

## ORIGINAL RESEARCH

### Assessment of serum magnesium levels in chronic heart failure patients

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#### ABSTRACT

**Background:** Hypomagnesemia, hypokalaemia, and hyponatremia are the common serum electrolyte abnormalities seen in chronic congestive heart failure. The present study was conducted to assess serum magnesium levels in chronic heart failure patients.

**Materials & Methods:** 90 chronic heart failure patients of both genders were divided into two groups. Group I were patients with normal magnesium levels ( $>2\text{mEq/L}$ ) and group II patients with low magnesium levels ( $\leq 2\text{ mEq/L}$ ).

**Results:** Out of 90 patients, males were 50 and females were 40. The mean BMI in group I was  $25.1\text{ kg/m}^2$  and in group II was  $27.5\text{ kg/m}^2$ . The mean SBP was  $122.4\text{ mm Hg}$  in group I and  $134.6\text{ mm Hg}$  in group II. DBP was  $70\text{ mm Hg}$  in group I and  $82.4\text{ mm Hg}$  in group II. There were 8 diabetes in group I and 21 in group II. The difference was significant ( $P < 0.05$ ). The mean LVEF in group I was  $35.6\%$  and in group II was  $36.7\%$ . The serum potassium was  $4.3\text{ mEq/L}$  in group I and  $4.1\text{ mEq/L}$  in group II. Serum creatinine was  $1.8\text{ mg/dL}$  in group I and  $1.2\text{ mg/dL}$  in group II. The difference was significant ( $P < 0.05$ ).

**Conclusion:** Chronic heart failure patients had low serum magnesium level. Low serum magnesium levels were predictor of deranged cardiac & biochemical profile seen in chronic heart failure patients.

**Key words:** magnesium, chronic heart failure, creatinine

#### INTRODUCTION

Hypomagnesemia, hypokalaemia, and hyponatremia are the common serum electrolyte abnormalities seen in chronic congestive heart failure. Among these, the two predominantly intracellular cations, magnesium and potassium, may contribute to the high mortality and sudden death associated with congestive heart failure.<sup>1</sup> Deficiencies in these two cations occur commonly in heart failure as a consequence of reduced intake or of increased losses. The former develops in response to consequences of the underlying disease, includes reduction in caloric intake and raise bowel edema. The losses are typically associated with heart failure therapy.<sup>2</sup>

In heart failure (HF) patients, factors like hyperactivity of the renin-angiotensin system (RAS), influence of drug therapy (loop and thiazide diuretics), undernutrition, and others, can causes hypokalemia and hypomagnesemia.<sup>3</sup> These conditions are well known to increase the risk of arrhythmia and sudden death.<sup>4</sup> When diuretics in the treatment of HF,

hypomagnesemia can lead to complications, which complicates arrhythmia and causes refractory hypokalemia; thus, HF patients with serum magnesium (sMg) concentration levels should be determined. Hypomagnesemia has been reported to be an independent risk factor for cardiovascular disease, and replacement therapy is considered necessary in terms of long-term prognosis.<sup>5</sup> The present study was conducted to assess serum magnesium levels in chronic heart failure patients.

## MATERIALS & METHODS

The present study comprised of 90 chronic heart failure patients of both genders. Written consent was taken from all for the participation in the study.

Data such as name, age, gender etc. was recorded. After that, Blood samples were obtained, serum magnesium levels were assessed. On the basis of magnesium levels, all the patients were divided into two groups. Group I were patients with normal magnesium levels ( $>2\text{mEq/L}$ ) and group II patients with low magnesium levels ( $\leq 2\text{mEq/L}$ ). Parameters such as blood pressure, LVEF (%), serum potassium (mEq/L) and serum creatinine (mg/dL) was recorded in all groups. Thus, data that obtained were subjected to statistical analysis. P value  $< 0.05$  was considered significant.

## RESULTS

**Table I Distribution of patients**

Total- 90		
Gender	Males	Females
Number	50	40

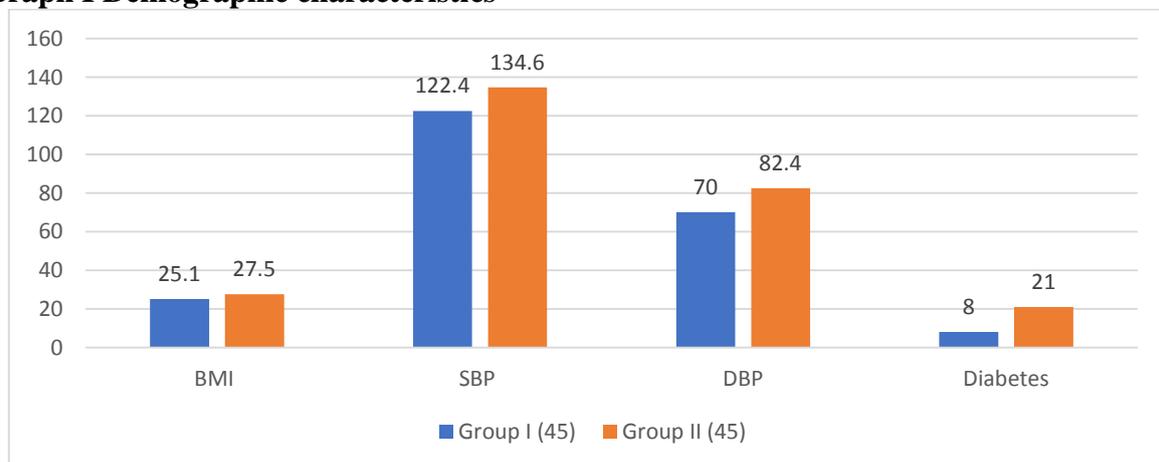
Table I shows that out of 90 patients, males were 50 and females were 40.

**Table II Demographic characteristics**

Parameters	Group I (45)	Group II (45)	P value
BMI	25.1	27.5	0.05
SBP	122.4	134.6	0.04
DBP	70	82.4	0.01
Diabetes	8	21	0.01

Table II, graph I shows that the mean BMI in group I was  $25.1\text{ kg/m}^2$  and in group II was  $27.5\text{ kg/m}^2$ . The mean SBP was 122.4 mm Hg in group I and 134.6 mm Hg in group II. DBP was 70 mm Hg in group I and 82.4 mm Hg in group II. There were 8 diabetes in group I and 21 in group II. The difference was significant ( $P < 0.05$ ).

**Graph I Demographic characteristics**



**Table III Assessment of parameters**

Parameters	Group I	Group II	P value
LVEF (%)	35.6%	36.7%	0.05
Serum potassium (mEq/L)	4.3	4.1	0.92
Serum creatinine (mg/dL)	1.8	1.2	0.01

Table III shows that mean LVEF in group I was 35.6% and in group II was 36.7%. The serum potassium was 4.3 mEq/L in group I and 4.1 mEq/L in group II. Serum creatinine was 1.8 mg/dL in group I and 1.2 mg/dL in group II. The difference was significant ( $P < 0.05$ ).

## DISCUSSION

Magnesium is the second in abundance intracellular ion. The relationship between magnesium and cardiovascular system, arterial hypertension, central nervous system, skeletal muscles and pregnancy, is an already established knowledge.<sup>6</sup> The main part of total body magnesium is concentrated in the bones, only 1% is in the serum while the 31% is in the intracellular space diluted in the cytoplasm or conjuncted to enzymes or ATP.<sup>7</sup> Normal values of serum magnesium are considered those between 0.75 and 1.5 mmol/L. Values below the threshold of 0.75 mmol/L are defined as hypomagnesemia.<sup>8</sup> The present study was conducted to assess serum magnesium levels in chronic heart failure patients.

We found that mean BMI in group I was 25.1 kg/m<sup>2</sup> and in group II was 27.5 kg/m<sup>2</sup>. The mean SBP was 122.4 mm Hg in group I and 134.6 mm Hg in group II. DBP was 70 mm Hg in group I and 82.4 mm Hg in group II. There were 8 diabetes in group I and 21 in group II. Whang et al<sup>9</sup> 26 reported that 19% of patients receiving digitalis therapy had a serum magnesium level <1.25 mEq/L. The variable frequency of hypomagnesemia in these reports maybe attributable to differences in the severity of heart failure, the degree of congestion, the dosages of loop diuretic agents, and the degree of neurohormonal activation.

We found that mean LVEF in group I was 35.6% and in group II was 36.7%. The serum potassium was 4.3 mEq/L in group I and 4.1 mEq/L in group II. Serum creatinine was 1.8 mg/dL in group I and 1.2 mg/dL in group II. Smetana et al<sup>10</sup> investigated the effects of angiotensin-converting-enzyme (ACE) inhibitor therapy on 66 patients with congestive heart failure resulting from dilated cardiomyopathy and coronary artery disease. They unearth that these drugs often ameliorate the electrolyte abnormalities in congestive heart failure, likely as of their ability in blocking the renin angiotensin aldosterone system.

Strong evidence has collected to implicate magnesium deficiency as an integral cause of digitalis-toxic associated arrhythmias. Models like dog was found hypomagnesemia to facilitate the induction of digitalis-toxic arrhythmias, most of which were terminated with intravenous magnesium. Even the heart rhythm of dogs with digitalis-toxic ventricular arrhythmias and normal serum magnesium levels converted to sinus rhythm with intravenous magnesium therapy; furthermore, pre-treatment of these dogs with magnesium concluded in raised dose of digoxin necessary to induce the arrhythmias. Lack of magnesium potentiates digitalis toxicity by two valuable mechanisms: it works synergistically with digoxin to suppress sodium-potassium ATPase activity, it & exacerbates the problem of refractory potassium repletion in concurrent magnesium and lack of potassium.<sup>11</sup>

Ceremuzyński et al<sup>12</sup> assessed the role of electrolyte imbalance in cardiac arrhythmias associated with congestive heart failure in 68 patients. On admission, hypomagnesemia was found in 38% and excessive magnesium loss in 72% of patients. Serum magnesium levels were lower and urine magnesium excretion was greater in patients with complex ventricular arrhythmias, both on admission and after treatment for heart failure. Intravenous administration of magnesium caused a significant decrease in the number of ventricular ectopic beats, couplets and episodes of non-sustained ventricular tachycardia.

Hypomagnesemia, probably related to increased urine magnesium excretion, is an essential feature of heart failure associated with complex ventricular arrhythmias.

The limitation the study is small sample size.

## CONCLUSION

Authors found that chronic heart failure patients had low serum magnesium level. Low serum magnesium levels were predictor of deranged cardiac and biochemical profile in chronic heart failure patients.

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