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Hypomagnesemia as a risk factor for the development of AKI and non-recovery of the renal function in critically ill patients with Acute Kidney Injury.

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Abstract

Background: The aim of the present study was to evaluate the role of hypomagnesemia as a risk factor for the development of acute kidney injury (AKI) and non-recovery of renal function in critically ill ICU patients.

Methods: A cohort study was conducted by collecting data from March 2021 to December 2022 on 90 patients who were admitted into an intensive care unit (ICU). Magnesium serum levels were measured on alternate days during ICU stay. Hypomagnesemia was defined as serum magnesium concentration of <0.70 mmol/L during ICU stay. AKI was defined on the criterion of The Risk, Injury, Failure, Loss and End-stage kidney disease (RIFLE). The Renal function recovery was defined as an absence of AKI by the RIFLE criteria over a 48-h period, or at ICU discharge, in the patients who developed AKI during ICU stay.

Results: The serum magnesium level was significantly lower in patients with AKI as compared to patients without AKI (0.60 ± 0.17 mmol/L in AKI versus 0.68 ± 0.18 mmol/L in no-AKI group). The serum magnesium was significantly higher in patients who recover renal function when compared with patients who did not have recovered renal function (0.68 ± 0.14 mmol/L versus 0.52 ± 0.17 mmol/L, P = 0.002).

Conclusions: Hypomagnesemia was an independent risk factor for AKI and non-recovery of renal function in a cohort of critically ill AKI patients.

Introduction

Acute kidney injury (AKI) is a common and important complication in patients admitted in the intensive care unit (ICU) which have a high mortality rate.

Hypomagnesemia is defined as a total serum magnesium concentration <0.70 mmol/L. It is a common electrolyte abnormality seen in >10% of hospitalized patients.

However the prevalence of hypomagnesemia increases to 60 to 65% in ICU patients due to multiple reasons which includes inadequate nutrition, hypo tension, hypo albuminemia, sepsis and the use of drugs like diuretics and aminoglycosides .[1]

The studies revealed that hypomagnesemia leads to decrease in the glomerular filtration rate (GFR) and the renal blood flow (RBF) which potentiates post-ischemic renal injury.[2] The studies had shown that administration of magnesium in experimental animal having ischemic renal injury results in increase in GFR and RBF and enhances renal recovery of AKI. [3]

Though the relation between hypomagnesemia and non-recovery of renal function in critical ill patients with AKI is obvious and there is paucity of literature measuring serum Magnesium level as a predictor of non-recovery of renal function in critical ill patients with AKI in Indian population, we aimed to fill this lacuna in literature.

Aim

The aim of this study was to evaluate the role of hypo magnesemia as a risk factor for the development of acute kidney injury (AKI) and non-recovery of renal function in critically ill patients.

Objectives

1. To evaluate serum magnesium level in ICU patients and see association of magnesium level with development of AKI.

2. To assess the relation of serum magnesium level with non-recovery from Acute Kidney Injury.

Methodology

- Study type: Prospective observational study
- Study place: This study was conducted in ICU of General Medicine Department of SMS Medical College and Attached group of Hospitals.
- Study period: 1 year duration till sample size was achieved and 2 months for data analysis.
- Sample size: Sample size was calculated to be 90 considering this population with expected probability of hypomagnesemia in non-recovered patients with acute kidney injury as 70% at confidence level 95%, acceptable error 10% and nonresponse rate of 10%

Inclusion criteria

1. All patient who are critically ill admitted in ICU and diagnosed by history, physical examination findings, laboratory abnormalities, ultrasound report.

2. Age ≥ 18 years.

Exclusion criteria

1. ICU stay <48 hours.

2. Age less than 18 years.

3. On admission in ICU serum creatinine >3.5mg/dl.

4. Patients refusing to give informed consent for the study.

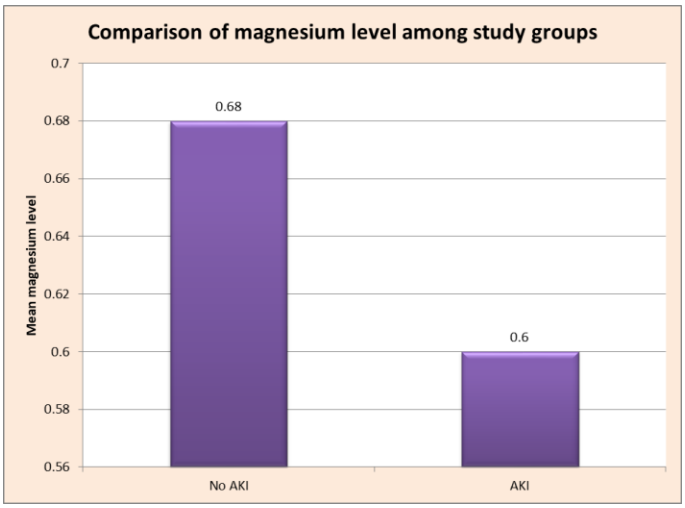
Results

In our study out of 90 patients included in study, 40 patients (44.4%) developed AKI and 50 patients (55.6) did not developed AKI.

Table 1: Comparison of magnesium level among study groups

Group	N	Mean ± SD	P value
No AKI	50	0.68 ± 0.18 mmol/L	<0.001 (S)
AKI	40	0.60 ± 0.17 mmol/L	

Graph 1:



Above table depicts there were significant differences in level of Magnesium in the two groups. The mean Mg level in AKI was significantly lower than in no-AKI group. (0.60 ± 0.17 mmol/L n AKI versus 0.68 ± 0.18 mmol/L in no-AKI group)

Table 2: Comparison of magnesium level in relation to recovery of AKI

Group	N	Mean ± SD	P value
No recovery	19	0.52 ± 0.17 mmol/L	0.002 (S)
Recovery	21	0.68 ± 0.14 mmol/L	

Comparison of magnesium level in relation to recovery of AKI Group N Mean ± SD P value No recovery 50 0.52 ± 0.17 0.002 (S) Recovery 40 0.68 ± 0.14 Above table depicts the recovery of AKI shows significant association with level of Magnesium.

The patients who recovered from AKI have significantly higher magnesium level than patients who did not recovered from AKI. (0.68 ± 0.14 in AKI Recovery group versus 0.52 ± 0.17 in no AKI Recovery group).

Graph 2:

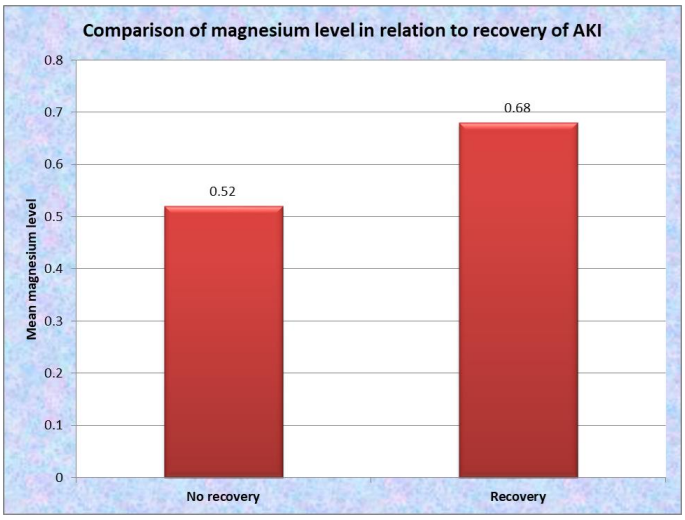
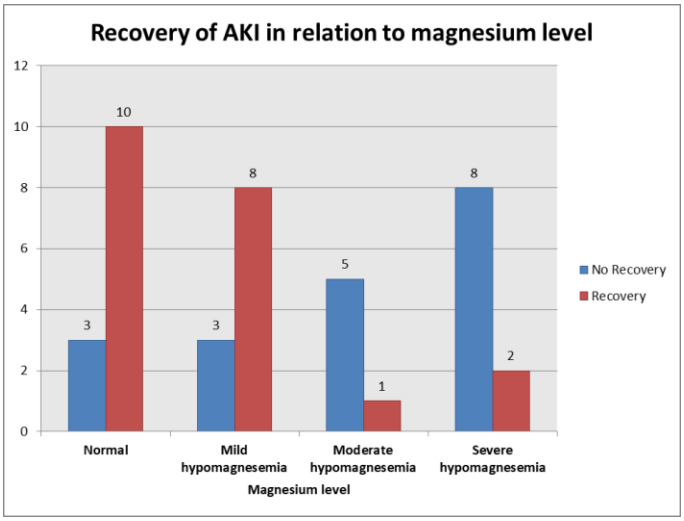


Table 3: Recovery of AKI in relation to magnesium level

Magnesium level	No Recovery		Recovery		Total
	N	%	N	%	
Normal	3	23.1	10	76.9	13
Mild hypomagnesemia	3	27.3	8	72.7	11
Moderate hypomagnesemia	5	83.3	1	16.7	6
Severe hypomagnesemia	8	80	2	20	10
Total	19	47.5	21	52.5	40
Chi - square = 12.239 with 3 degrees of freedom; P = 0.008 (S)					

Above table depicts the more patients with normal Magnesium level recovered than patients with hypo magnesemia. The number of patients recovered from AKI decreases with decreasing level of magnesium.

Graph 3:



Discussion

The most important findings of our study are the relation between low serum magnesium levels and development of AKI and the relation between low serum magnesium levels with non-recovery of AKI in critically ill patients. Our study demonstrated that hypomagnesemia was an independent risk factor for the development of AKI and non-recovery of renal function after an AKI episode in critically ill patients. Santos MS et al. in their study demonstrated a significant difference in renal function recovery between AIDS patients with and without hypomagnesemia[4].

The important causes for AKI in critically ill patients are ischemia and exposure to nephrotoxic drugs.[5] At physiological level Magnesium competes with the

calcium transport systems in the cell membrane which leads to decrease in the intracellular calcium concentrations, resulting in the relaxation of smooth muscle cells[6].

The researches revealed that hypomagnesemia potentiates the post-ischemic renal injury in rats, hypo magnesemia has negative impact on the GFR and the renal blood flow in zidovudine-treated rats.[7] The magnesium supplementation have shown to have beneficial effects in nephrotoxic drugs induced AKI experimental models.[8] The magnesium exerts it's effect by stimulating the release of nitric oxide (NO) which leads to renal vasodilation and have beneficial results on renal function. Magnesium administration induces a peripheral (primarily arteriolar) vasodilator effect, not only via an endothelium dependent release of Nitric oxide, but also through its ability to induce endothelium-independent vasodilatation by acting as a calcium channel antagonist.[9] Moreover infusion of magnesium leads to beneficial microcirculatory effects, such as an increase in red blood cell deformability, anti-inflammatory effects, a reduction of platelet aggregation, and maintenance of endothelial integrity.[10]

Conclusion- Hypomagnesemia has high prevalence in critical ill ICU patients and it is an independent risk factor for development of AKI and non-recovery of renal function among AKI patients in the ICU. Studies involving magnesium supplementation in critical ill ICU patients are warranted in order to ascertain whether hypomagnesemia is a determinant or simply a marker of critical illness.

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