ORIGINAL RESEARCH PAPER

OBSERVATIONS OF SERUM MAGNESIUM LEVEL IN RELATION TO COMPLICATIONS AND SEVERITY OF CHRONIC RENAL FAILURE

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ABSTRACT

Chronic kidney disease forms large pool of patients in the world. Cardiovascular disease is the leading cause of mortality and morbidity in patients with chronic kidney disease, which is partly explained by the fact that 40-70% of patients receiving dialysis have significant coronary artery disease. Recent clinical studies have shown that lower serum magnesium(Mg) levels are associated with vascular calcification and cardiovascular mortality among patients with end-stage renal disease(ESRD). AIMS & OBJECTIVES OF THE STUDY was to study the serum magnesium levels in chronic renal failure & to detect correlation if any of serum magnesium with clinical feature , severity of CRF on various parameters.

In this study clinical significance of serum magnesium levels in patients with CRF and its correlations with the sodium, potassium and calcium were studied.

There was significant increase in serum magnesium level in patients with CRF,Serum magnesium level increased with duration of the illness in CRF patients, Serum magnesium level was higher in the patients with bradycardia, Serum magnesium level was higher in patients with uremic encephalopathy, There was positive correlation between serum magnesium levels and blood urea, serum magnesium and serum creatinine& serum magnesium and serum potassium levels, There was negative correlation between serum magnesium and serum calcium level in CRF patients, No correlation was found between serum magnesium and serum sodium. Serum magnesium is a worthwhile tool in assessing duration of disease morbidity and mortality in patients with chronic renal failure. Its estimation may help in evaluating conservative treatment and dialysis in CRF patients.

INTRODUCTION

Chronic kidney disease forms large pool of patients in the world. Cardiovascular disease is the leading cause of mortality and morbidity in patients with chronic kidney disease, which is partly explained by the fact that 40-70% of patients receiving dialysis have significant coronary artery disease. Recent clinical studies have shown that lower serum magnesium(Mg) levels are associated with vascular calcification and cardiovascular mortality among patients with end-stage renal disease(ESRD). On the other hand hypermagnesemia inhibits parathyroid hormone secretion, which is considered an important independent risk factor for vascular calcification, left ventricular hypertrophy and mortality in ESRD patients..

Renal failure is the most common cause of hypermagnesemia, which is usually mild and asymptomatic even in ESRD patients. In CKD, until GFR falls to below 30ml/min, urinary Mg excretion may be normal or even increased. In patients with CKD on dialysis , bone Mg was increased by 66% In both cortical and trabecular bones, suggesting that dialysis patients have increased total body Mg stores.

Vitamin D has an important role in absorption of Mg in the jejunum in ESRD and healthy subjects.

Mg replacement or Mg containing agents like cathartics and antacids may cause severe or fatal hypermagnesemia even in patients without pre-existing renal dysfunction. Potential harmful effect of elevated Mg include altered nerve conduction velocity, increased pruritus , and alteration to osseous metabolism and parathyroid gland function.

The safe serum Mg level range and benefit of oral Mg supplementation for prevention and treatment of hypertension, diabetes, CVD and atherosclerosis also warrants further study.

AIMS & OBJECTIVES

1. To study the serum magnesium levels in chronic renal failure.
2. To detect correlation if any of serum magnesium with clinical feature, severity of CRF on various parameters.

MATERIALS AND METHODS

50 patients of chronic renal failure admitted in Department Of Medicine, Patna Medical College & Hospital, Patna, from opd or casualty during the period of April 2017 to July 2018 and 50 age and sex matched controls were taken for the study.

EXCLUSION CRITERIA

1) Diarrhea, Pancreatitis
2) Increased serum creatinine with no improvement for >3 months AND/OR
3) Uremic symptoms over 3 months with increased serum creatinine. AND/OR
4) Other – anemia, hyperphosphatemia, hypocalcemia.

RESULTS AND OBSERVATIONS

Fifty patients of chronic renal failure and fifty controls with matched age, sex were taken for present study.

In the present study, 50 patients of CRF were included, out of which 29 patients (58%) were male and 21 patients (42%) were females. On decade wise grouping, we found maximum number of patients between 51-60 years (22%).

Male to female ratio in the study group was 1.38:1.

Mean serum magnesium level increased with duration of illness in CRF patients which was statistically significant.

Mean serum magnesium levels in patients with bradycardia was significantly higher than in those with normal pulse rate which was statistically highly significant (<0.001).

Mean serum magnesium level in CRF patients with encephalopathy was higher compared to serum magnesium level in CRF patients without encephalopathy, which was found to be statistically highly significant (<0.001).

Mean value of urea in controls and patients showed a considerable difference, which was found to be highly significant (<0.001).

Creatinine levels in CRF patients were very high as compared to controls. This difference was statistically significant (<0.001).

INCLUSION CRITERIA

Patients with serum creatinine above 2 mg% with:
1) Abnormal finding on renal ultrasound – asymmetric kidney size, small kidney (<9cm) or large polycystic kidney. AND/OR
2) Increased serum creatinine with no improvement for >3 months AND/OR
3) Uremic symptoms over 3 months with increased serum creatinine. AND/OR
4) Other – anemia, hyperphosphatemia, hypocalcemia.
Mean serum magnesium level in CRF patients was high as compared to controls which was statistically significant (p<0.001). Positive correlation was found between serum magnesium and blood urea in CRF patients (r=+0.829 ; p<0.001) which was statistically highly significant.

Positive correlation was found between serum magnesium levels and serum creatinine in CRF patients (r=+0.873 ; p<0.001) which was statistically highly significant.

Significant positive correlation was found between serum magnesium levels and serum potassium levels in CRF patients (r=+0.861 ; t=0.05 ; p<0.001) which was statistically highly significant.

Significant negative correlation was found between serum magnesium levels and serum calcium levels in CRF patients (r= -0.857 ; t=45.02 ; p<0.001) which was statistically highly significant.

No correlation was found between serum magnesium levels and serum sodium levels in CRF patients (r=0.005 ; t=418.2 ; p>0.05).

DISCUSSION

In this study total no of 50 patients were selected. Patients age group varied between 12 to 70 years. Result were compared with result of 50 apparently healthy individuals (without chronic renal failure) with matched age and sex.

The result of the study on serum magnesium level in CRF patients showed there is significant elevation of serum magnesium level in cases as compared to controls.

In the study mean serum magnesium level in CRF patients was 3.96±0.58 mg/dl was markedly elevated as compared to control group (1.99±0.24 mg/dl) and it was statistically significant (p<0.001)

Sharma et al studied 50 patients of CRF with matched age and sex found that mean serum magnesium level were 4.10±0.85 mg/dl in cases and 2.40±0.14 mg/dl in controls which was statistically significant(p<0.001)

The present study showed that there was significant rise in serum magnesium levels in CRF patients in comparison to controls which is in concordance with previous studies done on CRF patients.

Sharma et al have suggested that raised serum magnesium level in CRF patients that was observed may be due to ingestion of antacid containing magnesium markedly increased parathyroid hormone secretion and decreased urinary excretion of magnesium.

Clarkson et al have observed low serum magnesium levels in their patients. They explained this discrepancy by decreased absorption from the gut and nausea causing diminished intake of magnesium diet.

Whether intestinal absorption of magnesium is altered is debatable. Kopple et al reported normal or near normal absorption of magnesium. Om the other hand Schemulin et al noted decreased absorption of magnesium from the gut and nausea causing diminished intake of magnesium.

In the present study serum magnesium levels increased with duration of the illness which was statistically significant (p<0.05). This is in concordance with the study done by Sharma et al. Heller and Hammerstein observed conduction defect in uremic patients even at much lower serum magnesium level.

In the present study there was a significant positive correlation between blood urea and serum magnesium & serum creatinine and serum magnesium level.

Sharma et al studied 50 patients of CRF and found positive correlation between serum magnesium and blood urea (r=+0.76, t=8.1, p<0.01) and between serum magnesium and serum creatinine(r=+0.65, t=5.9, p<0.01).

In the present study 50 patients with CRF were studied and found positive correlation between serum magnesium and blood urea (r=+0.829 , P<0.001) and between serum magnesium and serum creatinine (r=+0.873 ; p<0.001).

This study showed that there is significant positive correlation between serum magnesium and serum potassium and statistically insignificant correlation between serum magnesium and serum sodium.

Sharma et al studied 50 patients with CRF and found that there was significant positive correlation between serum magnesium and serum potassium (r=+0.40; t=3.65 ; p<0.05).

Present study was done on 50 CRF patients which showed positive correlation between serum magnesium and serum potassium (r=+0.861 ; t=0.05 ;p<0.001).

The study showed statistically insignificant correlation between serum magnesium and serum sodium.

Sharma et al studied 50 patients with CRF and found that there was no correlation between serum magnesium and serum sodium(r=0.11 ; t=0.79 ; p>0.05).

Present study was done on 50 CRF patients which showed no significant correlation between serum magnesium and serum sodium (r=0.005 ; t=418.2 ; p>0.05).

The study showed negative correlation between serum magnesium and serum calcium

Sharma et al studied 50 CRF patients and found that there was significant negative correlation between serum magnesium and serum calcium (r=-0.48, t=3.7; p<0.05).

Present study was done on 50 CRF patients which showed negative correlation between serum magnesium and serum calcium (r=-0.857 ; t=45.02 ; p<0.001).

Sharma et al reported the negative correlation was because hypermagnesemia reduces calcium reabsorption due to competition for a common reabsorption site at nephron.

The present study showed that the serum magnesium level was higher in the patients with uremic encephalopathy.

Sharma et al studied 50 CRF patients and found that mean serum magnesium level in CRF patients with uremic encephalopathy was 4.75±0.94 mg/dl as compared to those without 3.76±0.59 mg/dl.

In present study 50 CRF patients were studied and found that mean serum magnesium level in CRF patients with uremic encephalopathy was 5.17±0.06 mg/dl as compared to those without 3.88±0.49 mg/dl which was statistically significant (p<0.001)

Sharma et al showed that magnesium has direct CNS depressant action. Thus magnesium may serve as a prognostic indicator in CRF patients with uremic encephalopathy.

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SUMMARY
50 patients of chronic renal failure admitted to IPD of Dept. of Medicine, Patna Medical College & Hospital from opd or casualty during the period of April 2015 to March 2016 and 50 age and sex matched controls were taken for the study.

- Male to female ratio in study group was 1.38:1
- Mean blood urea and serum creatinine in CRF patients was 114.5±33.64mg/dl and 5.24±1.65mg/dl respectively.
- Mean blood urea and serum creatinine in control was 18.68±3.38mg/dl and 0.86±0.21mg/dl respectively.
- Mean value of serum magnesium in CRF patients and controls was 3.96±0.58mg/dl and 1.99±0.24mg/dl respectively.
- Serum magnesium level increased with duration of illness in CRF patients which was statistically significant (p<0.05)
- Mean serum magnesium level in CRF patients with bradycardia was 5.04±0.18mg/dl and in those with normal pulse rate was 4.02±0.31mg/dl.
- Serum magnesium was higher in patients with uremic encephalopathy 5.17±0.06mg/dl as compared to those without encephalopathy 3.88±0.49mg/dl which was statistically significant (p<0.001)
- There was positive correlation between serum magnesium and blood urea in CRF patients. R=+0.0829; p<0.001
- There was positive correlation between serum magnesium and serum creatinine in CRF patients. R=+0.873; p<0.001
- There was positive correlation between serum magnesium and serum potassium in CRF patients. R=+0.861 ; t=0.05 ; p<0.001
- There was negative correlation between serum magnesium and serum calcium levels in CRF patients. r=-0.857 ; t=45.02 ; p<0.001
- There was no correlation between serum magnesium and serum sodium in CRF patients. r=0.005 ; t=418.2 ; p>0.05

CONCLUSION
In this study clinical significance of serum magnesium levels in patients with CRF and its correlations with the sodium, potassium and calcium were studied

1. There was significant increase in serum magnesium level in patients with CRF.
2. Serum magnesium level increased with duration of the illness in CRF patients.
3. Serum magnesium level was higher in the patients with bradycardia.
4. Serum magnesium level was higher in patients with uremic encephalopathy.
5. There was positive correlation between serum magnesium levels and blood urea, serum magnesium and serum creatinine& serum magnesium and serum potassium levels.
6. There was negative correlation between serum magnesium and serum calcium level in CRF patients.
7. No correlation was found between serum magnesium and serum sodium

REFERENCES
9. Vernau. Magnesium metabolism. Recent advances in clinical biochemistry. chapter 3;1978