A Study of Electrolyte Imbalance in Acute Myocardial Infarction Patients at A Tertiary Care Hospital in Western Maharashtra

Shilpa Patil[1], Saurabh Gandhi[2], Piyush Prajapati[2], Shivraj Afzalpurkar[2], Omkar Patil[2], Mohit Khatri[3]

ABSTRACT

Introduction: Worldwide Acute myocardial infarction is associated with high morbidity and mortality in hospitalised patients. Serum sodium potassium and calcium are considered to be major electrolytes associated with electrophysiological properties of myocardial membrane. Serum electrolyte imbalances after an episode of acute myocardial infarction are common. These electrolytes play an important role in altering the prognosis of such myocardial infarction patients. Study aimed to compare the prevalence and pattern of dyselectrolytemia in both cases and controls.To determine the relation between in hospital mortality and electrolyte changes in cases and controls.

Material and methods: This was a prospective case control study. Cases Included 100 clinically diagnosed and angiographically proven cases of AMI. Controls are patients who admitted to general wards with minor illness.

Result: 27% of all MI patients found to be hyponatraemic. Around 24% found to be hypokalaemic and 49% of cases found to be hypocalcaemic.

Conclusion: Electrolyte imbalance maximally seen in Calcium. Hypocalcaemia was present in around half of total cases. Hypokalaemia was evident in a large number of patient in the early phase of AMI. Hyponatremia was a fairly common finding among acute MI patients.

Keywords Dyselectrolytemia, Myocardial infarction, Hyponatraemia, Hypokalaemia, Hypocalcaemia.

INTRODUCTION

Worldwide Acute myocardial infarction is associated with high morbidity and mortality in hospitalised patients. Worldwide there are 3 million sudden cardiac deaths per year due to acute myocardial infarction.[1] Serum sodium, potassium and calcium are considered to be major electrolytes associated with electrophysiological properties of myocardial membrane. The sarcolemma is impermeable to Na in the resting state. It has Na K ATPase pump that plays important role in establishing the resting potential. This pump exports Na from the cell out and imports K inside the cell against their concentration gradient. Thus intracellular K is relatively high and Na is low and extracellular Na is high and K is low. There are 4 phases of action potential dependant on sodium, potassium and calcium.

Serum electrolyte imbalances after an episode of acute myocardial infarction are common. But clinical importance of these imbalances in both STEMI and NSTEMI has not been fully understood. These electrolytes play an important role in altering the prognosis of such myocardial infarction patients.

Hyponatremia defined as serum sodium concentration <133 mEq/L. Hyponatremia is relatively common in patients with acute MI. Some studies have shown that hyponatremia is associated with poor outcomes in patients with STEMI and NSTEMI, and the risk of mortality increased with severity of hyponatremia.[2-4]

Hypokalaemia defined as serum potassium levels <3.5 mEq/L. The prevalence and role of hypokalaemia in myocardial infarction has been under investigation for a long time.[5,6] Hypokalaemia in patients with AMI is thought to predict increased in-hospital morbidity particularly arrhythmias and mortality.[7] Several studies have shown association between hypokalaemia with increased occurrence of cardiac arrhythmias in AMI patients.[8] Hypokalaemia was even found to be associated with larger infarcts.[7] Recent guidelines suggest that K levels should be monitored and routinely replaced in patient with heart failure and MI, even if on admission K appears normal.[9] Hyperkalaemia also associated with increased mortality and should be avoided. The total body potassium levels in body is 3500 mmol, out of which 98 % is intracellular. Its main regulation is by the renal excretion and shift between the intracellular and extracellular compartments.[10] Mainly the sodium potassium ATPase pump is responsible for preserving the intracellular potassium. Aldosterone and vasopressin stimulate the potassium secretion by up-regulating the luminal Na K ATPase pump and opening the luminal Na and K channels.[10] The sudden cardiac death after MI (death within 1 hour) is mainly due to alteration in the environment at the level of myocytes and purkinje fibres that are mainly regulated by electrolyte imbalances and autonomic nervous system activity.[11]

Since calcium is involved in the regulation of a variety of events leading to Coronary artery disease such as coronary spasm, thrombosis formation and disruption of atherosclerotic plaque. During MI, calcium handling between sarcoplasmic reticulum and myofilaments is disrupted and calcium is diverted to the mitochondria causing oedema. Defective calcium handling causes reversible as well as irreversible myocardial injury. Under normal conditions the sarcoplasmic calcium concentration is low with gradient between intra and extracellular compartment. During an action potential, voltage gated Na+ channels are activated and inward Na+ current induces a rapid depolarization of sarcolemma that opens L type Ca++ channels. The Ca++ influx triggers opening of Ryanodine (RYR) receptor leading to release of calcium into the cytosol initiating contraction. Cytosolic calcium accumulation plays a major role in initiation of programmed cell death.[12]

[1]Assistant Professor, Department of Medicine, KIMS, [2]Resident, 3rd year, [3]Resident, 1st year, Department of Medicine, Krishna hospital and medical research centre, Karad, Maharashtra, India

Corresponding author: Dr. Saurabh Gandhi, Room No 203, IHR, Krishna hospital campus, Karad, Maharashtra, India

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Hypophosphatemia in AMI is associated with LV dysfunction which results in increase in 30 day mortality. It is emphasized that decrease in cytosolic magnesium contribute to local release of myocardial catecholamines and generation of oxygen derived free radicals leading to sarcoplasmic reticulum dysfunction and loss of selective permeability of the sarcolemmal membrane.

The pattern and prevalence of these electrolyte imbalances in the era of primary intervention is yet to be fully described.[2] So we conducted this study to determine the prevalence and pattern of dyselectrolytemia with MI and to determine in-hospital mortality associated with dyselectrolytemia in early phase of MI. Furthermore we studied all electrolytes i.e. sodium, potassium, calcium, phosphorus and magnessium in both cases and control in this study.

Aims and Objectives of the study were to study the Prevalence of dyselectrolytemia in both cases and controls, the pattern of dyselectrolytemia in both cases and controls, compare the electrolyte imbalance among cases and controls, determine the relation between in hospital mortality and electrolyte changes in cases with acute MI and controls without MI and compare the electrolyte imbalance in STEMI and NSTEMI.

MATERIAL AND METHODS

This study was a Prospective case control study. Data was collected from the hospital records of Cardiology unit in Krishna Hospital, Karad. The study was approved by the Institutional Ethical Committee of Krishna institute of Medical Sciences Karad.

Inclusion criteria

Cases: Included 100 clinically diagnosed and angiographically proven cases of AMI. All patients were registered and admitted due to chest pain in Krishna hospital karad during 18 months (March 2015-August 2016). MI was diagnosed if a patient presented with chest pain > 20 min, had ST segment changes in ECG and serum CPK MB and Troponin I elevation. The cases included 31 female patients and 69 male patients with mean age of 62.84 years.

Controls: Data was obtained from patients who were admitted to general wards of Krishna hospital with minor illness and without myocardial disease or without systemic illness that affect electrolytes. Data of 100 age and sex matched non-infarct individuals with mean age of 58.17 years was taken during the same 18 months period. Patients were all registered and admitted to the Krishna Hospital.

Exclusion criteria

1. Patients with renal failure (both ARF and CRF).
2. Diabetic patients.
3. Acute and chronic Liver failure.
4. Acute gastroenteritis.
5. Malignancy.
6. Adrenal insufficiency
7. Hypertensive patients on potassium sparing diuretics.
8. COPD patients on B-agonists.

Study material

Data obtained for the study included:

- Clinical history, physical examination and drug history.
- Electrolyte levels (Sodium, Potassium, Calcium, Phosphorus, and Magnesium) measured within 48 hours of admission.
- RBS, blood urea, serum creatinine.
- All the biochemical analysis was done in the Krishna Hospital central lab using Ion selective electrodes (ISE) standard methods. Sodium levels of 133-146 mEq/L was considered as normal. The normal range for potassium was taken as 3.5-5.4 mEq/L, Calcium 8.5-10.5mg/dl, Phosphorus 2.5-4.5 mg/dl and magnesium 1.6-2.6mg/dl.

STATISTICAL ANALYSIS

In order to apply independent t-test to a study data the minimum sample size required is 30 cases and 30 controls. We have taken total 200 sample size (100 cases and 100 controls) in this study. For the statistical analysis SPSS package was used in the study. All values were expressed as mean ± SD. Continuous variables were compared using student 't' test. For all analysis 'p' value < 0.05 was defined as significant. Non parametric test -Fischer Exact test was applied. Shapiro Wilks test was applied to test the normality of distribution of data.

RESULTS

In our study the mean age of the cases was 62.89±11.85 years (Figure-1). Male prepoderence was seen in both cases and controls. In cases 69% patients were males whereas in controls 73% patients were males (Figure-2,3). In cases the average sodium levels was 135.93±3.96 mEq/L. 27% of all MI patients were found to be hyponatraemic with lowest value being 125 mEq/L. When compared to the control group the hyponatremia was significant with p value 0.0001 for hyponatremia (Table-1). Average value of potassium was 3.81±0.48 mEq/L. Around 24% were found to be hypokalaemic with a p value of 0.0402 for hypokalaemia. The lowest value was 2.6 mEq/L (Table-2). The average value of Calcium was 8.51±0.66 mg/dl and 49% of cases were found to be hypocalcaemic with lowest value being 7.2mg/dl. P value for hypocalcaemia was 0.0206 (Table-3). The average value of PO4 in cases was 3.25±0.6 mg/dl and Mg was 2.03±0.32 mg/dl. There was no statistically significant imbalance of other electrolytes like PO_4 and Mg.

Of the 100 cases 12 patients expired showing a mortality rate of 12%. Among these mortalities 7 patients were having hyponatraemia while 5 patients were having hypokalaemia and 8 were having hypocalcaemia. The control group had no reported mortality. In cases with no electrolyte abnormality had also no reported mortality.

DISCUSSION

Electrolyte imbalances are fairly common in the acute MI

Types of MI	Normal Sr. Na (%)	Hyponatremia (%)	Hypernatremia (%)	Total (%)
STEMI	64 (64%)	20 (20%)	0 (0%)	84 (84%)
NSTEMI	9 (9%)	7 (7%)	0 (0%)	16 (16%)
Total	73 (73%)	27 (27%)	0 (0%)	100 (100%)

Table-1: Frequency of Sr. sodium imbalance in Acute MI patients

patients. When measured on admission, the sodium potassium and calcium levels were found to be significantly reduced in our study as compared to non-infarct controls. Hypocalcaemia was evident in around half of patients with acute MI.

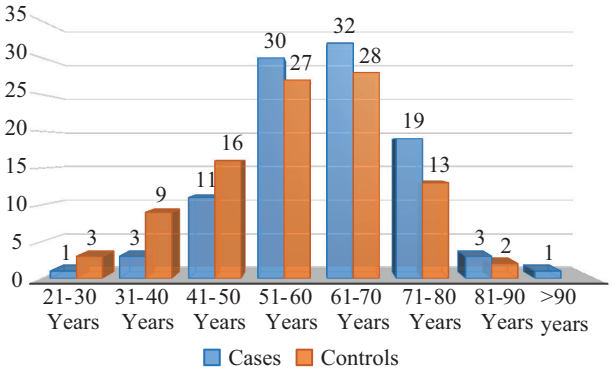

Figure-1: Age Distribution of cases and controls

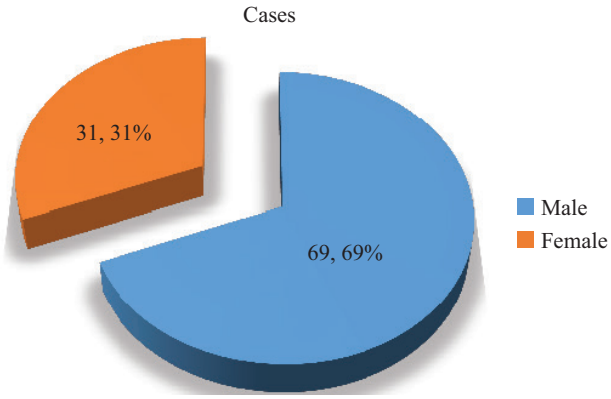

Figure-2: Gender Distribution of Cases

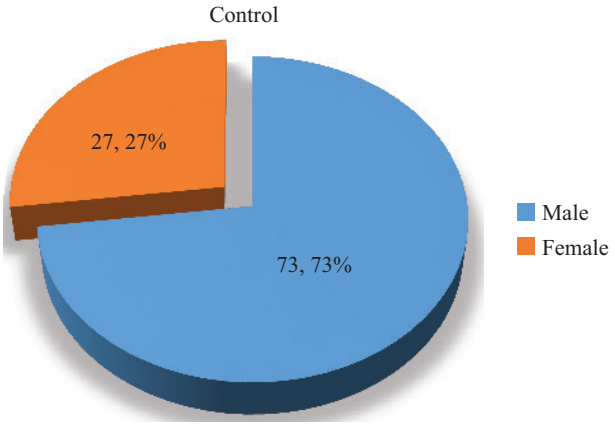

Figure-3: Gender distribution of controls

Hyponatremia often associated with increased morbidity and mortality in MI patients. MI patients with hyponatremia also found to have lower ejection fraction. A study conducted by Flear et al showed 45% of infarcted patients had hyponatremia and were associated with increased mortality.[13] In this study 27% of MI patients were found to have hyponatremia which was significantly higher than controls (9%). Hyponatremia could probably be due to non-osmotic secretion of vasopressin impairing the water excretion causing dilutional hyponatraemia. Sodium is freely filtered by the glomerulus, 70-80% gets reabsorbed in the proximal tubule, 20-25 % in the loop of Henle and remaining 5-10% in distal tubules.[16] Flear et al had hypothesized that the hypoxia and cardiac ischemia increased the cell membrane permeability to sodium ions, activation of sympathetic nervous system and rennin-angiotensin system.[13] None of the patients were found to be hypernatraemic in our study.

Hypokalaemia is associated with an increased risk of ventricular tachycardia and ventricular fibrillation.[14] Skeletal muscle acts as a reservoir pool for potassium maintaining potassium in vital organs such as the heart and brain. Hypokalaemia is mostly due to the stress induced catecholamine response (predominantly

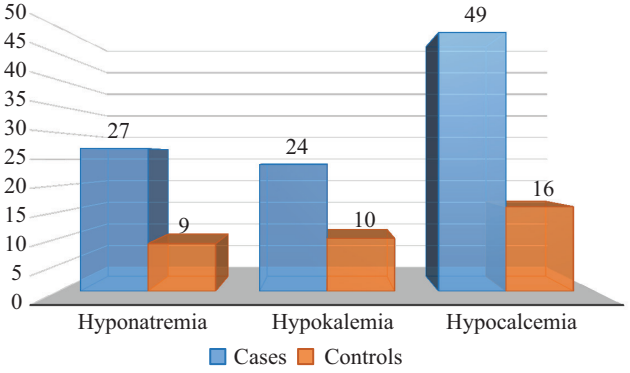

Figure-4: Pattern of dyselectrolytemia in cases and controls

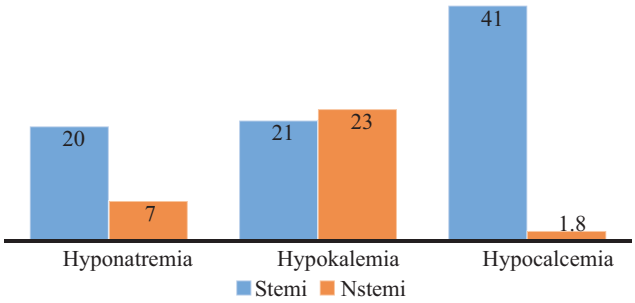

Figure-5: Comparison of dyselectrolytemia in STEMI and NSTEMI

Types of MI	Normal Sr. K (%)	Hypokalaemia (%)	Hyperkalaemia (%)	Total (%)
STEMI	63 (63%)	21 (21%)	0 (0%)	84 (84%)
NSTEMI	13 (13%)	3 (3%)	0 (0%)	16 (16%)
Total	76 (76%)	24 (24%)	0 (0%)	100 (100%)

Table 2: Frequency of Sr. potassium imbalance in Acute MI patients

Types of MI	Normal Sr. Ca (%)	Hypocalcaemia (%)	Hypercalcaemia (%)	Total (%)
STEMI	43 (43%)	41 (41%)	0 (0%)	84 (84%)
NSTEMI	8 (8%)	8 (8%)	0 (0%)	16 (16%)
Total	51 (51%)	49 (49%)	0 (0%)	100 (100%)

Table-3: Frequency of Sr. Calcium imbalance in Acute MI patients

epinephrine from the adrenal medulla) that function as hormones in such patients causing increased K uptake into cells.[7,15] In a study done by Goyal et al, it was found the hospital mortality in MI patients to be the least in patients with normal potassium levels (3.5-4.5mmol/l).[6] In our study 24% of cases were hypokalaemic which was significantly higher than controls (10%).

Calcium ions play a vital role in excitation-contraction of the cardiac muscle fibres and they are essential in both the cardiac and systemic vasculature.[17] Furthermore hypocalcaemia impairs myocardial contractility and there are several reports of congestive heart failure caused by severe hypocalcaemia[19] and cardiomyopathy in long-standing hypocalcaemia.[20] Moreover coronary spasm due to hypocalcaemia has been reported as the most likely mechanism of chest pain in young patients mimicking acute myocardial infarction.[18,21] In previous studies it was concluded that hypocalcaemic patients were older, had more cardiovascular risk factors, lower rate of emergency revascularization and higher in-hospital mortality as compared with hypercalcaemic patients. According to other study done by Dr.P.Kusumakumari high serum calcium was found to have correlation with myocardial infarction and serum calcium was found to be an independent prospective risk factor for MI suggesting that extracellular calcium plays a role in the atherosclerotic process.[22] In our study 49% of cases were hypocalcaemic and no patient was hypercalcaemic suggesting strong correlation between hypocalcaemia and cardiovascular risk. Further studies are required to determine the actual mechanism and whether patients with hypocalcaemia could benefit from calcium supplement.

There was no statistically significant imbalance of other electrolytes like PO_4 and Mg. The association of hyponatremia hypokalaemia or hypocalcaemia with early presentation in acute MI may alert the clinician about the acuteness and severity of patient's illness.

CONCLUSION

Electrolyte imbalance was maximally seen in Calcium. Hypocalcaemia was present in around half of total cases. Hypokalaemia was evident in a large number of patient in the early phase of AMI, mostly due to the catecholamine response in such patients. It has been associated with ventricular arrhythmias and increased mortality in post MI patients. Hyponatremia was a fairly common finding among acute MI patients, probably attributed to the non-osmotic secretion of vasopressin. There was increased mortality in all 3 electrolyte imbalances. The clinicians are advised to closely monitor these electrolyte changes and correct them as they seem to have adverse effects on the disease outcome and prognosis.

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