Magnesium (Mg) is involved in many important enzymatic processes, electrolyte balance, and skeletal metabolism. Clinically, Mg plays an important role in the regulation of vascular tone and heart rhythm, prevention of thrombosis, atherosclerosis, insulin resistance, arrhythmia, and osteoporosis, suppression of inflammation, etc (Figure).1-3 Furthermore, hypermagnesemia inhibits parathyroid hormone secretion, which is considered to be an independent important risk factor for vascular calcification, left ventricular hypertrophy (LVH) and mortality in endstage renal disease (ESRD) patients.4

Mg homeostasis in humans primarily depends on the balance between intestinal uptake and renal excretion. Mg deficiency can result from reduced dietary intake, intestinal malabsorption or renal loss. On the other hand, hypermagnesemia is most commonly caused by renal failure. However, many factors are involved in controlling Mg status in patients with renal failure, particularly ESRD. Some conditions lead to a negative Mg balance, such as excessive intake of diuretics, reduced gastrointestinal uptake because of acidosis, poor nutrition and absorption, and a low Mg concentration of the dialysate.5

Normally, the total body store of Mg is 21–28 g and only approximately 1–2% of total body Mg is present in the extracellular fluid. Clinically, the serum Mg concentration is measured, but like all predominantly intracellular ions, the serum Mg concentration does not necessarily reflect the total body Mg balance. Unfortunately, better determinates of total Mg balance are not readily accessible nor have they been standardized.

In this issue of the Journal, Ochi et al report that hair Mg, but not serum Mg, is negatively associated with LV wall thickness (LVWT) in hemodialysis patients.6 In their study, Mg concentration in the scalp hair was measured as a representation of intracellular Mg or tissue Mg storage. The authors considered that lower intracellular Mg concentration, as reflected by lower hair Mg concentration, causes reduced function or injury of cardiomyocytes, possibly leading to increased LVWT and LVH. The evaluation of intracellular Mg concentration using

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**Figure.** Possible mechanisms of association between magnesium and cardiovascular and metabolic disease. (Reproduced from Kanbay et al with permission)7

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Hair Mg and LV mass index (LVMI). Previous reports have shown that the intracellular Mg level is closely and inversely related to LV mass. Recent experimental and clinical investigations suggest an intriguing pathophysiological concept linking development of LVH with Mg deficiency; that is, Mg deficiency may induce cardiac fibrogenesis and myocardial damage correlates poorly with that in skeletal muscle. Animal research has documented that Mg supplementation prevents angiotensin-II-induced myocardial damage. LVH in patients with ESRD is independently associated with higher subsequent mortality, as is the case in the general population. Various risk factors for LVH have been suggested in patients with chronic renal failure, some of which are shown in the Table. If hair Mg is documented to relate independently to LVH in further research, then reduced hair Mg may be considered as an indicator of additional risk of LVH. Further investigation is needed to increase knowledge of the clinical significance of hair Mg.

References


