Case Report

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Hypermagnesemia and Dilated Cardiomyopathy in a 10-year-old Male Patient on Hemodialysis: A Case Report

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Abstract:

A combination of hypermagnesemia, dilated cardiomyopathy, hypertension, and chronic renal frailer is a rare presentation in children. One of important and rare side effects of chronic renal frailer is hypermagnesemia. In this article, we discuss the association between hypermagnesemia and cardiomyopathy and how patients with none oliguric chronic renal failure develop hypermagnesemia. A 10-year-old boy presented to our PICU ward with chief complaints of respiratory distress, lethargy, and weakness since three days ago. On physical examination, the patient had respiratory distress and was hypertensive and ill.

Keywords: Hypertension; Hypermagnesemia; Child; Cardiomyopathy; Hemodialysis.

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Introduction

Magnesium is an important intracellular cation. It is involved in energy metabolism, protein and nucleic acid synthesis, modulation of membrane transporters, and signal transduction (1, 2). The kidneys are responsible for magnesium homeostasis and there is no feedback mechanism to prevent magnesium absorption in the gastrointestinal tract (1,2, 3). The kidneys usually excrete excessive magnesium, but this ability is diminished in patients with progressive chronic kidney diseases and chronic renal failure (CRF). Hypermagnesemia must be taken into consideration in any patient presenting with chronic renal failure.

Recent advances in molecular genetics of hereditary hypermagnesemia have shown the role of a variety of genes in human epithelial Mg transport (3-9). Several studies suggest that Mg plays an important role against the development of cardiovascular diseases, infectious diseases, and malignant neoplasia and therefore changes in the Mg level may be related to mortality (10).

Hypermagnesemia is an uncommon laboratory finding and symptomatic hypermagnesemia is even less common. The plasma magnesium concentration is 1.5-2.3 mg/dL in healthy children (3).

Given the intracellular nature of this cation, serum magnesium concentrations poorly reflect the total body status (3, 8).

The kidney is the main organ responsible for magnesium homeostasis. Approximately 70–80% (2.4 g/day) of the total serum magnesium is filtered by the kidneys. Under normal circumstances, 95–97% is reabsorbed by the tubules (2,3). In contrast to other electrolytes, control of magnesium reabsorption does not appear to be tightly regulated by a specific hormone (1,3).

The most common cause of hypermagnesemia is renal failure; other causes include excessive intake, lithium therapy, hypothyroidism, addison disease, familial hypocalciuric hypercalcemia, and milk alkali syndrome (5,6).

Hypermagnesemia inhibits acetylcholine release at the neuromuscular junction, causing lethargy, sleepiness, hypotonia, hyporeflexia, hypotension, flushing and weakness, and paralysis. ECG changes include prolonged PR, QRS, and QT intervals (3-8). In hypermagnesemia, because Mg acts as a calcium channel blocker, the blood vessels tend to dilate, which leads to lower blood pressure levels. It also causes prolonged QT intervals, increased QRS duration, and bradycardia on electrocardiograms and cardiac arrest (5,7).

Other manifestations of hypermagnesemia include nausea, vomiting, and hypocalcemia. Prevention is essential; magnesium containing compounds should be used judiciously in children with renal insufficiency (1,2). Both admission hypermagnesemia and hypomagnesaemia are associated with an increased risk for in-hospital AKI (4, 5).

In CKD, the progressive decrease in the glomerular function leads to disturbances in the mineral metabolism that generally cause secondary hyperparathyroidism. This report provides an overview of the most recent state of knowledge concerning the mechanisms leading to the development of hypermagnesemia in the CKD setting (11). The dilated form of cardiomyopathy is typically characterized by a ventricular chamber enlargement, systolic dysfunction, normal wall thicknesses of the myocardium, and mitral regurgitation (12- 14). In this article, we discuss whether hypermagnesemia is associated with cardiomyopathy while we usually face hypomagnesaemia induced cardiomyopathy.

Case Report

A 10-year-old boy presented to our PICU ward with chief complaints of respiratory distress, chest pain, vomiting, headache, lethargy, and weakness since three days ago. He had a history of renal failure due to posterior ureteral valve and underwent surgery for a vesicostomy and obstruction repair. He received supplements, vitamins, Eprex (Erythropoietin), and ferrous sulfate for treatment of CRF. On physical examination, he had respiratory distress and was hypertensive and ill. The results of laboratory tests were as follows: K: 4.2mg/dl, Na: 142mg/dl, Mg: 5.2 mg/dl, FBS: 82mg/dl, BUN: 84 mg/dl, Creatinine: 6.2 mg/dl, VBG (pH: 7.21, HCO2: 5.9, PCO2: 14.7), U/A (WBC: 0-1, RBC: 0-1, SG: 1.025, Sugar: 1+, Ketone: 1+). He was admitted with a diagnosis of one of renal failure complication. After admission, all laboratory tests and cardiology consultation were requested.

Discussion

The fact that our patient had cardiomyopathy in association with hypermagnesemia does not clearly establish a direct relationship. To date, cardiomyopathy has been reported in some patients with hypomagnesemia (11-13). These cases are unusual for two reasons. First, concurrence of hypermagnesemia and cardiomyopathy is rare in chronic renal failure and there are no similar reports in the literature. Second, the patients developed hypermagnesemia during the course of this illness, which is uncommon in none oliguric patient.

Most patients with a normal renal function rapidly clear excess magnesium.

Intravenous hydration and loop diuretics can accelerate this process. In severe cases, especially in patients with underlying renal insufficiency, dialysis may be necessary. Hemodialysis acts faster than peritoneal dialysis. Exchange transfusion is another option in newborn infants. Supportive care includes monitoring of the cardiorespiratory status, fluid therapy, and monitoring of electrolyte levels (1-3).

In our case, a 10-year-old boy presented with respiratory distress, chest pain, vomiting, headache, lethargy and weakness since three days ago. He had none oliguric CRF and he was on hemodialysis from one-year ego.

The risk of hypermagnesemia after CRF is small and its concurrence with cardiomyopathy is rare (1-5).

The risk of hypermagnesemia after CRF depends on the renal function, oliguria and adequacy of hemodialysis (3). Serial evaluations of blood electrolytes levels and measurement of cardiac function are recommended every 3 months (1,15,16).

Conclusion

Regarding cardiomyopathy in association with hypermagnesemia and none oliguric renal failure, more studies are required to confirm the results of this study because no similar findings have been reported in other parts of the world. The possible pathophysiological mechanisms are not clear and more research is needed to find the reason. We

recommend more studies on electrolyte imbalance in CRF, especially larger, multi-center investigations.

Limitations of the Study

Our report was conducted in a restricted number of patients and was single-center. We suggest more investigations on this subject.

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Author's Contribution

Mohsen Akhavan Sepahi (MAS) and Rozita Hoseini (RH) were the principal investigators of the study. MAS and RH participated in preparing the concept, design and revision of the manuscript and critically evaluated the intellectual contents. The authors have read and approved the content of the manuscript and confirmed the accuracy or integrity of any part of the work.

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