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Opinion Article

Low Serum Concentrations in Magnesium Deficiency and Clinical Therapy

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DESCRIPTION

Magnesium is an essential mineral that plays a role in over 300 metabolic reactions in the human body. These reactions affect various processes such as protein synthesis, cellular energy production, DNA synthesis, nerve signal transmission, bone metabolism, blood pressure, and glucose and insulin metabolism. The human body contains about 21-28 g of magnesium, most of which is stored in the bones and non-muscular tissues. Only about 1% of magnesium is found in the extracellular fluid, and this is the fraction that is measured by serum magnesium tests. Serum magnesium concentration is normally maintained within a range of 1.8 to 2.4 mg/dL (0.75 to 1 mmol/L). However, serum magnesium level is not a reliable indicator of total body magnesium status, because it can be influenced by various factors such as protein binding, acid-base balance, hormonal regulation, and renal excretion. Low serum magnesium concentration, or hypomagnesemia, is generally defined as serum magnesium level below 1.5 mg/dL (0.6 mmol/L). Hypomagnesemia can result from inadequate magnesium intake or absorption, increased magnesium loss or excretion, or redistribution of magnesium from the extracellular to the intracellular space

Some of the common causes of hypomagnesemia are poor dietary intake of magnesium-rich foods such as green leafy vegetables, nuts, seeds, legumes, whole grains, and dairy products. This can lead to chronic magnesium deficiency, which is rare but can occur in conditions such as malnutrition, alcoholism, anorexia nervosa, and elderly population. The Impaired absorption of magnesium in the gastrointestinal tract due to some disease like celiac disease, Crohn's disease, chronic diarrhea, inflammatory bowel disease, short bowel syndrome, gastric bypass surgery, or use of proton pump inhibitors. Increased excretion of magnesium in the urine due to renal diseases such as chronic kidney disease, diabetic nephropathy, interstitial nephritis, or use of diuretics such as loop diuretics (e.g., furosemide) or thiazide diuretics (e.g., hydrochlorothiazide). These drugs can cause electrolyte imbalance and increase the urinary loss of magnesium along with sodium, potassium, and calcium. Increased loss of magnesium in the stool due to laxative abuse or steatorrhea (fatty stools) caused by pancreatic insufficiency or bile salt malabsorption.

Redistribution of magnesium from the extracellular to the intracellular space due to conditions such as re-feeding syndrome, insulin therapy for diabetic ketoacidosis or hyperglycemia, acute pancreatitis, sepsis, burns, trauma, or alcohol withdrawal. These conditions can cause a shift of magnesium into the cells along with phosphate and potassium. Hypomagnesemia can also be associated with other electrolyte disorders such as hypocalcemia (low serum calcium) and hypokalemia (low serum potassium). This is because magnesium plays a role in regulating the secretion and action of Parathyroid Hormone (PTH), which controls calcium homeostasis; and also affects the renal handling and cellular uptake of potassium. Therefore, low magnesium levels can impair calcium absorption and PTH secretion; and increase potassium excretion and loss from the cells. The symptoms of hypomagnesemia depend on the severity and duration of the condition. Mild to moderate hypomagnesemia may not cause any noticeable symptoms or may present with nonspecific signs such as nausea, vomiting, weakness, decreased appetite, and sleepiness.

CONCLUSION

A subclinical magnesium deficit has been linked to insufficient dietary Mg intake in Europe and North America, according to nutritional monitoring programmes. This is primarily because of the characteristics of the western diet, which is high in processed foods and low in micronutrients. Green vegetables are excellent suppliers of magnesium because the center of the pigment chlorophyll contains the element. In addition, foods like nuts, seeds, whole grains, and some legumes are rich sources of magnesium. Diet is not the sole factor, though. There has long been speculation that there are genetically based variations in how Mg is handled. These unusual illnesses helped to identify and characterize a few molecular participants in the homeostasis of magnesium.

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