

Case Report

Assessment of serum magnesium levels in patient suffering from renal lithiasis

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Accepted 03 October, 2014

Hypomagnesaemia is common finding in current medical practice mainly in critically ill patients. Magnesium has been directly implicated in hypokalemia, hypocalcaemia and dysrhythmias. We report a known case of renal lithiasis, in a 45 year old female patient, with chief complaint of severe pain in right lumbar region and breathlessness with confirmed electrolyte imbalance. The objective of the present case report was to investigate the incidence of hypomagnesaemia in the critically ill patient and multiple electrolyte imbalance. It was also observed that magnesium (Mg) supplementation corrected the underlying multiple electrolyte disturbances in the patient thus, establishing a positive correlation of magnesium with sodium (Na), potassium (K) and calcium (Ca).

Key words: Hypomagnesaemia, hypokalemia, hypocalcaemia, hyponatremia.

CASE REPORT

A 45 year old female patient, with a known case of renal lithiasis, was admitted in the Emergency, Sri Guru Ram Das Charitable Hospital, Vallah with the chief complaint of severe pain in right lumbar region and breathlessness. There was a history of fever, vomitings, burning micturition, anorexia and hypertension. The ultrasound report of the patient presented right renal lithiasis stone size (8 mm). The ECG findings of the patient presented atrial flutter and fibrillation. There was no history of rigor and chill, hematemesis, hematuria, jaundice, tuberculosis, epilepsy and drug history. The vitals of the patient on admission were as follows: Pulse- 98/min; BP- 168/100 mm of Hg and Respiratory Rate- 18/min, HR- 130/min.

A number of investigations were performed in the Clinical Biochemistry Laboratory, Sri Guru Ram Das Charitable Hospital, Vallah, Amritsar as shown in the Table 1 below.

The patient's investigation reports showed hypo-kalemia, which was accompanied by hyponatremia and hypocalcaemia. Urinary sodium and potassium were 180 meq/L (Normal range: 40-220 meq/L) and 75 meq/L (Normal range: 25-120 meq/L) respectively. The patient's serum and urinary pH were normal. Meanwhile the patient, along with dietary supplementation, was given intravenous KCl and Ca gluconate but the hypokalemia and hypocalcaemia still persisted. On the 4th day of admission the patient was investigated for serum Mg and the patient presented hypomagnesaemia. On the 5th and 6th day the investigations were repeated and the hypokalemic, hypocalcaemic and the hyponatremic state of the patient started improving with Mg supplementation. As soon as the supplementation of Mg was stopped, the patient presented hypokalemia and hypocalcaemia (as shown in the table on 7th day); this improved again after the supplementation of Mg, along with K and Ca. Thus, it was observed that it was only after Mg supplementation that the blood levels of Na, K and Ca along with Mg reached the near normal levels. Normal levels of the electrolytes are Na: 135-148 meq/l; K: 3.5-5.3 meq/l; Ca:

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Table 1. Investigations and supplementations.

Day	st ₁	nd ₂	rd ₃	th ₄	th ₅	th ₆	th ₇	th ₈	th ₉	th ₁₀
Serum Na ⁺ (meq/l)	125	127	127	126	130	137	138	130	135	137
Serum K ⁺ (meq/l)	2.7	2.8	2.9	3.2	3.6	3.7	3.3	3.5	3.7	3.1
Serum Ca ²⁺ (mg/dl)	7.6	7.8	7.7	7.8	8.2	9.0	9.1	8.6	9.0	9.1
Serum Mg ²⁺ (meq/l)	-	-	-	0.9	1.2	1.7	1.9	1.3	1.5	1.6
KCl sol.	1 amp	1amp	1amp	1 amp	1 amp	1amp	1amp	1amp	1amp	-
Ca Gluconate sol.	1 amp	1amp	1amp	1amp	1amp	-				
MgSO ₄ sol	-	-	1 amp	1 amp	1 amp	1amp	-	1amp	1amp	-

8.5-10.5 mg/dl and Mg: 1.3-2.5 meq/l.

DISCUSSION

In this case report we have described the improvement in Na,K and Ca levels after Mg supplementation in a patient suffering from renal lithiasis. The patient developed hypokalemia, hypocalcaemia and hyponatremia, the hypokalemia and hypocalcaemia did not improve on intravenous K and Ca administration. However, a resolution was achieved when the patient was administered Mg intravenously and the hypokalemic, hypocalcaemic, hyponatremic and hypomagnesaemic conditions improved.

Hypomagnesaemia is a common finding in current medical practice, mainly in critically ill patients (Guerin et al., 1996). The etiology of Mg deficiency includes gastrointestinal and renal wasting, drug induced loss, endocrine disorders, metabolic disease, redistribution of magnesium stores and other conditions (al-Ghamdi et al., 1994). Mg regulates hundreds of enzyme systems especially reactions that involve Adenosine Triphosphate (ATP), have an absolute requirement for magnesium. By regulating enzymes controlling intracellular Ca, magnesium affects smooth muscle vasoconstriction, important to the underlying pathophysiology of several critical illnesses. Mg has been directly implicated in hypocalcaemia, hypokalemia and dysrhythmias (Tong and Rude, 2005).

The patient included in this case report developed hypomagnesaemia due to inadequate intake, renal wasting, calculus formation, arrhythmia and hypertension which resulted in electrolyte imbalance. The kidneys play a major role in magnesium homeostasis and the maintenance of plasma Mg concentration. Approximately 80% of plasma Mg is ultrafilterable. Under normal circumstances, approximately 95% of filtered Mg is reabsorbed by various parts of the nephron (Cole and Quamme, 2000). Urinary Mg is an inhibitor of urinary crystal formation and some studies have shown a lower urinary excretion of Mg in patients with stones. Mg deficiency due to etiologies other than renal wasting are associated with

hypomagnesaemia and could play a role in pre-disposition to urinary calculus formation (Nijenhuis et al., 2005).

Hypomagnesaemia is recognized to cause cardiac arrhythmias. Changes in electrocardiogram findings include prolongation of conduction and slight ST depression. The ionic basis of the effect of magnesium depletion on cardiac arrhythmia may be related to the impairment of the membrane sodium-potassium pump and the increased outward movement of potassium through the potassium channels in cardiac cells, leading to shortening of the action potential and increasing susceptibility to cardiac arrhythmias. A repetitive polymorphous ventricular tachycardia with prolongation of QT interval has been reported in conjunction with hypomagnesaemia. Mg has also been suggested to play a role in blood pressure regulation. Hypertension appears to be uniformly characterized by a decrease in intracellular free Mg causing an increase in total peripheral resistance due to increased vascular tone and reactivity (Mahendra et al., 2006). Thus, the renal wasting, arrhythmias, hypertension resulted in hypomagnesaemia which contributed to electrolyte imbalance.

Hypokalemia is a common event in hypomagnesaemic patients occurring in 40 to 60% of cases (Whang and Ryder, 1990). Isolated disturbances of K balance do not produce secondary abnormalities in Mg homeostasis. In contrast, primary disturbances in Mg balance particularly Mg depletion, produce secondary K depletion. Potassium secretion from the cell of thick ascending limb and cortical collecting tubule is mediated by ATP inhibitable luminal K channels (Nicholas et al., 1994). Hypo-magnesaemia is associated with reduction in the cell magnesium concentration which may then lead to decline in ATP activity and due to removal of ATP inhibition; there is an increase in the number of open K channels. These changes would promote K secretion from the cell into the lumen and enhanced urinary losses. The hypo-kalemia in this setting is relatively refractory to K supplementation and requires correction of Mg deficit (Whang et al., 1992). Because of the inhibition of Na⁺ K⁺ - ATPase,

there occurs depletion of Na along with K. Thus, in our case report we observed that the patient was administered KCl intravenously but the condition of the patient improved temporarily till K supplementation which reverted back to hypokalemia as soon as the K supplementation was stopped. However, after Mg supplementation along with K supplementation the levels of serum K and Na started improving and finally reached the normal limits and the condition of the patient improved.

Hypocalcaemia is another common manifestation in hypomagnesaemia. Symptomatic hypocalcaemia is usually seen in moderate to severe deficiency and there is a positive correlation between serum Mg and Ca concentration. A proportion of circulating Mg is protein bound, such that only 70% of total plasma Mg is ultrafilterable (Quamme, 1997). In adults, the thick ascending limb of the loop of Henle is the main site of magnesium reabsorption. The $\text{Ca}^{2+} / \text{Mg}^{2+}$ - sensing receptor (CASR), a member of G-protein coupled receptor family, is an important regulator of magnesium homeostasis (Brown et al., 1993). In hypomagnesaemic and hypocalcaemic states, the rates of calcium and magnesium reabsorption in the loop of Henle are increased via CASR mediated stimulation of $\text{Na}^{+} \text{K}^{+} 2\text{Cl}^{-}$ cotransporter and apical Renal Outer Medulla Potassium (ROMK) channel (Brown and MacLeod, 2001). Thus, in our patient because of low calcium level, calcium was administered intravenously but the levels of Ca in serum improved temporarily till Ca supplementation which reverted back to hypocalcaemia as soon as the Ca supplementation was stopped. However, by administering Mg along with Ca intra-venously, levels of Ca in the patient started improving and finally reached the normal levels.

Thus, in the present case report, the patient suffering from renal lithiasis developed electrolyte imbalance due to hypomagnesaemia. It was observed that despite the supplementation of K and Ca in the patient, the condition of the patient did not improve but with Mg supplementation, K, Ca and Na levels in the patient improved. It may further be noted that the Mg supplementation reduced the frequency of potentially fatal ventricular arrhythmia in the patient; similar finding was also observed in a study by Mahandera et al. (Mahendra et al., 2006). Thus, the present case report has established a correlation of Mg with K, Ca and Na and proves that Mg supplementation is necessary in a critically ill patient and also Mg supplementation helps to improve the cardiac abnormalities. Therefore, the above findings should be used to identify the patients at risk for such deficiencies.

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