KSN SEOUL, KOREA 2023
April 27(Thu) - 30(Sun), 2023
Coex, Seoul, Korea

Submission No.: FE01-9199
Session Title: Fluid & Electrolyte
Date & Time, Place: April 28 (Fri), 08:30 - 10:30, Room 1

Common Cases Referred to Nephrologists for Magnesium Disturbance

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Magnesium is the second most common intracellular cation and the fourth most common extracellular cation. The human body possesses 24–26 g (1,000 mmol) of magnesium. Magnesium is required for normal metabolic function. This mineral is a cofactor in a variety of enzymatic reactions, including energy metabolism and DNA and protein synthesis. Magnesium regulates ion channels and functions as an enzyme cofactor in numerous biochemical reactions. Hypomagnesemia is defined as serum magnesium levels below.1.8 mg/dL. Hypomagnesemia is not uncommon among non-dialysis CKD patients despite their decreased glomerular filtration rates; even in CKD stages G4 and G5, the prevalence rate was approximately 15%. Hypomagnesemia may occur as a result of impaired gastrointestinal absorption or abnormal renal wasting. According to the pathophysiologic mechanism, the causes of hypomagnesemia can be classified as follows: decreased intake, transcellular shift, gastrointestinal losses, transdermal losses, and renal losses. Tubular dysfunction/interstitial fibrosis may play a pivotal role in the development of hypomagnesemia in CKD, which impairs tubular magnesium reabsorption, among other potential causes of hypomagnesemia. In turn, magnesium deficiency may contribute to the progression of CKD. Hypomagnesemia is a prevalent condition that contributes to morbidity and mortality. Magnesium level is associated with vascular calcification and mortality among CKD patients. Oral magnesium supplementation, adjunctive oral medications to stimulate renal tubular reabsorption of magnesium, and parenteral magnesium supplementation are pharmacologic treatment options for hypomagnesemia. Magnesium sulfate is commonly used for intravenous electrolyte solutions and is the preferred salt for parenteral administration. Hypermagnesemia is defined as magnesium levels of 2.5 mg/dL or higher. Depending on the study, the incidence of hypermagnesemia in hospitalized patients ranges from 3–5% to 10–12%. The incidence is higher among patients with suboptimal kidney function and intensive care unit patients. The nervous, cardiovascular, respiratory, and gastrointestinal systems are most frequently affected by magnesium toxicity. In mild instances, symptoms may be limited to nausea, dizziness, and confusion. Identifying and eliminating the offending agent is the first step in treating hypermagnesemia. Administration of a dose of 100–200 mg of elemental calcium over 5–10 min counteract the neuromuscular and cardiovascular ramifications of hypermagnesemia. In cases of severe (levels >6 mg/dL), symptomatic hypermagnesemia in which patients have severe symptoms or anuria, dialysis is indicated.