Magnesium and Drug Interactions: Review

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ABSTRACT: Unfortunately, hypomagnesaemia is the most underdiagnosed electrolyte deficiency in current medical practice, means of diagnosing clinical magnesium deficiency of a lesser degree than other electrolyte deficiency associated with overt signs, such as convulsions or cardiac arrhythmias or other electrocardiographic changes are not readily accessible. Plasma magnesium levels are unreliable as an index of its cellular inadequacy, because the means of evaluating magnesium status are complicated. Until magnesium clinical methodology is improved and made available, the importance of correcting magnesium deficiency in man’s diet and of preventing intensification of a deficit when needs are increased by physiologic or pathologic processes and drugs, it will have to be inferential-based on suggestion of experimental and epidemiologic observations. Because magnesium has pharmacologic activities that has been recognized for many years, demonstration of the correction of abnormal acute neurolgic and cardiac signs (even though such signs are characteristic of acute magnesium deficiency) are not readily accepted as evidence that magnesium deficiency can contribute to diseases in which such magnesium-responsive signs are seen. With notable exceptions, there has been clinical neglect of magnesium in most medical centers all over the world. This is unfortunate because many of the pathogenic changes produced by experimental magnesium deficiency or loss resemble many of those of chronic diseases that are responsible for intractable medical problems. This review is to emphasize that magnesium is essential in health and disease status, and to call the medical and scientific organization for the objective examination of the evidence, and for the exploration of the possibility that the prophylactic and treatment uses of magnesium might be effective, and this review also shows the mechanisms of interactions between some drugs and magnesium.

Keywords: Magnesium; Drug Interaction

OVERVIEW:

Total body magnesium content is approximately 25 g, of which 50-60% resides in bone in the normal adult. One third of skeletal magnesium is exchangeable, and it is this fraction that may serve as reservoir for maintaining a normal extracellular magnesium concentration (1). Extracellular magnesium accounts for about 1% of total body magnesium; magnesium is required cofactor for over 300 enzyme systems (2). It is required for both anaerobic and aerobic energy generation and for glycolysis, either indirectly as a part of the Mg-ATP complex or directly as an enzyme activator (3).

Magnesium is a central electrolyte in a large number of cellular metabolic reactions, including DNA and protein synthesis, neurotransmission, and hormone receptor binding (4). It is a component of guanine triphosphatase and cofactor for N-K-ATPase, adenylyl cyclase and phosphofructokinase. Magnesium is present in great concentration within the cell, and is the second most abundant intracellular cation after potassium; magnesium has also been shown to be required for mitochondria to carry out oxidative phosphorylation (2).

The mitochondrial enzymes utilize the magnesium chelate of ATP and ADP as the actual substrates for phosphate transfer reactions. Magnesium presence is important for maintaining an adequate supply of purine and pyrimidine nucleotides required for the increased DNA and RNA synthesis that occur during cell proliferation (5-6). Magnesium transport into or out of cells appears to require the presence of carrier-mediated transport system (7-8). The molecular characteristics of the magnesium transport proteins have not been described. Magnesium transport in mammalian cells may be influenced by hormonal and pharmacological factors (7-8).

Magnesium is necessary for Na-K-ATPase activity, which is responsible for active transport of potassium (10). Magnesium regulates the outward movement of potassium in myocardial cells (11). The arrhythmogenic effect of magnesium deficiency may be related to magnesium’s role in maintaining intracellular potassium. Magnesium has been called “nature’s physiological calcium channel blocker” (12). Since
calcium plays an important role in skeletal and smooth muscle contraction, a state of vasospasms. Magnesium depletion is found in a number of diseases of cardiovascular and neuromuscular function, in malabsorption syndrome and in alcoholism. Among conditions that increase the risk of magnesium deficiency are 1: metabolic factors that affect the absorption, distribution and excretion of this mineral, 2: disease and therapy, 3: physiologic states that increase requirement for nutrients, and 4: nutritional imbalance.

**ABSORPTION AND EXCRETION:** Magnesium absorption in both children and adult is inversely proportional to the amount of magnesium ingested. A principal factor that regulates intestinal magnesium transport has not been described. Vitamin D and its metabolite 25-hydroxyvitamin D and 1, 25 dihydroxyvitamin D enhance intestinal magnesium absorption to small extent. Kidneys are organs mainly responsible for maintaining an internal environment compatible with life processes, and the principal organs involved in magnesium homeostasis over a rather wide range of magnesium intake. However, no one has described a hormone or factor that is responsible for renal magnesium homeostasis, hyper or hypoparathyroidism usually have normal serum magnesium concentrations and a normal tubular maximum for magnesium, it is probable that parathyroid hormone is not an important regulator for magnesium homeostasis. Glucagon, calcitonin and ADH affect magnesium transport in loop of Henle in a manner similar to parathyroid hormone, but the physiological relevance of these actions is unknown. Little is known about the effect of vitamin D on renal magnesium handling. Several studies have found that high sodium and calcium intake may result in increased renal magnesium excretion.

Urine is the major route for excretion of magnesium; changes in urinary magnesium excretion are much greater in magnitude and easier to measure and detect. There is an apparent obligatory urinary loss of magnesium in human, even on marked restriction of magnesium intake. Severe magnesium deficiency may have resulted in renal damage. The renal handling of magnesium in human is a filtration-reabsorption process; there is no tubular secretion of magnesium. Hypermagnesemia has not been documented magnesium depletion may result in muscle cramps, hypertension, coronary and cerebral following the intake of high levels of dietary magnesium in absence of either intestinal or renal disease. Hypermagnesemia can occur in individuals with impaired renal function and is most commonly associated with the combination of impaired renal function and excessive intake of nonfood magnesium as antacids. Hypermagnesemia resulting from impaired renal function and/or intravenous administration of magnesium can result in more serious neurological and cardiac symptoms, but elevated serum magnesium concentrations greater than 4.8 to 8.4 mg/dl must be attained before onset of these symptoms. Death from very large exposures to magnesium in the form of magnesium sulfate or magnesium oxide has been reported following cardiac arrest, especially in individuals with renal insufficiency. However, intake of non-food magnesium has rarely been reported to cause symptomatic hypermagnesemia in individuals with normal renal function. The 24 hours magnesium urinary excretion was significantly higher in boys than in girls. To evaluate magnesium status in the body, attention could be paid to the effects of changes in magnesium intake on urinary magnesium-creatinine ratios, and also on magnesium tolerance test, which is based on the renal excretion of a parenterally administered magnesium load, has been used for many years. It is considered by some to be accurate means of assessing magnesium status in adults, but not in infants and children.

**METABOLISM:** Metabolism of several mineral and trace elements including magnesium are still unclear, and recommended dietary allowance is unavailable for many essential elements. Aging, stress, diseases, pollution, style of life and drugs often change the bioavailability of some essential minerals and trace elements including magnesium are still unclear, and recommended dietary allowance is unavailable for many essential elements. Aging, stress, diseases, pollution, style of life and drugs often change the bioavailability of some essential minerals and trace elements in living organisms and human beings which evolve electrolytes disorder. Deposition of some essential and/or toxic elements in cells and tissues causes cell degeneration which is involved in oxidative stress, inflammation, chronic diseases and cancer. However, bioremediation (regeneration) of living cells and tissues still difficult. Homeostasis of magnesium in extracellular fluids (maintaining normal level of serum magnesium) is regulated by intestinal and renal functions.
Balance fluxes of magnesium between intracellular fluid and extracellular fluid are essential for cells especially bone, muscle, soft tissue and erythrocyte. 

Unfortunately, serum magnesium deficiency is often undetected and long term of damage and death could result from chronic magnesium depletion, yet serum levels of magnesium are remarkably constant, and magnesium is the most underdiagnosed electrolyte deficiency in current medical practice. Digitalis toxic patients are likely to be magnesium depletion even though their serum magnesium level may be normal. Lymphocyte potassium and magnesium had decreased in spite of normal plasma electrolyte levels after using captopril. During magnesium deficiency metabolic alterations are due to decrease intracellular contents of magnesium and potassium, and increased intracellular contents of sodium and calcium, these are more notably in soft tissues than in serum. In rats with magnesium deficiency calcium deposition has been found in heart, pancreas and kidneys. Also in other experiments, rats which have been fed a magnesium deficient diet, the wet and dry weight of the kidney were significantly increased, and the concentrations of calcium and phosphorous in the kidney were significantly higher than rats fed control diet. Also, deposition of calcium was observed in the renal tubules of the corticomedullary junction and medulla of these rats. However, dietary magnesium level of approximately twice the normal level effectively reduces kidney calcification.

Decrease blood and tissues magnesium predispose to arterial hypertension, and may be related to increase vascular reactivity and predispose to a lower energy production in cardiac cells, as well as to disturbances of endothelial nitric oxide release. In vitro magnesium ions block the entry of calcium ions into smooth muscle cells, decrease the peripheral vascular resistance, relieve peripheral and coronary spasms and decrease blood pressure. Calcium overload is toxic to cells; it is now felt that calcium may be the final mediator of cellular destruction for a variety of agents which produce experimental cardiac necrosis. The toxic agents, catecholamines, cardiac glycosides, vitamin D all increase cytoplasm calcium, and in massive doses appear to overwhelm the cell with it. Cell death is ascribed to exhaustion of ATP supply caused by activation of calcium stimulated ATPase and to poisoning of mitochondria by calcium overload. Also, calcium ion is a very busy ion with lots of different targets and mediators scattered throughout the animal and plant cells. However, the tools are now being invented to accurately map in cellular space and time the calcium ion spikes, waves and oscillations that start the several stages of the cell cycle and dffpoptosis. Clearly these new more sophisticated tools will give us insights into the awesome networks used by calcium ion in its roles of cell cycle driver, differentiator and killer.

In fact, it is now clear that the clinical significance of hypomagnesaemia as an isolated finding is speculative at best, intracellular deficit and/or functional disorders which can with confidence be ascribed to magnesium deficiency may or may not be found in hypomagnesaemia individuals, and conversely, clear cut evidence has been presented for significant defects in total intracellular ionic magnesium in the absence of hypomagnesaemia. Of particular importance with respect to the pathogenic effect of magnesium depletion is the role of magnesium in regulating potassium fluxes. The arrhythmias of magnesium deficiency are probably due to the loss of intracellular potassium, since without adequate magnesium, cells are unable to maintain normal concentrations of intracellular potassium. The potassium released by cells subsequently lost from the body and this manifests as arrhythmia. It will be difficult or impossible to replace the lost of potassium until magnesium deficiency is corrected, since the administered potassium is not retained by the cells.

Evidence from clinical and experimental studies including whole animal and cell culture experiments indicate that homeostasis of magnesium and potassium are closely related in the whole organism, also deficiencies of magnesium and potassium frequently co-exist with gastrointestinal and especially renal losses from diuretic and nephrotoxic drug treatment being mainly responsible. Other evidence indicates that magnesium has a direct effect on potassium transport at the cellular level. Include the effect on Na-K-ATPase, Na-K-Cl co- transport, K channels, charge screening and permeability effects on membranes. Interrelations of
magnesium and potassium in cardiac tissue have probably the greatest clinical significance in term of arrhythmia, digoxin toxicity and myocardial infarction \textsuperscript{(55)}. Most of the early pathologic consequences of magnesium depletion are neurologic or neuromuscular defects of which probably reflect the influence of magnesium on potassium flux within tissues.

Magnesium deficit produces anorexia, nausea, muscle weakness, lethargy, staggering and weight loss, and progressively increasing with the severity and duration of magnesium depletion are manifestation of hyperirritability, hyperexcitability, muscle spasms and tetany, leading ultimately to convulsions \textsuperscript{(53,56)}. Magnesium deficiency causes early cytological and immunological modification in the spleen, which appear before macroscopical changes in this organ and before clinical symptoms of inflammation. These changes could be related to the altered immune response of deficient body \textsuperscript{(57)}. Several hormones have a significant role in magnesium metabolism, stress hormones (glucocorticoids, mineralocorticoids and catecholamines) increase magnesium excretion in urine, hormones maintaining pregnancy also increase secretion of magnesium in urine. Patients with diabetes mellitus had significantly higher urinary excretion of magnesium than healthy subjects \textsuperscript{(20,58-59)}.

Routine serum magnesium determinations in hospital population suggest a prevalence of hypomagnesaemia between 7 and 11\% \textsuperscript{(60-61)}. In intensive care units the reported incidence is between 8 and 20\% \textsuperscript{(62-63)}. Derangements in other electrolyte imbalances are often associated with hypomagnesaemia, it was reported that 42, 29, 23 and 22\% incidences of hypomagnesaemia in patients with hypokalemia, hypophosphatemia, hyponatremia and hypocalcemia, respectively \textsuperscript{(64)}. There is an increased interest in the role of magnesium repletion in preventing and managing electrolyte disorders and related chronic diseases such as hypertension, cardiovascular diseases and diabetes. Usefulness of increasing magnesium intake as a life style modification in management of hypertension was supported, however, its antihypertensive effect may be small \textsuperscript{(65)}. Recently, ion specific electrode has become available for determining ionized magnesium in the plasma. Early results suggest that this may be a better index of magnesium status than the total serum magnesium concentration, few studies have been conducted under varying condition to assess its validity and further evaluation is necessary \textsuperscript{(66-67)}. Future studies will be aimed at elucidating mechanisms of Mg-K interrelation at the cellular level using new techniques with the ability to detect concentrations and modulations of free intracellular magnesium \textsuperscript{(55)}.

**DRUGS AND REPLACEMENT THERAPY:**
Hormones and hormonal replacement therapy showed interactions with magnesium status. Gonad steroids cause a decrease in serum magnesium by increase cell influx of magnesium and inhibiting bone resorption \textsuperscript{(68)}, and using oral contraceptives causes lowering in serum magnesium \textsuperscript{(69)}. The benefit of treatment cardiovascular disorder by estrogen might drive from the role of estrogen in increasing intracellular levels of magnesium \textsuperscript{(70-72)}. Physiological concentrations of both estrogen and progesterone help cerebral vascular smooth cells sustain normal magnesium ion concentrations which are beneficial to vascular function \textsuperscript{(73)}. Ionized and total magnesium were shown to be inversely related to serum estrogen level, low serum magnesium was also noticed with high level serum of progesterone. However, serum level of calcium were significantly higher in menopausal than in young women, therefore, sex hormones induced changes in magnesium and calcium were deemed sufficient to affect the vasculature directly, particularly in women who are marginally magnesium deficient \textsuperscript{(74-75)}. The following drugs have showed different effects on magnesium status by different mechanisms.

**AMILORIDE:** Amiloride has a magnesium-sparing effect in addition to its potassium-sparing effect, consequently there is possibility that individuals who take magnesium supplement while also taking Amiloride could built up excessively high level of magnesium. The concurrent use of hydrochlorothiazide and Amiloride would make this accumulating, unlikely given the magnesium depletion action of hydrochlorothiazide \textsuperscript{(76-77)}. By a direct renal action Amiloride exert its sparing effect on magnesium. However, alteration in glomerular filtration rate, the filtered load of magnesium, arterial blood pressure, the status of extracellular fluid volume, plasma aldosterone concentration and acid base balance were not involve in mechanisms of magnesium excretion sparing effect by Amiloride \textsuperscript{(76)}. Amiloride and trimterene also reduce urinary magnesium excretion \textsuperscript{(77)}.
AMINOGLYCOSIDE: Aminoglycoside, such as Gentamycin cause renal tubular damage which lead to hypomagnesaemia, hypocalcaemia, hypokalemia and alkalosis. Hypomagnesaemia could be especially common among children with cystic fibrosis who have a history of repeated use of Aminoglycosides (78-80). Others found that acute Gentamycin infusion was associated with significant hypercalciuria and hypermagnesiuria with absence of changes in renal cellular morphology and without underlining renal tubular damage (81). Hypomagnesaemia in children with cystic fibrosis treated with Aminoglycosides reveal excessive renal loss of magnesium. It is postulated that renal tubular damage secondary to the cumulative effects of repeated courses of Aminoglycosides resulted in hypomagnesemia (82), and the early physiological effects of Gentamycin on the kidney occurs in a different nephron segment from any subsequent nephrotoxicity (83). Gentamycin calciuria is related to non-antibacterial properties that may interfere with transtubular calcium transport (calcium channel blockade, Na, K-ATPase inhibition or competition with calcium for binding to the brush-border membrane). Calciuric effects of Gentamycin and Furosemide were additives, and caused by different mechanisms whereas the non-calciuric diuretic (chlorothiazide) had no effect on Gentamycin calciuria. Poly-L-aspartic acid cause isolated calciuria similar in magnitude and character to Gentamycin. Gentamycin and Poly-L-spartic acid calciuria may reflect interference with intra cellular events related to transtubular calcium transport (84).

CALCIUM CHANNEL BLOCKERS (Diltiazem): Patients with variant angina often suffer from magnesium deficiency and it is corrected after treatment with calcium antagonists (85-86).

Captopril: Captopril increase lymphocyte magnesium levels (87). Captopril treatment can produce translocation and/or elimination of zinc, copper, magnesium and calcium in various tissues. Captopril significantly decreased zinc concentration in liver, copper concentration in liver, adrenals, jejenum, urine and hair and magnesium concentrations in blood and urine. A significant increase was observed in testicular and epididymal zinc, in heart, epididymal, and fecal copper, in magnesium concentration of lung, kidneys, adrenals, jejenum, epididymis and hair and in calcium concentration in brain, heart, lung, kidneys, spleen and stomach (88). Increased intracellular potassium and magnesium may be one mechanism whereby angiotensin-converting enzyme inhibitors reduce arrhythmias and improve survival in patients with congestive heart failure (37).

CIPROFLOXACIN: Absorption of Ciprofloxacin is reduced by 50 to 90% in the presence of antacids containing magnesium (89-92). Other cations, such as calcium, iron and probably zinc appear to interact in a similar manner. Chelation between the quinolone and cation is the most likely mechanism (93). Ferrous sulfate and multivitamins with zinc significantly impaired the absorption of Ciprofloxacin (94).

CISPLATIN: Cisplatin induce hypomagnesemia through its renal toxicity possibly by a direct injury to mechanisms of magnesium reabsorption in the ascending limb of the loop of Henle as well as the distal tubule, consequent, Cisplatin increases the urinary loss of magnesium. This drug-induced impaired of the renal tubules ability to conserve magnesium may persist for months, or possibly years, after discontinuing use of the drug (95-97). Beside renal magnesium wasting, a clear-cut tendency towards reduced calciuria associated with normal or slightly elevated plasma calcium was observed. Plasma potassium tended to be low and metabolic alkalosis (98), potassium supplementation to increase serum calcium induced tetany, hypokalemia in the face of hypomagnesemia may have a membrane-stabilizing effect and preserve excitability (99). Renal excretion of phosphorous and magnesium were increased in patients and hypercalciuria was occasionally seen, divalent ions disorders are the most likely potential complications (96). The incidence and severity of hypomagnesemia are dose dependent, and the cause of higher incidence of hypomagnesemia is unknown, but may be related to an interaction of Cisplatin with another drug (100). Magnesium supplements were recommended to be given to patient receiving Cisplatin during the preisplatin hydration period to prevent hypomagnesemia (101).

CYCLOSPORINE: Cyclosporine has been linked to reduced serum level of magnesium, systemic depletion of magnesium produce a high of seizure due to Cyclosporine-induced toxicity to nervous system. Patients taking Cyclosporine therapy should have their magnesium levels.
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tested regularly, and monitoring red blood cell magnesium levels, rather than serum magnesium, which is the most accurate method for diagnosing a deficiency. Magnesium supplementation prevents magnesium deficiency and subsequent neurotoxicity \[102-103\].

**Digoxin:** Digoxin decreases intracellular magnesium, thereby causing increase urinary magnesium loss. Magnesium deficiencies induced by concomitant diuretic use are very common in individual using digoxin. Hypomagnesemia may predispose to digitalis toxicity \[95,104\]. Hypomagnesemia is known to produce a wide variety of clinical presentations, including neuromuscular irritability, cardiac arrhythmias, and increased sensitivity to digoxin. Magnesium deficiency also inhibits the therapeutic efficacy of digoxin in controlling atrial fibrillation. Refractory hypokalemia and hypocalcemia can be caused by concomitant hypomagnesemia and can be corrected with magnesium therapy \[95,105-106\].

Unfortunately, magnesium deficiency is not considered unless serum level falls below acceptable levels. Also, serum magnesium is a very poor indicator of how much magnesium is actually in the tissues. Serum magnesium concentration is maintained within a narrow range by the kidney and small intestine since under conditions of magnesium deprivation both organs increase their fractional absorption of magnesium. If magnesium depletion continues, the bone store contributes by exchanging part of its content with extracellular fluid. The serum magnesium can be normal in the presence of intracellular magnesium depletion, and the occurrence of a low level usually indicates significant magnesium deficiency. The detection of magnesium deficiency can be increased by measuring magnesium concentration in the urine or using the parenteral magnesium load test \[104\].

Decreased cellular magnesium content with normal serum magnesium level predisposes to digitalis-toxic arrhythmias. Intravenous bolus administration of magnesium sulphate, followed by intramuscular magnesium repletion, abolished the digitalis-toxic arrhythmia \[107-108\].

Magnesium deficiency may be a contributory factor in patient with chronic atrial fibrillation with prevalence of ventricular premature beats, and magnesium loading test showed that there was an overall relationship between magnesium retention and number of ventricular premature beats \[106\]. Magnesium deficiency was the most frequent identified significant electrolyte disturbance in relation to digoxin toxicity, and in the presence of magnesium deficiency digoxin toxicity developed at relatively low serum digoxin concentration \[105\]. Routine serum magnesium determination in patients receiving digitalis, who often are also receiving diuretics, may assist in identifying the toxic effect of digoxil \[109\]. Individual taking digoxin will almost always benefit from supplementation of magnesium \[108,110\].

**Loop diuretics and thiazide diuretics:** Potassium depletion diuretics increase potassium excretion and in practice, they also usually deplete blood levels of magnesium. In turn, the drug induced magnesium deficiency can contribute to further potassium depletion. Ultimately, the relationship between these two patterns of depletion can be difficult to determine \[111-112\]. A lack of magnesium interferes with healthy cardiac muscle function, this is especially important for patients on both diuretics and digitalis as they are more likely to develop arrhythmias if not adequately supplemented with magnesium. Serum levels of magnesium are not adequately sensitive to mild to moderate levels of depletion and thus are poor indicator of nutritional status. In practice, it is generally advisable for individuals taking any potassium depleting diuretic, other than those with kidney failure, to supplement with both potassium and magnesium \[57\].

**Lithium:** Magnesium and lithium are chemically related. Magnesium is an essential ion in many enzyme systems and lithium is of value in the treatment of manic-depressive disease. A significant sex difference in mean plasma magnesium level was reported in depressed patients. The consumption of lithium carbonate may cause high blood levels of magnesium \[113-115\].

**Ranitidine:** Antacids (magnesium hydroxide) reduce the bioavailability of the H2 receptor antagonists, antacid decreased Ranitidine and Cimitidine absorption by 20% only when high antacid doses are used and when the drugs are administered simultaneously \[116-117\].

**Tetracycline:** Magnesium interferes with tetracycline absorption and reduces its effectiveness by chelating the drug, and chelates of magnesium and tetracycline may play a role in the toxicity of tetracycline. Magnesium in the form of supplement should be avoided while using tetracycline. Nevertheless, tetracycline and antacids are often used together in combination.
therapies for Helicobacter pylori\textsuperscript{(118)}. Tetracyclin absorption may be decreased by more than 90% by this interaction. Finally, bioavailability of other drugs like beta-adrenergic blocking drugs (atenolol or propranolol) did not reduced during antacid treatment, also nonsteroid anti-inflammatory drugs such as naproxen, tenoxicam, ketoprofen, ibuprofen and piroxicam are not affected in their absorption by antacid treatment, and also theophiline bioavailability is unchanged when the drug is given together with antacids\textsuperscript{(119-120)}.

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