Original Article

Dyselectrolytemia in Acute Myocardial Infarction - A Retrospective Study

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ABSTRACT

Background: *Myocardial infarction(MI) is a dreadful complication of cardiovascular disease causing increasing mortality worldwide. Sudden cardiac deaths occur worldwide at rates of around 3 million per year. Electrolyte imbalances after an episode of acute MI are common. Clinical importance of these imbalances in ST elevation Myocardial Infarction (STEMI) in the era of primary intervention has not been fully understood.*

Objectives: *To determine the pattern of dyselectrolytemia in post MI patients and to compare the electrolyte levels among MI and non-MIpatients.*

Materials & Methods: *This was a retrospective case control study including 50 consecutive acute MI patients admitted to the coronary care unit and 50 controls. Plasma sodium, potassium and chloride levels were obtained in all MI patients within 48 hours of admission. Patient history, physical examination, drug history and laboratory results were recorded by chart abstraction.*

Conclusion: *Hypokalemia and hyponatremia were fairly common findings among acute MI patients. Serum chloride levels showed no particular pattern or significant difference among the two groups.*

Key Words: *Myocardial infarction, Serum chloride, Serum creatinine, Serum potassium, Serum sodium.*

INTRODUCTION

Acute myocardial infarction (AMI) is one of the most common diagnosis made in hospitalized patients in industrialized countries. It is a growing cause of death worldwide. Sudden cardiac deaths occur worldwide at rates of around 3 million per year.[1] Serum Sodium, potassium and chloride are considered to be major determinants of electrophysiological properties of myocardial membrane. Electrolyte imbalances after an episode of AMI are common. Clinical importance of these imbalances in ST elevation myocardial infarction (STEMI) in the era of primary intervention has not been fully understood.[2]

The sarcolemma, which in the resting state is largely impermeable to Na^+, has a Na^+K^+ATPase pump that extrudes Na^+ from the cell; this pump plays a critical role in establishing the resting potential. Thus, intracellular [K^+] is relatively high and [Na^+] is far lower, while, conversely, extracellular [Na^+] is high and [K^+] is low. There are four phases of

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the action potential dependent on Na^+, K^+ and Ca^{++}. [3]

Several electrolyte changes have been reported to follow AMI. These electrolyte levels, being modifiable hold an important role in altering the prognosis of such myocardial infarction (MI) patients.

The T wave changes commonly found in the ECG of MI patients are attributed to changes in potassium levels. Potassium homeostasis is critical to prevent the adverse events in patients with cardiovascular disease.[4]

The prevalence and role of hypokalemia, in myocardial ischemia and MI in particular, has been under investigation for a long time.[5] Although controversial, hypokalemia in patients with AMI is thought to predict increased in-hospital morbidity, particularly arrhythmias and mortality.[6] Several studies have shown association between hypokalemia, even mild hypokalemia with increased occurrence of cardiac arrhythmias in AMI patients.[7] Hypokalemia defined as serum potassium levels <3.5 mmol/l is portrayed as a determinant of excessive morbidity in such patients, particularly malignant ventricular arrhythmia.[5] Hypokalemia was even found to be associated with larger infarcts.[6] It is important to find the relation between mean post admission potassium levels and the clinical outcomes, as these potassium levels are potentially modifiable during hospitalization. Recent guidelines suggest that K^+ levels should be monitored and routinely replaced in patient with heart failure and MI, even if initial K^+ appears normal.[8] Potassium levels of > 4.5 mmol/l are also associated with increased mortality and are to be avoided.[4]

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 .[9] 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.[9]

The sudden cardiac death after MI (death within 1 hour) is due to the interplay of perturbations of the environment at the level of myocytes and purkinje fibres, that are mainly regulated by electrolyte imbalances and autonomic nervous system activity.[1] The therapeutic doses of beta 2 adrenergic agonist in inhalation or injectable form, and also insulin are known to reduce the serum potassium levels.[1]

Serum sodium imbalance has been also recorded in early phase of MI in some studies.[10] Hyponatremia defined as serum sodium concentration <135 mmol/l is relatively common among patients hospitalized with acute MI.[2] Some studies have shown that hyponatremia is linked to poor outcomes in patients with STEMI and non ST elevation coronary syndromes, and the risk of mortality increased with severity of hyponatremia.[11]

A study done earlier by Flear et al found that decreased serum chloride levels to be a common finding among acute MI patients.[12] The pattern and prevalence of these electrolyte imbalances in the era of primary intervention is yet to be fully described, in rural areas. This

study was taken up to determine the prevalence and pattern of dyselectrolytemia, and in-hospital mortality in early phase of MI.

OBJECTIVES

1. Study the pattern dyselectrolytemia in both cases and controls.
2. Compare the electrolyte imbalance among cases and controls.
3. Detect the percent of electrolyte changes in cases with acute MI and controls without MI.

MATERIAL AND METHODS

This study was a Retrospective case control study. Cases: included 50 clinically diagnosed consecutive cases of AMI. The patients were all registered and admitted due to MI pain the Coronary care unit of R.L Jalappa Narayana Hrudayalaya hospital during 3 months (Feb - Apr 2010). MI was diagnosed if a patient presented with chest pain > 20 min, had ST segment changes in ECG and plasma CK MB elevation. The cases included 13 female patients, with an average age of 52 ± 12 yrs.

Controls: Data was obtained from subjects OPD cards and hospital registers who came for regular checkups, minor surgeries and without myocardial disease, or systemic ailments that affect electrolytes. Data of 50 age and sex matched non infarct individuals with an average age of 49 ± 15 yrs was taken during the same 3 month period. Subjects were all registered or admitted to the R.L Jalappa Hospital.

Exclusion criteria - Patients with renal insufficiency were excluded based on serum creatinine levels and hyperglycemic MI patients were excluded. Known cases of liver failure, chronic vomiting, diarrhea patients, malignancy, adrenal insufficiency and hypertensive patients on potassium sparing diuretics were excluded based on history.

Study material - Data was collected from the hospital records of Coronary Care Unit of Narayana Hrudayalaya unit in R.L Jalappa Hospital, Kolar. Data obtained for the study included: Electrolyte levels (Sodium, Potassium, Chloride) measured within 48 hours of admission, RBS, blood urea, serum creatinine, along with clinical history, physical examination history and drug history by chart abstraction. All the biochemical analysis was done in the RLJ Hospital central lab using Ion selective electrodes (ISE) standard methods.

Sodium levels < 135 mEq/L by 48 hrs after hospitalization was considered as hyponatremia. The upper limit being 145 mmol/l. The normal range for potassium was taken as 3.5-5.1 mmol/l and chloride levels of 98-107mmol/l. Routine quality checks were followed during analysis for quality assurance.

The study was approved by the Institutional Ethical Committee of Sri Devaraj Urs Medical College, Kolar. In order to apply independent t-test to a study data, the minimum sample size required is 30 cases and 30 controls. Hence total of 100 samples were taken 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 to

the data to assess the significance of difference.

Shapiro Wilks test was applied to test the normality of distribution of data.

RESULTS

In our study the average age of the cases were 58.75±12 years. Around 36% were found to be hypokalemic among the MI patients with a p value of <0.001 for hypokalemia. (Table 1) The lowest level being 2.6 mmol/l. (fig 1)

In cases the average sodium levels was 135± 5.17 mmol/l . 43% of all MI patients were found to be hyponatremic. (fig 2) When compared to the control group, the dyselectrolytemia was significant with p value <0.05 for hyponatremia. Chloride levels showed no significant difference among the two groups.

Of the 50 cases, 6 patients expired eventually while hospitalized showing a mortality rate of 3%. Among these mortalities 3 were hyponatremic and 3 were hypokalemic. The control group had no reported mortality.

DISCUSSION

Electrolyte imbalances are fairly common in the acute phase of the MI patients. When measured within 48 hours of admission, the sodium and potassium levels were found to be significantly reduced in our study when compared to non-infarct controls.

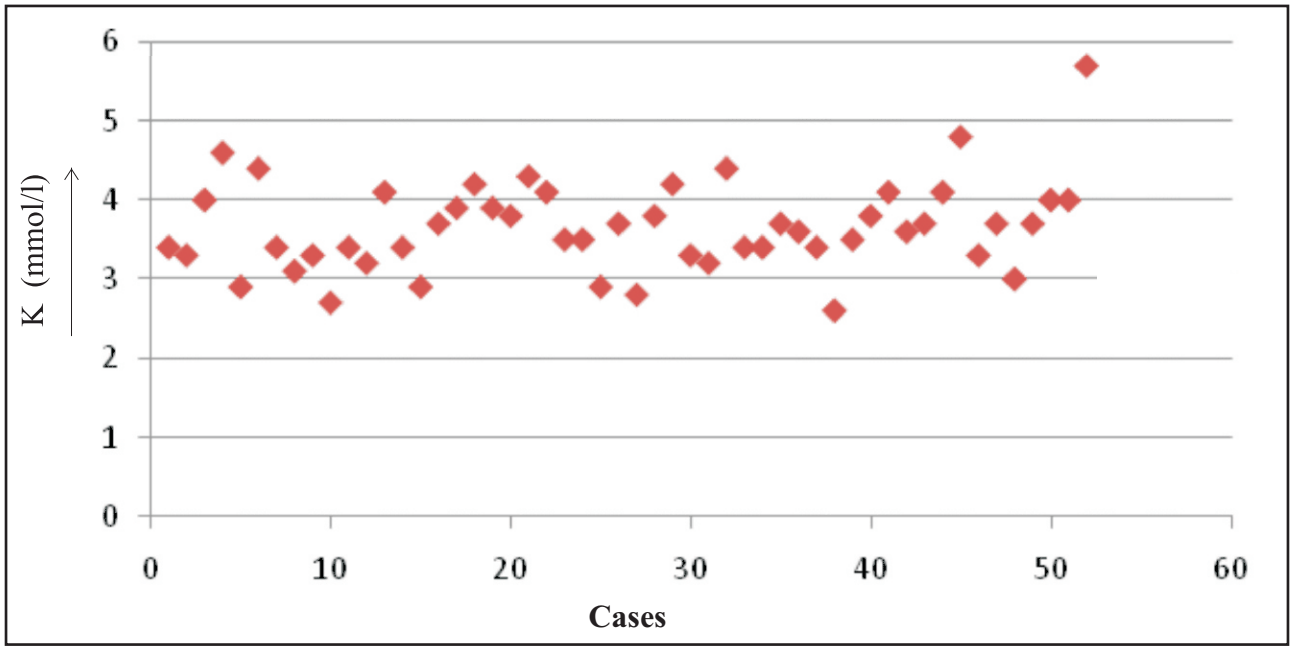
Hypokalemia was evident in a large number of patients. Only one patient with MI had hyperkalemia in the range of 5.2m Eq/l. In MI, hypokalemia has been found to be associated with an increased risk of ventricular tachycardia and ventricular fibrillation.[13] Hypokalemia prolongs ventricular repolarization, often with prominent U waves.[3] The incidence of ventricular fibrillation has been found to be five-fold higher in patients with a low serum potassium.[1] Skeletal muscle acts as a reservoir pool for potassium, maintaining potassium in vital organs such as the heart and brain. This hypokalemia is mostly due to the stress induced catecholamine response (predominantly epinephrine from the adrenal medulla) that function as hormones, in such patients causing increased K^+ uptake into cells.[6,14] The beta 2-adrenoceptor agonists stimulate Na^+/K^+ pump mediated potassium uptake in skeletal muscles of experimental animals and humans.[1]A study done by Goyal et al found the hospital mortality in MI patients to be the least in patients with potassium levels of (3.5-4.5mmol/l), thus showing a relation between the hospital mortality and mean post admission levels.[4] Hyperkalemia too has been shown to be associated with reduced ventricular excitability and other causes of cardiac arrest, like sinus arrest and complete block.

Similar studies done by Goldberg et al observed that hyponatremia on admission or during the first 72 hours of hospitalization in STEMI was independently associated with increased 30-day mortality risk and more post-discharge re-admission for heart failure and death in long-term follow-up. It was similarly found that hyponatremia was more often associated with increased morbidity and mortality in MI patients.[11] The risk of in-hospital mortality is found to increase with the severity of hyponatremia.[11] The relation between the development of hyponatremia and

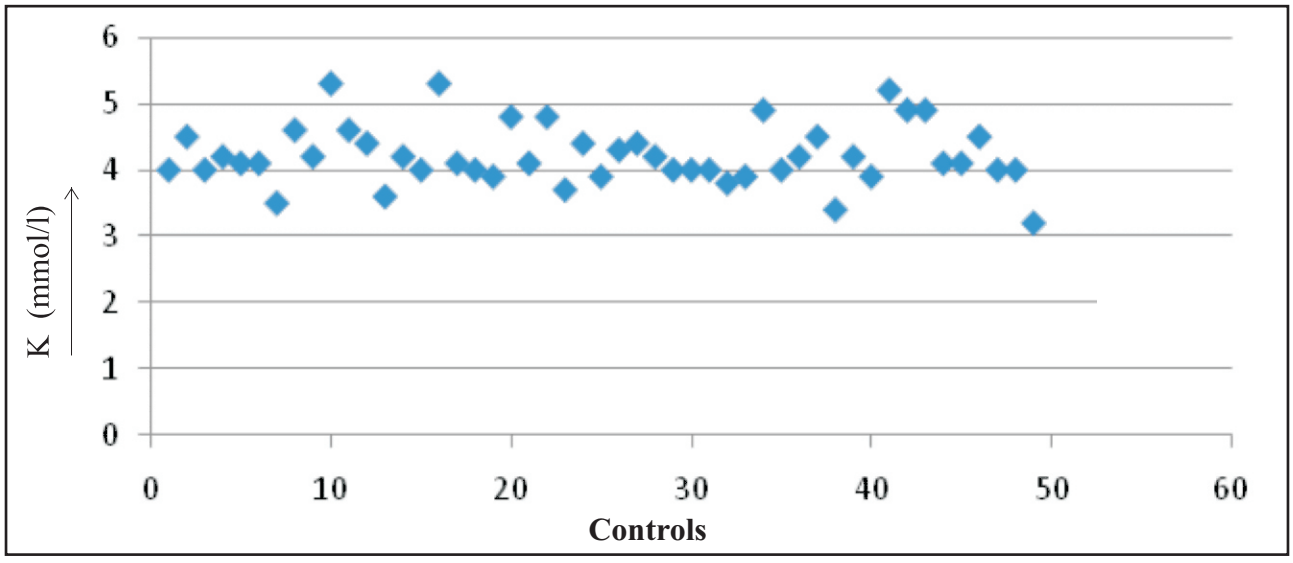
Table 1: Mean values of parameters of cases and controls

Parameter	Controls	Cases	'p' value
Age	55.44+15.5	58.76+12.21	0.234
Sodium	137.5+2.79	135+5.17	<0.05*
Potassium	4.22±4.45	3.66±0.56	<0.001**
Chloride	99.7±1.41	101±7.8	0.301

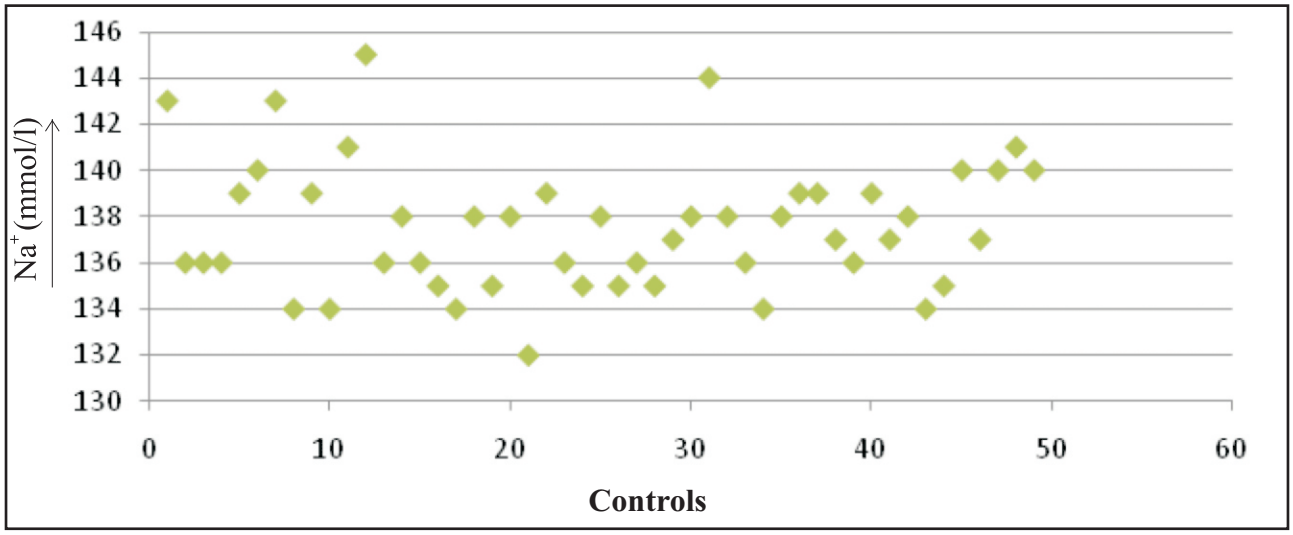
*** Statistically significant**



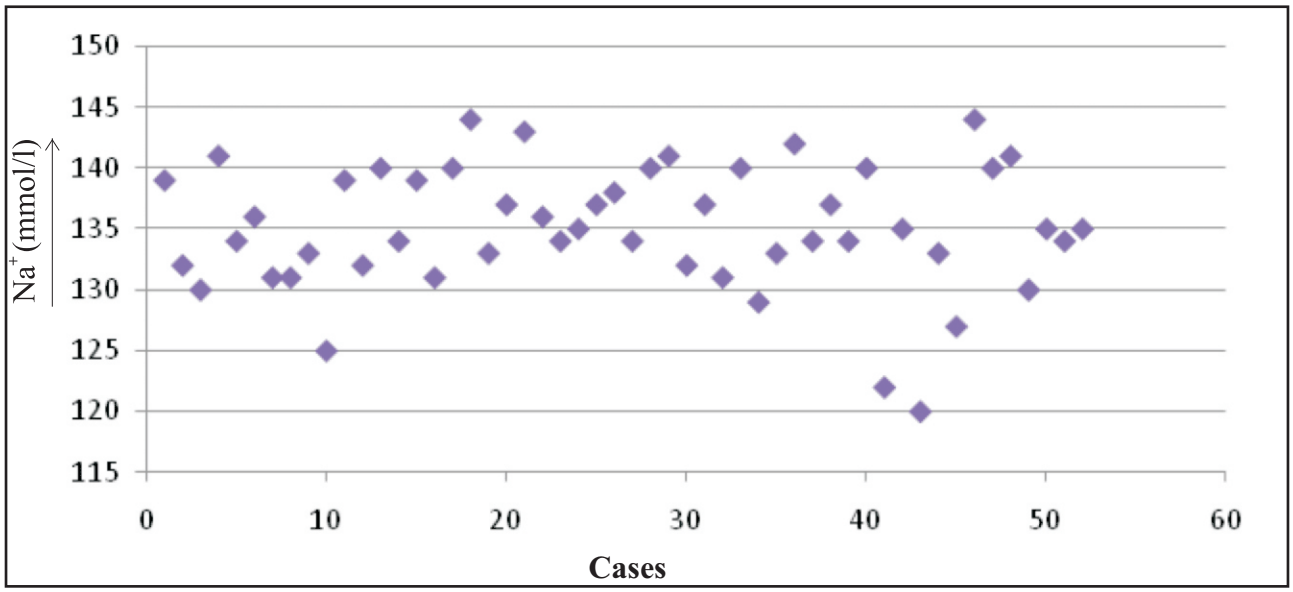
Potassium levels in cases



Potassium levels in controls



Sodium levels of controls



Sodium levels of cases.

the time of myocardial injury seems to be important, because as the time from acute injury increased, the prognostic relation with hyponatremia became less significant.[15] MI patients with hyponatremia were also found to have lower ejection fraction, when compared to non- hyponatremic patients.[11]

A study conducted by Flear et al titled MI defined hyponatremia as below 135 mmol/l found 45 % of infarcted patients to have hyponatremia, and associated increased mortality.[12] Similarly in our study 43 % of MI patients were found to have hyponatremia. Hyponatremia could probably be attributed to the non osmotic secretion of vasopressin, impairing the water excretion causing dilutional hyponatremia. AVP or vasopressin is known to regulate vascular tone and cardiac contraction and may adversely affect cardiac hemodynamics and myocardial remodeling.[2] Sodium is freely filtered by the glomerulus, 70-80% gets reabsorbed in the proximal tubule, 20-25 % in

the loop of henle, in the distal tubules the interaction between the aldosterone with the coupled Na^+ K^+ and Na^+ H^+ exchange systems directly results in the reabsorption of Na from the remaining 5- 10 % of the filtered load. Flear et al had hypothetized that the hypoxia and cardiac ischemia increased the cell membrane permeability to sodium ions, activation of sympathetic nervous system ,and rennin angiotensin system.[12] None of the patients were found to be hypernatremic in laboratory results.

The association of hyponatremia or hypokalemia with early presentation in acute MI, may alert the the clinician about the acuteness and severity of patients illness, since these attributes constitute as substrates for emergence of complications in acute MI patients.

CONCLUSION

- Hypokalemia was evident in a large number of patient in the early phase of AMI, mostly due to the catecholamine response in such patients. Its 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.
- Serum chloride levels showed no particular pattern or significant difference among the two groups.

No specific pattern was observed in the chloride level imbalance. The clinicians are advised to closely monitor these electrolyte changes and correct them as they seem to have adverse effects on the disease outcome.

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