A CLINICAL STUDY OF SERUM ELECTROLYTES (NA, K, CL) AND SERUM MAGNESIUM LEVELS IN PATIENTS OF ACUTE MYOCARDIAL INFARCTION
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ABSTRACT

BACKGROUND
Myocardial Infarction (MI) is the term used to describe a state of myocardial necrosis secondary to an acute interruption of the coronary blood supply.¹ It is one of the manifestations of coronary heart disease leading to morbidity and mortality.² World Health Organization (WHO) has declared cardiovascular disease as a modern epidemic.³ Most of the myocardial infarctions result due to disruption in the vascular endothelium associated with atherosclerotic plaque, which in turn stimulates the formation of an intracoronary thrombus, which further leads to occlusion of coronary artery blood flow, if this occlusion persists for more than 20 minutes can result in irreversible myocardial cell damage and even cell death. Severity of the condition is dependent on three factors- the level of occlusion, length of time of occlusion and presence or absence of collateral circulation. Rupturing of the plaque causes complete coronary occlusion, which usually results in STEMI. This arises most often from a plaque that previously caused less than 50% lumen occlusion.⁴ Clinical diagnosis as well as diagnostic classification is commonly based on electrocardiographic findings to differentiate between the two types of MI. There are mainly two types, STEMI and NSTEMI. Complications of acute MI are many, which in turn leads to high incidences of mortality, but among all arrhythmias, cardiogenic shock and heart failure are found to be commonly associated with it along with electrolyte disturbances.

The aim of the study is to observe the prevalence of various electrolyte (Na, K, Cl and Mg) imbalances along with complication of cardiogenic shock, arrhythmias and heart failure in the patients of acute myocardial infarction.

MATERIALS AND METHODS
This is a prospective study in which the 100 patient admitted with signs and symptoms of acute myocardial infarction diagnosed clinically both males and females were selected over 1 year. Patients presented with symptoms of AMI within 48 hours of onset with history of chest discomfort, ECG changes of acute myocardial infarction and rise of cardiac enzymes.

RESULTS
In this study, mean age of male patients 54.04 ± 11.49 and female patients 60.30 ± 11.78. The serum magnesium, sodium and potassium levels were significantly lower in the AMI patients at baseline and gradually becomes near normal on 4th day.

CONCLUSION
So, estimation of serum magnesium, sodium and potassium levels can help to assess prognosis in AMI patients.

KEYWORDS
Serum Electrolytes, Serum Magnesium, Myocardial Infarction, Potassium Homeostasis, Hypertension.

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BACKGROUND
Myocardial Infarction (MI) is the term used to describe a state of myocardial necrosis secondary to an acute interruption of the coronary blood supply.¹ It is one of the manifestations of coronary heart disease leading to morbidity and mortality.² World Health Organization (WHO) has declared cardiovascular disease as a modern epidemic.³ Most of the myocardial infarctions result due to disruption in the vascular endothelium associated with atherosclerotic plaque, which intern stimulates the formation of an intracoronary thrombus, which further leads to occlusion of coronary artery blood flow, if this occlusion persists for more than 20 minutes can results in irreversible myocardial cell damage and even cell death. Severity of the condition is dependent on three factors- the level of occlusion, length of time of occlusion and presence or absence of collateral circulation. Rupturing of the plaque causes complete coronary occlusion, which usually results in STEMI. This arises most often from a plaque that previously caused less than 50% lumen occlusion.⁴ Clinical diagnosis as well as diagnostic classification is commonly based on

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that magnesium has a vital role in ventricular fibrillation, which causes sudden death in IHD. The coronary vasospasm resulting from magnesium deficiency has been suggested as another important factor in the sudden death of IHD. Magnesium deficiency was also postulated to have role in the genesis of atheromatous plaques in that it leads to hyperlipidaemia. Also, myocardial infarction is one of the common causes of death at present where prognosis depends on multiple factor of which many still remain unexplained. This study is designed to know the relationship between serum electrolyte levels (Na, K, Cl, Mg) and patients with acute myocardial infarction and with its complications.

MATERIALS AND METHODS
This is a prospective study in which the patient admitted with signs and symptoms of acute myocardial infarction diagnosed clinically, both males and females, in C. U. Shah Medical College and Hospital, Surendranagar, over a period of August 2014 to August 2015. 100 cases were selected over 1 year.

Inclusion Criteria- Patients presented with symptoms of AMI within 48 hours of onset. History of chest discomfort. ECG changes of acute myocardial infarction and rise of cardiac enzymes.

Exclusion Criteria- History of chronic ischaemic heart disease and patients with chronic kidney disease. Blood sample cases selected were subjected to a detailed history and thorough physical examination, routine investigation like haemoglobin, blood count, urine examination, blood sugar, serum creatinine, serum electrolytes and cardiac enzymes was performed in cases. The baseline data was taken of the patients who fulfilled the selection criteria. The purpose and procedure of the study was explained to the patients and consent was taken from all patients. Prior to the study, blood sample of the subjects was taken on the day one of admission and on day 5th. In the study, SPSS 16 was used to analyse the data. The descriptive analysis was done to find mean and standard deviation.

RESULTS
Among 100 subjects, 70 males and 30 females, the mean age of male patients was 54.04 ± 11.49 and female patients was 60.30 ± 11.78.

### Table 1. Intra Comparison of Day 1 and Day 5 of Na, K, CL and Mg in Hypertension in Both Genders

<table>
<thead>
<tr>
<th>Gender</th>
<th>Duration</th>
<th>Mean</th>
<th>SD</th>
<th>P Value</th>
<th>Mean</th>
<th>SD</th>
<th>P Value</th>
<th>Mean</th>
<th>SD</th>
<th>P Value</th>
<th>Mean</th>
<th>SD</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male (hypertensive)</td>
<td>Pre (Day 1)</td>
<td>138.9</td>
<td>3.25</td>
<td>0.147</td>
<td>4.08</td>
<td>0.80</td>
<td>0.213</td>
<td>96.7</td>
<td>4.82</td>
<td>0.923</td>
<td>2.10</td>
<td>0.20</td>
<td>0.000</td>
</tr>
<tr>
<td></td>
<td>Post (Day 5)</td>
<td>140.8</td>
<td>0.98</td>
<td></td>
<td>4.32</td>
<td>0.36</td>
<td></td>
<td>96.8</td>
<td>2.97</td>
<td></td>
<td>2.55</td>
<td>0.23</td>
<td></td>
</tr>
<tr>
<td>Male (non-hypertensive)</td>
<td>Pre (Day 1)</td>
<td>138.5</td>
<td>3.51</td>
<td>0.383</td>
<td>3.63</td>
<td>0.39</td>
<td>0.003</td>
<td>98.1</td>
<td>1.81</td>
<td></td>
<td>0.001</td>
<td>0.17</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post (Day 5)</td>
<td>140.7</td>
<td>0.78</td>
<td></td>
<td>4.04</td>
<td>0.45</td>
<td></td>
<td>99.3</td>
<td>1.47</td>
<td></td>
<td>2.49</td>
<td>0.22</td>
<td></td>
</tr>
<tr>
<td>Female (with hypertension)</td>
<td>Pre (Day 1)</td>
<td>138.6</td>
<td>3.62</td>
<td>0.051</td>
<td>4.24</td>
<td>0.96</td>
<td>0.585</td>
<td>98.2</td>
<td>2.68</td>
<td></td>
<td>0.018</td>
<td>0.26</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post (Day 5)</td>
<td>140.34</td>
<td>0.98</td>
<td></td>
<td>4.11</td>
<td>0.49</td>
<td></td>
<td>99.0</td>
<td>2.65</td>
<td></td>
<td>2.29</td>
<td>0.39</td>
<td></td>
</tr>
<tr>
<td>Female (without hypertension)</td>
<td>Pre (Day 1)</td>
<td>139.7</td>
<td>1.45</td>
<td>0.002</td>
<td>3.76</td>
<td>0.43</td>
<td>0.000</td>
<td>99.7</td>
<td>3.87</td>
<td></td>
<td>0.228</td>
<td>0.00</td>
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</tr>
<tr>
<td></td>
<td>Post (Day 5)</td>
<td>140.4</td>
<td>1.29</td>
<td></td>
<td>4.26</td>
<td>0.54</td>
<td></td>
<td>98.6</td>
<td>2.85</td>
<td></td>
<td>2.48</td>
<td>0.24</td>
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</tr>
</tbody>
</table>

According to parametric paired t-test, p-value show significant difference between pre and post values on day 1 and day 5 in both groups except in p-value for CL showing no significant difference between pre and post values of CL of day 1 and day 5 in male hypertensive, p-value for Na, K there is no significant difference between pre and post values of NA, K of day 1 and day 5 in females with hypertension and also p-value for CL is 0.228 showing that there is no significant difference between pre and post values of CL of day 1 and day 5 in females without hypertension.

### Table 2. Intra Comparison of Day 1 and Day 5 of Na, K, CL and Mg in Diabetes in Both Genders

<table>
<thead>
<tr>
<th>Gender</th>
<th>Duration</th>
<th>Mean</th>
<th>SD</th>
<th>P Value</th>
<th>Mean</th>
<th>SD</th>
<th>P Value</th>
<th>Mean</th>
<th>SD</th>
<th>P Value</th>
<th>Mean</th>
<th>SD</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male (diabetic)</td>
<td>Pre (Day 1)</td>
<td>136.9</td>
<td>2.88</td>
<td>0.025</td>
<td>4.43</td>
<td>0.75</td>
<td>0.826</td>
<td>98.3</td>
<td>5.71</td>
<td></td>
<td>0.550</td>
<td>0.36</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post (Day 5)</td>
<td>139.8</td>
<td>0.28</td>
<td></td>
<td>4.37</td>
<td>0.16</td>
<td></td>
<td>99.0</td>
<td>3.69</td>
<td></td>
<td>2.35</td>
<td>0.45</td>
<td></td>
</tr>
<tr>
<td>Male (without diabetic)</td>
<td>Pre (Day 1)</td>
<td>139.09</td>
<td>2.95</td>
<td>0.210</td>
<td>3.58</td>
<td>0.65</td>
<td>0.001</td>
<td>99.7</td>
<td>4.03</td>
<td></td>
<td>0.755</td>
<td>1.98</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post (Day 5)</td>
<td>140.9</td>
<td>0.95</td>
<td></td>
<td>4.05</td>
<td>0.45</td>
<td></td>
<td>99.9</td>
<td>2.37</td>
<td></td>
<td>2.45</td>
<td>0.24</td>
<td></td>
</tr>
<tr>
<td>Female (diabetic)</td>
<td>Pre (Day 1)</td>
<td>139.56</td>
<td>3.51</td>
<td>0.239</td>
<td>4.07</td>
<td>0.69</td>
<td>0.220</td>
<td>97.8</td>
<td>4.12</td>
<td></td>
<td>0.755</td>
<td>1.83</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post (Day 5)</td>
<td>140.45</td>
<td>1.18</td>
<td></td>
<td>4.33</td>
<td>0.37</td>
<td></td>
<td>98.05</td>
<td>2.90</td>
<td></td>
<td>2.33</td>
<td>0.31</td>
<td></td>
</tr>
<tr>
<td>Female (without diabetic)</td>
<td>Pre (Day 1)</td>
<td>139.1</td>
<td>1.39</td>
<td>0.000</td>
<td>3.66</td>
<td>0.39</td>
<td>0.001</td>
<td>98.25</td>
<td>2.42</td>
<td></td>
<td>0.002</td>
<td>1.98</td>
<td></td>
</tr>
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<td></td>
<td>Post (Day 5)</td>
<td>140.1</td>
<td>1.14</td>
<td></td>
<td>4.06</td>
<td>0.52</td>
<td></td>
<td>99.74</td>
<td>2.88</td>
<td></td>
<td>2.40</td>
<td>0.28</td>
<td></td>
</tr>
</tbody>
</table>

In males, K, CL show no significant difference between pre and post.
NA, CL has no significant difference between pre and post values of NA, CL of day 1 and day 5 in male non-diabetics.
NA, K, CL show no significant difference between pre and post values of NA, K, CL of day 1 and day 5 in females with diabetes.
NA, K, CL, Mg show significant difference between pre and post values of NA, K, CL, Mg on day 1 and day 5 in female without diabetes.

### Table 3. Intra Comparison of Day 1 and Day 5 of Na, K, CL and Mg in Cardiac Failure in Both Genders

<table>
<thead>
<tr>
<th>Gender</th>
<th>Duration</th>
<th>Mean</th>
<th>SD</th>
<th>P Value</th>
<th>Mean</th>
<th>SD</th>
<th>P Value</th>
<th>Mean</th>
<th>SD</th>
<th>P Value</th>
<th>Mean</th>
<th>SD</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male (cardiac failure)</td>
<td>Pre (Day 1)</td>
<td>135.5</td>
<td>4.48</td>
<td>0.004</td>
<td>4.08</td>
<td>0.89</td>
<td>0.335</td>
<td>98.4</td>
<td>5.04</td>
<td></td>
<td>0.061</td>
<td>1.95</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post (Day 5)</td>
<td>140.4</td>
<td>1.10</td>
<td></td>
<td>4.27</td>
<td>0.43</td>
<td></td>
<td>99.9</td>
<td>2.76</td>
<td></td>
<td>2.35</td>
<td>0.33</td>
<td></td>
</tr>
<tr>
<td>Male (without cardiac failure)</td>
<td>Pre (Day 1)</td>
<td>139.6</td>
<td>0.92</td>
<td>0.001</td>
<td>3.68</td>
<td>0.39</td>
<td>0.001</td>
<td>98.2</td>
<td>1.02</td>
<td></td>
<td>0.010</td>
<td>2.06</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post (Day 5)</td>
<td>139.3</td>
<td>0.67</td>
<td></td>
<td>4.08</td>
<td>0.44</td>
<td></td>
<td>99.0</td>
<td>1.03</td>
<td></td>
<td>2.53</td>
<td>0.21</td>
<td></td>
</tr>
<tr>
<td>Female (cardiac failure)</td>
<td>Pre (Day 1)</td>
<td>137.67</td>
<td>2.66</td>
<td>0.010</td>
<td>3.90</td>
<td>0.88</td>
<td>0.486</td>
<td>99.82</td>
<td>3.24</td>
<td></td>
<td>0.037</td>
<td>1.82</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post (Day 5)</td>
<td>140.9</td>
<td>0.98</td>
<td></td>
<td>4.02</td>
<td>0.60</td>
<td></td>
<td>97.04</td>
<td>3.26</td>
<td></td>
<td>2.23</td>
<td>0.33</td>
<td></td>
</tr>
<tr>
<td>Female (without cardiac failure)</td>
<td>Pre(Day1)</td>
<td>139.5</td>
<td>1.31</td>
<td>0.011</td>
<td>3.75</td>
<td>0.18</td>
<td>0.000</td>
<td>98.09</td>
<td>3.42</td>
<td></td>
<td>0.227</td>
<td>2.04</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Post(Day5)</td>
<td>141.67</td>
<td>1.05</td>
<td></td>
<td>4.29</td>
<td>0.29</td>
<td></td>
<td>99.08</td>
<td>2.64</td>
<td></td>
<td>2.55</td>
<td>0.23</td>
<td></td>
</tr>
</tbody>
</table>

Paired t-test, p-value for K, CL show no significant difference between pre and post values of K, CL of day 1 and day 5 in males with cardiac failure.
K has no significant difference between pre and post values of K on day 1 and day 5 in females with cardiac failure.
CL has no significant difference between pre and post values of CL on day 1 and day 5 in females without cardiac failure.
P value for K show significant difference between pre and post values of K of day 1 and day 5 in males with cardiogenic shock. NA show no significant difference between pre and post values of NA of day 1 and day 5 in males without cardiogenic shock.

K, Cl, Mg are not significant between pre and post values of K, Cl, Mg on day 1 and day 5 in females with cardiogenic shock.

Cl show no significant difference between pre and post values of Cl on day 1 and day 5 in females without cardiogenic shock.

**DISCUSSION**

Myocardial Infarction (MI) is the irreversible necrosis of heart muscle secondary to prolonged ischaemia. The occurrence of myocardial infarction was more in males as compared to females in our present study. The predilection of acute myocardial infarction for male sex in present study is in agreement with the studies by Dyckner T et al and Erik. In the present study, the serum magnesium, sodium and potassium levels were significantly lower in the AMI patients, which was similar to those, which were seen in other studies. Our study also supported the hypothesis that the alteration in the electrolyte levels could prompt the pathological events in coronary heart diseases. Fall in serum electrolyte levels on day of admission in AMI is similar to finding was observed by Shah et al who reported hyponatraemia on day first. Flear and Hilton reported a progressive fall in the mean daily serum sodium concentration until day 4 and rise thereafter in all cases. Significant decrease in serum sodium concentration in both sexes was also reported by Flear and Singh, which is similar to our observation.

**Serum Sodium Level and Complications**

Hyponatraemia is defined as serum sodium level <136 mmol/L. In acute myocardial infarction, nonosmotic release of vasopressin may occur due to the acute development of left ventricular dysfunction; in response to pain, nausea and major stress, the most common mechanisms of hyponatraemia in adults; or in response to the administration of analgesics and diuretics. In this setting, vasopressin level increases concomitantly with the activation of other neurohormones such as renin and norepinephrine. Moreover, the renal effect of vasopressin is enhanced in heart failure as the vasopressin-regulated water in the collecting duct is up regulated. Mean serum sodium level at baseline in our study of males (n=70) was 137.88 ± 2.73 and of females (n=30) was 138.81 ± 2.59. The mean serum sodium level in cardiac failure of males (n=15) was 135.50 ± 4.48 and of females (n=10) 137.67 ± 2.66 at baselines. While mean serum sodium level in cardiogenic shock of males (n=10) was 134.56 ± 2.67 and of females (n=5) 135.57 ± 3.16 at baselines. Mean serum sodium level in study by Esha Mati et al (n=50) was 135 ± 5.17, while in Vinod Walli et al (n=36), it was 129.47 ± 4.87. There is not much significant differences observed in serum chloride levels in various groups. Serum potassium concentration was decreased significantly in patients of acute myocardial infarction with arrhythmias in our study. In present study, the mean of baseline of K was 3.77 ± 0.55 in males and 3.85 ± 4.18 in females. The mean of K in males at baseline was 3.79 ± 0.97 with arrhythmia and without arrhythmia K 3.94 ± 0.37. The mean of K in female at baseline was with arrhythmia 3.79 ± 0.79 and without arrhythmia was 3.85 ± 0.31. Salamon et al and Huliting J et al also observed hypokalaemia in patients of acute myocardial infarction in their studies. In present study, ventricular premature beats were present more commonly in hypokalaemic group similar to T Dyckner et al and Erik J observed higher incidence of ventricular premature beats in hypokalaemic group as compared to normokalaemic and hyperkalaemic group. Highest incidence of ventricular tachycardia was observed in hypokalaemic group by Dyckner and Erik J and Salomon et al. In our study, also ventricular tachycardia was present in significant number of patients with hypokalaemia. In present study, ventricular fibrillation was present only in hypokalaemic group. Similarly, Friedensohn A, Duke M and others observed higher incidence of ventricular fibrillation in hypokalaemic group. Serum magnesium concentration was decreased significantly in patients of acute myocardial infarction with arrhythmias in our study.

The possible mechanism for the low concentrations of sodium and potassium in AMI is the impairment of the Na/K pump and the Na/Ca exchanger. The active transport of these ions across cell membrane involves ATPase, which is in turn dependent on Mg for its activity. The theory of Seelig and Heggtveit states that the magnesium deficient status in turn dependent on Mg for its activity. The theory of Seelig and Heggtveit states that the magnesium deficient status and the magnesium deficiency status and an altered electrolyte concentration are caused by the reduction of the Na/K ATPase activity leading to sodium accumulation in the cardiac myocytes. Elevated myocardial sodium levels would result in the reversal of the Na+/K+ exchange and a rise in the intracellular calcium levels. This intracellular shift in calcium decreases the ionised calcium levels in the serum. This explains the electrical instability in the hearts of magnesium deficient individuals. Dimtruk in his series of 67 patients of ischaemic heart disease showed a distinct reduction of plasma magnesium during the first 3 days following onset of disease, the level normalised by 15-25 days from onset of the disease.

<table>
<thead>
<tr>
<th></th>
<th>Pre (Day 1)</th>
<th>Post (Day 5)</th>
<th>Mean Change</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male (without cardiogenic shock)</td>
<td>139.9 ± 2.48</td>
<td>140.7 ± 0.96</td>
<td>-0.8 ± 2.82</td>
<td>0.027</td>
</tr>
<tr>
<td>Female (with cardiogenic shock)</td>
<td>135.57 ± 3.16</td>
<td>139.09 ± 0.40</td>
<td>-0.48 ± 3.57</td>
<td>0.840</td>
</tr>
<tr>
<td>Female (without cardiogenic shock)</td>
<td>140.5 ± 0.28</td>
<td>141.45 ± 0.80</td>
<td>-0.85 ± 3.85</td>
<td>0.000</td>
</tr>
</tbody>
</table>

**Table 4. Intra Comparison of Day 1 and Day 5 of Na, K, Cl and Mg in Cardiogenic Shock in Both Genders**

CONCLUSION
Serum magnesium, sodium and potassium levels were significantly lower at baseline and gradually becomes near normal on 4th day in the AMI patients, so estimation of these can help to assess prognosis. Hypokalaemia and hypomagnesaemia are an important predictor of life-threatening arrhythmias occurring in acute myocardial infarction and should be treated.

REFERENCES