Evaluation of serum electrolytes in Ischemic Heart Disease patients.

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Background: Electrolyte abnormalities in cardiovascular emergencies are widely studied all over the world as they are mostly found to be associated with cardiovascular morbidity and mortality. Hypokalemia and hypomagnesemia are associated with risk of sudden death.

Aim and Objective: To evaluate serum levels of magnesium, sodium, potassium, chloride and bicarbonate in patients with Ischemic Heart Disease (IHD).

Materials and Methods: The study group comprises of 30 Ischemic Heart Disease Patients (Group 2) and 30 healthy volunteers (Group 1) who were age sex matched. Serum electrolytes namely sodium, potassium and chloride were estimated using ISE electrolyte analyser. Serum magnesium and bicarbonate were estimated by semiautoanalyser.

Statistical Analysis: Statistical analysis was done using SPSS Version 16.

Results: There is significant reduction (p = 0.000) in serum magnesium levels in IHD patients of Group 2 when compared to Group 1 controls. The serum potassium levels were significantly low (p= 0.001) in group 2 patients from that of their healthy counterparts. There is also a decrease (p = 0.027) in serum bicarbonate levels in IHD patients compared to controls. There was no variation in serum sodium and chloride levels among the 2 groups.

Conclusion: Statistically significant reduction in serum magnesium levels in IHD has to be viewed with caution as it has an important role in the pathogenesis of atherosclerosis. Hypokalemia is an independent risk factor for heart failure. Serum potassium and bicarbonate though decreased in the IHD patients are still in the low normal range.

Keywords: Ischemic heart disease, Renal outer medullary channel, Na-K-ATPase.

INTRODUCTION:

Life threatening electrolyte abnormalities have been known to affect the prognosis and outcome of the disease status, in different clinical settings. Alterations in the levels of serum electrolytes have also been associated with increased cardiovascular morbidity and mortality. Among the electrolytes, serum Mg has been known to influence endothelial function, inflammation, blood pressure and Diabetes but a direct relation with Coronary Heart Disease risk has not been established. Serum and dietary Mg have been inversely associated with Coronary heart disease risk in some studies, but others have not observed the relationship. Intracellular and extracellular K balance determines the excitability of nerve and muscles, including the myocardium. Hypokalemia have been reported in patients with Acute Myocardial Ischemia. It is also known to increase the risk of arrhythmias. Hyperkalemia is most commonly seen in end stage renal disease (ESRD). Sodium balance is essential to maintain plasma osmolality and blood pressure which are all related to normal cardiac homeostasis.
Maintenance of electrolyte balance could be very important in the management of patients with IHD and prevention of its complications. So we chose to assess the changes in serum electrolyte levels namely Mg, K, Na, Cl, and HCO3 in patients with clinically diagnosed IHD. Understanding the underlying electrolyte abnormalities could be of help in framing treatment protocols to correct electrolyte abnormalities at a very early stage.

MATERIALS AND METHODS:
Study Design: Case Control Study
Sampling: Non random sampling is followed. We fixed 30 numbers for the study group and 30 numbers for the control group.
Study group:
A comparative study was conducted. 60 individuals were included in the study. Subjects in the age group of 35 to 75 years were taken up for the study. We divided them into 2 groups. 30 patients admitted to the Cardiac Intensive Care Unit of VIMS hospital, Salem with complaints of chest pain formed the study group 2. They were clinically diagnosed to have ischemic heart disease and supported by ECG findings. 30 age sex matched volunteers formed group 1.
Patients with renal diseases, having serum creatinine of more than 3mg/dl, severe hypoalbuminemia with serum albumin less than 2 gms/dl were excluded from the study.
Institutional Ethical Committee clearance was obtained. Subjects history and details were taken according to the standard proforma. Informed consent was obtained after explaining the study protocol to the patients attenders.
Sample collection: 4ml of venous blood was collected in clot activator tubes from all the participants taking part in the study.

BIOCHEMICAL METHODS:
Serum electrolytes namely sodium (Na), potassium (K) and chloride (Cl) levels were estimated using the electrolyte analyser (ISE) while serum magnesium was analysed by Xylydyl Blue method using semiautoanalysers Photometer 5010. Bicarbonate levels were estimated using the semiautoanalyser.

RESULTS:
There is significant reduction (p = 0.000) in serum magnesium levels in IHD patients of group 2 (2.3±0.48 mg/dl) when compared to group 1 controls (2.7±0.33 mg/dl) as per table 1. But we noticed a significant decrease (p = 0.001) in serum potassium levels among the group 2 patients (3.98 ± 0.38 mEq/L) from that of their healthy counterparts (4.29 ± 0.3 mEq/L). There is also a decrease (p = 0.027) in serum bicarbonate levels in IHD patients (23.33 ± 2.06 mEq/L) compared to the control group (24.53 ± 2.03 mEq/L ). Serum sodium and chloride levels did not vary among the 2 groups.

DISCUSSION:
We have observed significant reduction in serum Mg levels among the IHD patients compared to healthy individuals in the control group. This finding is in accordance with few studies which have also shown lower Mg levels in IHD. According to Arcand et al, intake of inadequate dietary Mg correlated with the occurrence of heart failure in more than 50% of the patients.[9] In a study conducted by Stevanovic among the Siberian population revealed the association between intake of dietary Mg and reduced risk of Coronary heart disease.[10] Studies by Al-Delaimy suggest that Mg intake has inverse association with the risk of CHD.[11] While some studies could not identify any link between coronary heart disease and Mg deficiency.[12,13]
Mg supplementation have been found to improve myocardial metabolism, inhibits calcium accumulation and death of cardiac myocytes,
improves peripheral vascular resistance, reduces cardiac arrhythmias and improves lipid metabolism. Mg protects the body from the action of oxygen derived free radicals. It improves endothelial function and inhibits platelet aggregation and adhesion.[14] Some studies have shown that intake of dietary mg helped to lower blood triglyceride level and increase HDL levels.[15]

Mg has an important role in the maintenance of cardiovascular homeostasis. Several mechanisms have been proposed for the role played by Mg in the pathogenesis of IHD. Hypomagnesemia could also be the result of serious ischemia.[16] Mg deficiency is associated with oxidative stress, cytokine synthesis, nitrogen oxides, inflammatory mediators and adhesion molecules on microvascular endothelial cells which are all known risk factors for cardiovascular disease (CVD) and atherogenesis.[17,18] Mg deficiency causes hyperreactivity of coronary arteries to vasoconstrictive stimuli. The effects of magnesium on cardiac electrophysiology is by impairment of Na-K ATPase, for which Mg is an essential cofactor. Hypomagnesemia should be corrected in IHD patients for the prevention of arrhythmias.

We have observed significantly low serum potassium levels in patients with ischemic heart disease compared to the healthy controls. Cardiovascular diseases are known to be associated with hypokalemia and K depletion in the heart, for some time.[8,19] Increased dietary intake of potassium is found to exert a protective effect against stroke and might also reduce the incidence of CVD.

Mg and K metabolism are closely linked. Mg deficiency is closely associated with hypokalemia.[20] Hypomagnesemia causes hypokalemia by various mechanisms involving Na-K ATPase and renal outer medullary potassium (ROMK) channels in the kidneys as shown in figure 1. Mg deficiency results in insufficient action of Na-K pump causing increase in intracellular sodium and depletion of intracellular potassium levels.[21] The cells are unable to retain the difference in K between the intracellular & extracellular space, resulting in intracellular potassium depletion.[22] If this decreased cellular uptake of K, occurs along with increased urinary or gastrointestinal excretion, it would lead to K wasting and hypokalemia.[23] Mg deficiency is also associated with enhanced renal excretion of K, resulting in coexistent hypokalemia.

Mg deficiency affects K homeostasis by blocking K channels in a voltage-dependent manner. Normally K is freely filtered by the glomerulus and reabsorbed by the proximal tubule and the loop of Henle. Urinary excretion of K is mainly due to the secretion that occurs at the distal tubules and collecting ducts. In the distal tubules and cortical collecting ducts, K is taken up into the cells across the basolateral membrane via Na-k-ATPases and secreted into the luminal fluid via the apical K channels- ROMK and maxi-K channels. ROMK channels are inward rectifying potassium channels responsible for basal K secretion and are present in the apical (luminal ) membrane of the distal nephrons.[24]
Mg does not affect the reabsorption of K into the renal tubular cells. But it affects K secretion through ROMK channels in the distal nephrons. Intracellular Mg binds and blocks the pore of the ROMK channel from inside, thereby limiting the secretion of K into the renal tubular fluid (inward rectification) as shown in figure 2. Thus intracellular magnesium levels would significantly determine the ROMK – mediated potassium secretion, in the distal nephrons. Hence in Mg deficiency, there is K loss in urine resulting in hypokalemia.

CONCLUSION:
We have analysed electrolyte levels namely Na, K, Mg, bicarbonate in IHD patients. We have observed significant changes in serum magnesium and potassium levels. There is no significant change in sodium and chloride levels. Low serum Mg could in turn cause hypokalemia by various mechanisms. A constant check on the serum electrolyte levels in IHD patients, could improve the prognosis of patients with ischemic heart disease. Understanding these electrolyte changes is very important as they are commonly associated with cardiovascular emergencies leading on to cardiac arrest. Therapeutic strategies to initiate treatment of electrolyte disorders at an early stage could help to prevent the occurrence of adverse cardiac outcomes.

REFERENCES:


TABLE 1: Comparison of serum electrolytes between Ischemic heart disease patients and controls.

<table>
<thead>
<tr>
<th>PARAMETER</th>
<th>Mean ± SD</th>
<th>Mean ± SD</th>
<th>P-VALUE</th>
</tr>
</thead>
<tbody>
<tr>
<td>1 Magnesium mg/dl</td>
<td>2.7 ± 0.33</td>
<td>2.3 ± 0.48</td>
<td>0.000*</td>
</tr>
<tr>
<td>2 Sodium mEq l</td>
<td>138.73 ± 2.12</td>
<td>138.26 ± 2.35</td>
<td>0.359</td>
</tr>
<tr>
<td>3 Potassium mEq/l</td>
<td>4.29 ± 0.3</td>
<td>3.98 ± 0.38</td>
<td>0.001*</td>
</tr>
<tr>
<td>4 Bicarbonate mEq/l</td>
<td>24.53 ± 2.03</td>
<td>23.33 ± 2.06</td>
<td>0.002*</td>
</tr>
<tr>
<td>5 Chloride mEq/l</td>
<td>99.23 ± 2.71</td>
<td>100.97 ± 5.68</td>
<td>0.137</td>
</tr>
</tbody>
</table>

* - P<0.05- highly significant