Magnesium - Clinical Applications

EMC 360

Key: Mg

Ma, Cline: Mg section of Fluid + 'lyte chapter

" Magnesium can be used to treat every disease … except… "

- **Corey Slovis**, MD

Objectives

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

- Review the functions, physiology, and pathophysiology of magnesium .
- Review the clinical settings or risks for magnesium emergencies .
- Appreciate that Mg^{++} disorders are "clinical diagnoses"
- Appreciate the need for early consideration and rapid intervention in certain patients with suspected hypomagnesia.

Objectives (cont.)

- Appreciate that the rapid intervention in patients with hypomagnesemic states and with other medical emergencies is extremely safe - with one exception.
- Discuss the prehospital management (including recommended dosages) of the above conditions .

Magnesium As An Antiarrhythmic

- Acute MI
- Refractory VT
- Refractory VF
- Torsades
- TCA OD
- Digitalis OD
- Quinidine Toxicity
- Hypokalemic Arrhythmias
- Prolonged Q-T Syndromes
- Any ventricular arrhythmia not due to hypermagnesemia

Use of Magnesium in Acute Myocardial Infarction

- During AMI magnesium levels fall due to a number of factors including :
- an intracellular shift of Mg Hypomagnesemia
  - with or without hypokalemia (potassium levels also may fall or may be low in AMI)
  - predisposing to arrhythmias.

Dose of Magnesium in ACLS

- Unstable Patient
  - 2 grams IV push
    - (1 gm. = 2 cc of 50% solution) 1-2 grams Q 1 minute
- Stable Patient
  - 1-2 grams over 20 minutes
  - Then 1-2 grams per hour

Magnesium For Specific Ventricular Arrhythmias

- Cardiac Arrest
- Ventricular Fibrillation
- Ventricular Tachycardia (esp. Torsades de Pointes),
- "Magnesium should be used for cases of refractory (1) VT and (2) VF"

Magnesium for Torsades

- Torsades de Pointes or "twisting of pointes" Ventricular Tachycardia
- TdP has numerous etiologies but generally can be grouped into the causes of the prolonged Q-T syndromes :

Etiology of Prolonged Q-T Interval / Torsades

- *Electrolyte Deficiency*
  - Hypokalemia
  - **Hypomagnesemia**
  - Hypocalcemia
- *Drugs*
  - Phenothiazines
  - **Cyclic Antidepressants**
  - Quinidine
  - Procainamide
- *Structural*
  - Cardiac (**ischemia**, contusion, myocarditis)
  - Neurologic (stroke, bleed, etc.)
- *Miscellaneous*
  - Hypothermia
  - Hypothyroidism

Magnesium for Torsades

Mechanism of Action

- Reverses underlying etiology
- Decreases Q-T interval
- Non Arrhythmogenic
- Rapidly effective

Non-magnesium Therapies for Torsades:

Non-magnesium therapies have been used in the past for Torsades:

- Lidocaine
  Isoproterinol
  Beta Blockade
  Cardioversion
  Defibrillation
- Overdrive Pacing

Magnesium vs Non-magnesium for Torsades

- Safer than isoproterinol
- Safer than beta blockade
- Safer than cardioversion and defibrillation
- Quicker than pacing
- Excellent in ischemia
- Highly specific and effective

Dose of Magnesium for Torsades

- Unstable Patient
  - 2 grams IV push 1-2 grams Q 1 minute
- Stable Patient
  - 2 grams over 20 minutes
  - 2 grams per hour

Magnesium for Digitalis OD

Digitalis Toxicity

- A poisoned Na-K pump : leak of K out of the cell and Na into the cell
- Hyperkalemia
- Spontaneous depolarizations (PVC, VT, VF, Torsades)
- Increased toxicity with hypomagnesemia
- Increased toxicity with hypokalemia

Magnesium for Digitalis OD

- Stimulates the Na-K ATPase pump
- Increased phase 4 (resting membrane) negativity and restores a normal resting membrane potential.
- Decreased spontaneous depolarizations
- Reverses hyperkalemia due to digitalis poisoning
- Counteracts calcium's effect on digitalis poisoned cells

Magnesium in SVTs

- AF
- PSVT
- MAT [wandering atrial pacemaker; in COPD].

Mg $^{++}$ Mechanism of Action 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

High Risk For Clinically Significant Hypomagnesemia

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

Magnesium for Non Cardiac Emergencies

- ETOH withdrawal
- COPD
- Eclampsia
- Seizures
- Tetanus

Mg $^{++}$ and ETOH withdrawal

- Magnesium is not well studied in ETOH withdrawal.
- There are numerous studies - old and new that show :
  - ETOH abuse increases Mg loss
  - ETOH abusers have a poor intake of Mg
  - low Mg increases likelihood of seizures and DT's.

Magnesium in Alcohol Withdrawal

- Decreases incidence of seizures
- Decreases tremulousness
- Decreases arrhythmias during withdrawal
- Decreases potassium losses
- Increases efficacy Thiamine
- Corrects total body deficit

Magnesium in Alcohol Withdrawal

- Magnesium dose in Treatment of Hypomagnesemia in ETOH withdrawal:
- Total body deficit is 10-20 grams
- Takes 5-7 days to replete Mg^{++} deficit
- Give:
  - 2 grams over 20-60 minutes
  - 5 gram, Q 8 hr IV
  - Don't forget to give: Thiamine

Magnesium in Eclampsia

- Its major benefit is that it blocks vasospasm:
- Eclamptic vasospasm ~ meditated by calcium
- Magnesium is a calcium blocker
- Magnesium blocks vasospasm

Magnesium in Eclampsia

- Lowers incidence of seizures
- Corrects hyperreflexia
- Controls blood pressure
- Titratable
- Non-toxic to the non-term fetus
  (may cause resp. depression in newborn)

Mg $^{++}$ Dosage for Eclampsia

- 4 grams $MgSO_4$ over 5-10 minutes
- Then 1-2 grams per hour
- D/C if:
  - loss of DTR's
  - respiratory depression
  - (urine output below 25 cc/hr)
  - (Mg level above 10mg/dl)

Possible Toxicity of Magnesium in Eclampsia

- Decreased muscle tone
- Decreased respiratory function
- Tocolytic (halts labor)
- Hypotension (especially with other agents)

Magnesium in Seizures

- Magnesium may also be used for noneclamptic seizure patients
- HypoMg correlates with increased seizure frequency

Magnesium in Tetanus-like emergencies

- For some of the same reasons as for seizures , the Mg^{++} ion's mechanism may be helpful for patients with tetanic, tonic muscle contractions.
- Mg^{++} has not yet, however, been well studied as an anti-seizure or anti-tetanic agent

Magnesium in COPD +/or Asthma

Studies on Mg^{++} for RAD began to appear in 80's

- Rolla, et al. Anal Allergy 1988
  10 patients, 2 grams/20 min; FEV1 increased by 9%
- Okayama, et al Journal of Asthma, 1991 *Severe asthma (2 cases of status) patients who failed to respond to standard Tx.*
- Conclusion based on a still limited number of studies, magnesium results in: bronchial muscle relaxation and can be used as an adjunct to standard Albuterol therapy

Magnesium in Bronchospasm

- Calcium blockade
- Smooth muscle relaxation
- Maybe
  - Blocks acetylcholine release
  - Blocks histamine release

Mg Treatment of Bronchospasm

- Not currently recommended for mild asthma
- Not to be substituted for standard therapy
  [Albuterol; ipratropium; methylprednisolone]
- Shortlived effect
- Improves respiratory functions
  [increases FEV1 and FVC]
- Dose:
  - Ped: 50 mg/kg IV over 20-30 min.
  - 1-2 gm. IV over 20-30 min.

Is it Possible to Get Too Much Mg^{++} / Hypermagnesemia

- Renal Failure
  - Magnesium ingestion
  - Lithium ingestion / OD
  - Suspect a coexisting hyperkalemia
- Treatment
  - Treat as a Ca^{++} blocker OD
  - Treat any coexisting hyperkalemia
    - CaCl 10% 5ml IV
    - Bicarb 50 mEq IV
    - Lasix 80 mg IV

Summary

We have discussed:

- Types of clinical settings for Mg^{++} emergencies
  - Many of which are "clinical diagnoses," based on suspicion
- Importance of early administration of Mg^{++} in certain patients
  - Recognizing that a rapid intervention with Mg^{++} in many of these medical emergencies is extremely safe - with one exception.
- Recommended prehospital dosages of Mg^{++}