mechanism in SVTs

- Mg block of AV conduction
- reduction of sympathetic influences on AV node
- altered slow channel calcium movement
- Hypokalemia correlates with hypomagnesemia
  - AF and MAT are associated with hypokalemia
  - Only recently we have begun to appreciate how important magnesium is and how often we miss magnesium deficiency:

"Magnesium - The Forgotten Electrolyte"

- "Hypomagnesemia is *the* most underdiagnosed electrolyte deficiency in current practice"
  - Geiderman et al JACEP 1979

Hypomagnesemia

- Common
- Often underdiagnosed in critically ill patients"

Hypomagnesemia

- Total Body Stores and Accurate Mg $^{++}$ Level
  - labs can not yet reliably do an ionized Mg ++ testing
  - Lab values cannot tell you the total body stores or intracellular magnesium status of a patient.
  - An important rule is:
    - FOR SYMPTOMATIC PATIENTS
    - in the setting of hypomagnesemia, DON'T WORRY ABOUT THE EVENTUAL TOTAL SERUM MAGNESIUM LEVELS determined after arrival in ED,
    - OR YOU WILL MISS CLINICALLY SIGNIFICANT HYPOMAGNESEMIA

The 5 Causes of Hypomagnesemia

- Dietary
- Gastrointestinal
- Renal losses (diuretics)
- Drug
- Endocrine-Metabolic

High Risk For Clinically Significant Hypomagnesemia

- Patients on diuretics
- Alcoholic or malnourished patients
- Patients with hypokalemia
- Patients with AMI
- Patients with ventricular arrhythmias

ECG Changes of Hypomagnesemia

- Acute (mild-moderate) Chronic (severe)
- tall peaked T-waves
- prolonged P-R interval *
- asymmetric T-waves
  widened
- QRS *
- Flat T-waves
  low
- T-waves

(often looks like HyperK) (often looks like HypoK and HypoCA)

Toxicity of Magnesium

- Hypermagnesemia (and Side Effects of rapid Magnesium Infusion)
- Flushing (common), diaphoresis, and drowsiness
- Hyporeflexia / loss of DTR's
- Hypotension and hypoventilation
- Conduction disturbances and arrhythmias
- Cardiac or respiratory arrest

Symptoms and levels do not correlate perfectly but loss of DTRs preceeds the more serious side effects

Clinical Effects of Hypermagnesemia

Effect	Mg Level (mg/dl)
Decreased DTR's	4-5
Hypotension	5-7
Respiratory Insufficiency	10
Heart Block	10-15
Respiratory Paralysis	15+
Cardiac Arrest	15-24

Hypermagnesemia

- Rare
- Usually introgenic or in chronic renal failure
- Beware Mg cathartics and antiacids (Maalox; MOM)
- Symptoms rare below 8-10 mg/dl

Magnesium in Alcohol Withdrawal

- Decreases potassium losses
- Total body Magnesium deficit is 10-20 grams

Magnesium in Eclampsia

- Its major benefit is that it blocks vasospasm:
  - Magnesium is a calcium blocker
  - Magnesium blocks vasospasm

  and

- Lowers incidence of seizures

Magnesium and Seizures

- Magnesium infusion corrects abnormal neuronal firing
- A change of 1 mEq/l dramatically suppresses EEG spikes

Magnesium in COPD +/or Asthma

- Magnesium :
  - Bronchial muscle relaxation

Magnesium in Bronchospasm

- Magnesium is a calcium blocker
  - Smooth muscle relaxation

Hypermagnesemia

- Renal Failure
  - Magnesium ingestion +/-
  - Lithium ingestion
  - Suspect a coexisting hyperkalemia

Summary