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## Editorial:

# MAGNESIUM IN PERITONEAL DIALISIS

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Version en español

Magnesium, the fourth most abundant cation in the body, is the second most abundant intracellular cation (after potassium), whose reference value range in adults is 1.7-2.4 mg / dL (0.7-1.0 mmol / L or 1.4-2.0 mEq / L). In patients treated with dialysis, magnesium homeostasis depends on dietary intake, intestinal absorption and renal (residual diuresis) and dialytic elimination. In turn, serum magnesium levels can be influenced by significant changes in dietary intake, residual renal function, dialysis dose of dialysis and its gastrointestinal loss[1].

Magnesium plays an essential role in numerous biological processes, including cardiovascular function. Although it is known that hypomagnesemia plays a role in the pathogenesis of arterial hypertension, endothelial dysfunction, dyslipidemia and inflammation. Hypomagnesemia is independently associated with mortality and cardiovascular mortality.

When the association between serum magnesium levels and mortality was explored, in relation to serum magnesium variability with respect to mortality in patients on peritoneal dialysis, the results revealed an independent relationship between hypomagnesemia and high risk of cardiovascular mortality[1,2].

Studies have shown a strong inverse association of serum magnesium levels with insulin resistance, new onset of diabetes mellitus, oxidative stress, endothelial dysfunction, hypertension, atherosclerosis and systemic inflammation. Observational studies have also shown a significant association between hypomagnesemia and a higher overall mortality in patients with advanced chronic kidney disease not on dialysis, or chronic kidney disease on chronic hemodialysis. It has also been documented that hypomagnesemia is associated with heart rhythm disorders, cardiovascular events, dyslipidemia, metabolic syndrome, endothelial dysfunction, atherosclerosis and vascular

calcification[2].

On the other hand it is known that patients with chronic renal failure have a higher magnesium body content, as well as in chronic dialysis patients the serum magnesium concentration is parallel to the magnesium level of the dialysate. Magnesium in the dialysate produces a decrease in the serum concentration of magnesium and vice versa[1].

Navarro et al. have documented a significant inverse correlation between intact parathormone serum level (iPTH) and serum magnesium level in a study in peritoneal dialysis patients whose peritonenal bath contained 1.5 mEq / L of magnesium. However, this correlation was even greater when the peritoneal dialysate was low in magnesium (0.5 mEq / L or 0.75 mEq / L). The authors evaluated in 56 patients the relationship between serum parathyroid hormone and magnesium levels in patients with continuous ambulatory peritoneal dialysis using low magnesium peritoneal dialysate; finding that the serum level of iPTH did not correlate with the serum level of magnesium, although it did so inversely with the serum level of total and ionized calcium.

However, despite these findings, it is suggested that magnesium plays an important role in the regulation of the level of iPTH, since intravenous infusion of magnesium sulfate can significantly suppress the secretion of iPTH in patients with primary hyperparathyroidism. Although the results of the work did not demonstrate the relationship between magnesemia and iPTH, there are reports that documented the inverse and significant correlation between serum levels of iPTH and magnesium in patients on chronic dialysis[1].

The losses of magnesium in the dialysate are aggravated by the significant restrictions in the renal diet. Taken together, this highlights the risk of hypomagnesemia in patients with peritoneal dialysis, where a low serum magnesium level may be a marker of generalized malnutrition and inflammation, both associated with increased vascular calcification, and a lower serum magnesium level associated with an increase in mortality in dialysis patients [3].

Finally, a significant association between hypomagnesemia and progression of peripheral arterial calcification, calcification of the mitral annulus and atherosclerosis of the common carotid artery has been documented. Thus, interventions such as magnesium supplementation in patients with hypomagnesemia, the use of magnesium-based phosphate binders or the use of magnesium-rich peritoneal dialysis solutions may help to reduce the effect on vascular calcification on peritoneal dialysis[4].

Hypomagnesemia is associated with greater morbidity and mortality in the dialysis population, while adequate nutrition, magnesium concentration in the peritoneal dialysis bath, and the use of magnesium-based phosphate binders would help to avoid it.

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