

## ORIGINAL ARTICLE

# STUDY OF SERUM SODIUM AND POTASSIUM DISTURBANCES IN PATIENTS OF ACUTE MYOCARDIAL INFARCTION

Amita A Gandhi<sup>1</sup>, Pankaj J Akholkar<sup>2</sup>, Vahid S Bharmal<sup>3</sup>

**Author's Affiliations:** <sup>1</sup>Assistant Professor, <sup>2</sup>Associate Professor, Medicine, GMERS Medical College, Sola, Ahmedabad; <sup>3</sup>Associate Professor, Medicine, M P Shah medical college, Jamnagar, Gujarat

**Correspondence:** Dr. Amita A. Gandhi, Email: amita67@gmail.com

## ABSTRACT

**Background:** The incidence of myocardial infarction is increasing specially in developing countries. Different electrolytes such as potassium and sodium play an important role in the cell metabolism, electrical conduction and membrane excitability. Abnormalities of these electrolytes due to different causes can lead to a significant cardiac life threatening events.

**Objective :** To ascertain the electrolytes status in patients of acute ST elevation myocardial infarction (STEMI) at the time of hospitalization and to study the relationship between hyponatremia and hypokalemia with ventricular arrhythmia and survival status of the patient on day seven of the admission.

**Methods:** Fifty patients of acute myocardial infarction were interrogated for history of presenting illness, examined for vitals, systemic examination and investigated by collection of blood sample and ECG.

**Conclusion:** There was increased mortality in patients with hyponatremia, but p value was not statistically significant (p value 0.15). There was definite correlation between hypokalemia and ventricular arrhythmias and there was increased tendency towards developing ventricular arrhythmias in patients with hypokalemia. (p value 0.009).

**Key words:** Acute myocardial infarction, Electrolytes, Serum Sodium, Serum potassium

## INTRODUCTION

Cardiovascular disease is one of the leading causes of morbidity and mortality across the world. World Health Organization (WHO) has declared cardiovascular disease as a modern epidemic.<sup>1</sup> Acute Myocardial Infarction is one of the manifestations of coronary heart disease leading to morbidity and mortality. Arrhythmias and hemodynamic abnormalities in left ventricular dysfunction are the major causes of mortality along with acute myocardial infarction. The arrhythmias predisposing factors are: autonomic nervous system dysfunction, electrolyte disorders, left ventricular dysfunction, myocardial ischemia and medications.<sup>2</sup> Different electrolytes such as potassium and sodium play an important role in the cell metabolism, electrical conduction and membrane excitability. Abnormalities of these electrolytes due to different causes can lead to a significant cardiac life threatening events.<sup>3</sup>

## METHODOLOGY

This was an observational prospective study. Informed written consent was taken from all subjects. After obtaining detailed medical history and clinical examination of the patient blood sample were taken for laboratory investigations including Serum Electrolytes, Blood Sugar, Renal function test, Liver Function Test, and treatment of the patient was initiated with standard anti ischemic therapy and serial ECG of the patient were done. Serum electrolytes done were serum sodium, serum potassium.

At the beginning of admission and in a resting position, the blood sample was taken from patients' left cubital vein. The sucking pressure in venipuncture was tried to be very slow. The blood samples were slowly transferred to the test tube and then to the laboratory without any shake.

**Inclusion Criteria**

Patients of STEMI (ST elevation myocardial infarction) as diagnosed clinically, by ECG and by biomarkers. All patients of AMI (acute myocardial infarction) were medicated with angiotensin converting enzyme inhibitors or angiotensin receptor blockers over and above the standard thrombolytic, anti ischemic and anti platelet therapy.

**Exclusion Criteria:** Anaemia, significant hepatic, renal and pulmonary disease, diabetes mellitus, infection, hypo and hyperthyroidism.

All the patients of the STEMI were grouped according to

Serum sodium (mmol/l) <136mmol/l, 136-145mmol/l, >145mmol/l

Serum potassium (mmol/l) <3.5mmol/l, 3.5-4.5mmol/l, >4.5mmol/l

Observed outcome was survival status on the seventh day of admission and development of ventricular Arrhythmias. The observed clinical outcome was analysed by Chi square test .P value of less than 0.05 was taken as statistically significant.

**RESULTS**

In our study population, 64 % (n=32) were male and 36% (n=18) were female. Out of these 22% (n=11) patients died during first seven days. Amongst the patients died 14% (n=7) were male while 8% (n=4) were female.

Majority of study subjects (76%) had normal serum sodium of 136-145 mmol/l. Study subjects with serum sodium level < 136 mmol/L were (14 %) & serum sodium level >145 were 10 % (n=5).

Table 2 shows that mortality in patients with serum sodium <136 mmol/l was 42.8 % (3 out of 7), while in patients with normal serum sodium level was 18 % (7 out of 38) and in patients with serum sodium level > 145 mmol/l was 20 % (1 out of 5). Thus patients with hyponatremia had higher rate of mortality, but p value is not significant (p value 0.15).

Table 3 shows majority of patients 80 % (n=40) had normal serum potassium level, while 12 % (n=6) had serum potassium level less than 3.5 mmol/l and 8 % (n=4) had more than 5.5 mmol/l.

**Table 1- Distribution of study subjects according to serum sodium level.**

Serum Sodium Level (mmol/L)	Total Patient (%) (n=50)	Male (%) (n=32)	Female (%) (n=18)
< 136	7(14)	5(15.6)	2(11.1)
136-145	38(76)	25(78.1)	13(72.2)
> 145	5(10)	2(6.3)	3(16.7)

**Table 2 -Occurrence of Mortality as per serum sodium level**

Serum Sodium Level (mmol/L)	Total Patients (%) (n=50)	Mortality (%) (n=11)	Male (%) (n=7)	Female (%) (n=4)
< 136	7(14%)	3(27.3%)	2(28.6%)	1(25%)
136-145	38(76%)	7(63.6%)	5(71.4%)	2(50%)
> 145	5(10%)	1(9.1%)	0(0%)	1(25%)

**Table 3- Distribution of Study subjects according to serum potassium level**

Serum Potassium Level (mmol/L)	Total Patients (%) (n=50)	Male (%) (n=32)	Female (%) (n=18)
< 3.5	6(12)	4(12.5)	2(11.1)
3.5-5.5	40(80)	25(78.1)	15(83.3)
> 5.5	4(8)	3(9.4)	1(5.6)

**Table 4-Ventricular Arrhythmias in study subjects according to serum potassium level**

Serum Potassium level(mmol/L)	Total Patients (%) (n=50)	Ventricular arrhythmias (%) (n=12)	Male (%) (n=8)	Female (%) (n=4)
< 3.5	6(12)	4(33.3)	3(37.5)	1(25)
3.5-5.5	40(80)	7(58.3)	4(50)	3(75)
> 5.5	4(8)	1(8.3)	1(12.5)	0(0)

Table 4 shows that 66.77 % ( 4 out of 6 ) patients with serum potassium level less than 3.5mmol/L had developed ventricular arrhythmias and these four patients had serum potassium level  $\leq 3$ mmol/L. 25% (1 out of 4 ) patients with serum potassium  $> 5.5$ mmol/L had developed ventricular arrhythmias. 17.5% (7 out of 40) patients with serum potassium level 3.5-5.5mmol/L had developed ventricular arrhythmias. Thus patients with hypokalemia are found to be associated with increased incidence of ventricular Arrhythmias and p value is significant (p 0.009).

Out of 31 subjects with serum potassium level  $\leq 4.5$ mmol/L, nine subjects had developed ventricular arrhythmias and four subjects with serum potassium level  $\leq 3$ mmol/L had developed ventricular arrhythmias. Thus there was increased trend towards developing ventricular arrhythmias as serum potassium level decreases from 4.5mmol/L and once it decreases  $\leq 3$ mmol/L incidence of arrhythmias reached 100%.

## DISCUSSION

Patients of STEMI were examined within 24 hrs to collect information about their electrolytes as early as possible after the episode so that mortality can be viewed in light of prevailing electrolyte status.

**Serum Sodium Level, Survival Status & Complications:** Hyponatremia 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 hyponatremia in adults; or in response to the administration of analgesics and diuretics.<sup>4,5</sup> In this setting, vasopressin level increases concomitantly with the activation of other neurohormones such as renin and nor epinephrine.<sup>6</sup> Moreover, the renal effect of vasopressin is enhanced in heart failure, as the vasopressin-regulated water in the collecting duct is up regulated.<sup>7</sup>

Mean serum sodium level in study by Esha mati<sup>8</sup> et al (n=50) was  $135 \pm 5.17$  while in Vinod<sup>9</sup> wali et al (n=36) it was  $129.47 \pm 4.87$ . Mean serum sodium level in our study was  $137.64 \pm 9.08$ .

Hyponatremia was found in 14% (n=7) patients in our study, while in study by Alexander Goldberg<sup>10</sup> 12.5% (n=131), and in Singla et al<sup>11</sup> 23.1% (n=341) had hyponatremia.

Out of 7(14%) patients who had hyponatremia 4 (8%) patients developed congestive heart failure during hospital stay. Out of 38 patients with normal serum sodium level 5(10%) had developed congestive heart failure. So it is evident from our study that hyponatremia is associated Heart failure and mortality was also on higher side.

Mean sodium level of the patients survived was  $139.35 \pm 5.68$  and in patients who died was  $131.54 \pm 15.16$ . So, it was found that mortality was higher in patients with hyponatremia, though difference was not statistically significant perhaps due to small sample size.

**Serum Potassium Level, Survival Status & Complications:** Normal potassium level considered in our study was 3.5-5.5 mmol/L. Hypokalemia is defined as serum potassium level  $< 3.5$  mmol/L. In STEMI hypokalemia has been found to be associated with increased risk of ventricular tachycardia and ventricular fibrillation.<sup>12</sup> The physiological mechanisms that may account for the relationship of hypokalemia to cardiac arrhythmias are not fully explained; however, it has been suggested that low extracellular potassium could increase automaticity (increased slope of diastolic depolarization) resulting in a decreased conduction velocity.

In our study mean serum potassium level was  $4.214 \pm 0.79$ . In study by Esha mati et al<sup>8</sup> (n=50) mean serum potassium level was  $3.66 \pm 0.56$ . In study by Vinod Wali et al<sup>9</sup> (n=36) mean serum potassium level was  $4.18 \pm 0.63$ .

In our study in patients with myocardial infarction 12%(n=6) had hypokalemia. In study conducted by Taysir S Garadah,<sup>13</sup> 30%(n=84) out of 274 patients had hypokalemia. In study by Azin Alizadehasl<sup>14</sup> about 12.2% (n=61) of the overall 500 patients studied, had significant hypokalemia. In study by Richard J. Solomon<sup>12</sup>, 14%(n=21) of the overall 151 patients studied had hypokalemia.

**Incidence of ventricular arrhythmias:** Serum potassium  $< 3.5$  meql/dl, four (66.67%) patients had Ventricular Arrhythmias (p value 0.009) While it was observed in 4(10%) patients out of 40 normokalemic patients. Out of 40 patients with normal serum potassium level, mortality was found in nine (22.5%) patients. Amongst four patients who had hyperkalemia two patients developed ventricular tachycardia.

In a study by Jan Erik Nordrehaug<sup>15</sup>, MD, ventricular fibrillation occurred in 17.2% of 122 hypokalemic patients and in 7.5% of 952 normokalemic patients (p  $< 0.01$ ).

In study by Richard J. Solomon<sup>12</sup>, 24.5% (n=37) patients experienced either ventricular tachycardia or ventricular fibrillation. 67% of patients with a serum potassium of less than 3.1 mmol/L had these serious ventricular arrhythmias compared to 40% of patients with a serum potassium between 3.1 and 3.5 mmol/L and 20 % of normokalemic patients

In study by Azin Alizadehasl<sup>14</sup>, Ventricular Tachycardia (VT) was found in 29.1% of patients with hypokalemia as compared to 17.8% in normokalemic patients. Also, total mortality was more frequent in hypokalemic group than normokalemic group (20.6% versus 16.9%). Thus, hypokalemia was an important predictor of malignant arrhythmias, mortality and poor outcome in AMI patients.

#### Association of ventricular arrhythmia with serum potassium level

In our study mean potassium level of patients without ventricular Arrhythmias was  $4.34 \pm 0.65$  and of patients with ventricular Arrhythmias was  $3.8 \pm 1.08$ . Statically significant association was found between hypokalemia and ventricular arrhythmias as p value was 0.009.

In one study conducted by Taysir s Garadah<sup>13</sup> the incidence of hypokalemia was significantly higher in patients with arrhythmias compared to those without arrhythmias. Out of 130 patients with arrhythmias 61 (46.97%) patients had hypokalemia compared to 23 (15%) out of 144 patients without arrhythmias

In all these study, study design was large in comparison to our study & single variable was studied in them so better correlation ( $P < 0.001$ ) was noted between hypokalemia and Arrhythmias in patients with acute myocardial infarction.

#### CONCLUSION

From this study we conclude that there was increased mortality in patients with hyponatremia, but p value was non significant ( $p = 0.15$ ). There was definite correlation between hypokalemia and ventricular arrhythmias and increased tendency towards developing ventricular arrhythmias in patients with hypokalemia. ( $p < 0.01$ ). As the serum potassium was decreasing from 4.5mmol/L, chance of developing ventricular arrhythmias was increasing and once the value of potassium declined to  $\leq 3$ mmol/l, there was 100% chance of developing ventricular arrhythmias. There was no

correlation found between hypernatremia and mortality. There was no correlation found between increased potassium level and ventricular arrhythmias in our study.

#### REFERENCES

1. K Park. Park's Textbook of Preventive and Social Medicine, 22nd ed. Jabalpur: Bhanot Publishers; 2013. p 338.
2. Vera Z, Janzen D, Desai J. Acute hypokalemia and inducibility of ventricular tachyarrhythmia in a nonischemic canine model. *Chest*. 1991;100(5):1414-20.
3. Tada Y, Nakamura T, Funayama H, Sugawara Y, Ako J, Ishikawa S, Momomura S. Early development of hyponatremia implicates short and long term outcomes in ST elevation acute myocardial infarction. *Circ J* 2011;75:1927-1933
4. Adrogue HJ, Madias NE. Hyponatremia. *N Engl J Med* 2000; 342:1581-1589.
5. Rowe JW, Shelton RL, Helderman JH. Influence of the emetic reflex on vasopressin release in man. *Kidney Int*. 1979; 16:729-735
6. McAlpine HM, Morton JJ, Leckie B, Rumley A, Gillen G, Dargie HJ. Neuroendocrine activation after acute myocardial infarction, *Br Heart J* 1988;60:117-124
7. Kumar S, Berl T. Sodium. *Lancet* 1998; 352:220-228
8. Esha Mati, Krisnamurthy N, Ashakiran S, Sumathi M E, Prasad R: Dyselectrolytemia in Acute Myocardial Infarction-A Retrospective study. *J clin Biomed Sci* 2012;2(4):167-174
9. Wali V, Singi Y: Study of Serum Sodium and Potassium in Acute Myocardial Infarction. *Journal of clinical and diagnostic research*. 2014 Nov Vol 8(11):7-9
10. Goldberg A, Hammerman H, Petcherski S, Nassar M, Zdoroviyak A, Yalonetsky S, Kapeliovich M, Agmon Y et al: Prognostic importance of hyponatremia in acute ST-elevation myocardial infarction. *Arch Intern Med*. 2006;166(7):781-786. doi:10.1001/archinte.166.7.78115
11. Singla I, Zahid M, Good CB, Macioce A, Sonel AF Effect of hyponatremia ( $< 135$  mEq/L) on outcome in patients with non-ST-elevation acute coronary syndrome. *Am J Cardiology* 2007 Aug;100(3):406-8
12. Solomon RJ, Cole AG. Importance of Potassium in Patients with Acute Myocardial Infarction. *Acta Med Scand Suppl* 1981; 647: 87-93.
13. Taysir S Garadah, Salah Kassab, Jamal Golbahar, Association of Hypokalemia with Severe Arrhythmias in Patients with Acute Myocardial Infarction *British Heart Journal* 1983 50: 525-9. *Bahrain Medical Bulletin*, Vol. 33, No. 1, March 2010
14. Azin Alizadehasl, Farnaz Sepasi, Rasoul Azarfarin and Shamsi Ghaffari Hypokalemia, Arrhythmias and Early Outcomes in Acute Myocardial Infarction *Research Journal of Biological Sciences* :2008:Vol 3:Issue9:1130-1132
15. Jan Erik Nordrehaug Malignant arrhythmia in relation to serum potassium in acute myocardial infarction *The American journal of cardiology* August 30, 1985 Volume 56, Issue 6, Pages D20-D23