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 results 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 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 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.

HOW TO CITE THIS ARTICLE: Parmar KB, Sharma RN, Shah J. A clinical study of serum electrolytes (NA, K, CL) and serum magnesium levels in patients of acute myocardial infarction. J. Evid. Based Med. Healthc. 2017; 4(61), 3691-3695. DOI: 10.18410/jebmh/2017/736

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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

Financial or Other, Competing Interest: None.
Submission 20-06-2017, Peer Review 01-07-2017,
Acceptance 24-07-2017, Published 31-07-2017.
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DOI: 10.18410/jebmh/2017/736

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 other frequently associated complication of AMI is heart failure, which if present increases the risk of death by at least 3 to 4 fold. Several factors like recurrent myocardial ischaemia, infarct size, mechanical complications, etc. influences the left ventricular systolic dysfunction after myocardial infarction.[5] It has been found that various electrolytes like potassium, sodium, magnesium and chloride etc. plays an important role in the cell metabolism, electrical conduction and membrane excitability. Abnormality of these electrolytes due to any reason can result in significant cardiac life-threatening events.[6] It has been found that until about 60 minutes after the time the cells are known to be irreversibly injured. No significant shift in electrolytes of ischaemic myocardial cells occurs. Although, the clinical importance of these imbalances in ST Elevation Myocardial Infarction (STEMI) in the era of primary intervention has not been fully understood. Following AMI, several electrolyte changes have been reported and levels of these modifiable electrolytes has an important role in altering the prognosis of MI patients. Potassium homeostasis is therefore critical to prevent the adverse events in patients with cardiovascular disease. The role of hypokalaemia and its prevalence in myocardial ischaemia and particularly in MI patients have been under investigation since long.[7] There are several studies, which have shown association between hypokalaemia even mild form of it with increased occurrence of cardiac arrhythmias on AMI patients.[8] Hypokalaemia can be defined as serum potassium levels <3.5 mmol/L, which intern is a determinant of higher morbidity in such patients. In some studies, imbalance in the serum sodium levels has also been recorded in the early phase of MI. On the other hand, there are several studies, which have described hyponatraemia as serum sodium concentration <135 mmol/L as a common finding in hospitalised patients of MI. Flear et al in their study has also found that decreased serum chloride levels to be a common finding in acute MI patients[9] with increased risk of mortality.[10] Magnesium has been implicated in the pathogenesis of acute myocardial infarction and its complications like arrhythmias and also has significant role in other cardiovascular diseases as well. Magnesium ions are considered essential for the maintenance of the functional integrity of the myocardium. Investigations revealed that magnesium level in the blood is decreased in the first 48 hours following an acute myocardial infarction and then increased steadily to reach the normal level in about three weeks' time. The heart muscle subjected to myocardial infarction was found to contain a low magnesium concentration. These findings directly correlated with the resultant complications of myocardial infarction, such as arrhythmias. It has been pointed out 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.

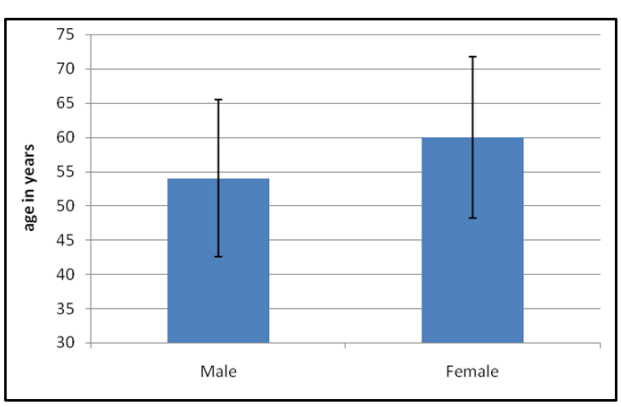
MATERIALS AND METHODS

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



Age Distribution

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.

		Na			K			CL			Mg		
Gender	**Duration**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P valve**
Male (hypertensive)	Pre (Day 1)	138.98	3.25	0.147	4.08	0.80	0.213	96.7	4.82	0.923	2.10	0.20	0.000
	Post (Day 5)	140.8	0.98		4.32	0.36		96.8	2.97		2.55	0.23	
Male (non-hypertensive)	Pre (Day 1)	138.56	3.51	0.383	3.63	0.39	0.003	98.1	1.81	0.001	2.01	0.17	0.000
	Post (Day 5)	140.7	0.78		4.04	0.45		99.3	1.47		2.49	0.22	
Female (with hypertension)	Pre (Day 1)	138.67	2.62	0.051	4.24	0.96	0.585	98.20	2.68	0.018	1.86	0.28	0.001
	Post (Day 5)	140.34	0.98		4.11	0.49		99.04	2.65		2.29	0.39	
Female (with hypertension)	Pre (Day 1)	139.7	1.45	0.002	3.76	0.43	0.000	99.7	3.87	0.228	2.00	0.15	0.000
	Post (Day 5)	140.4	1.29		4.26	0.54		98.6	2.85		2.48	0.24	

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

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.

		Na			K			Cl			Mg		
Gender	**Duration**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Valve**
Male (diabetic)	Pre (Day 1)	136.9	2.88	0.025	4.43	0.75	0.826	98.3	5.71	0.550	1.93	0.36	0.000
	Post (Day 5)	139.8	0.28		4.37	0.16		99.03	3.69		2.35	0.45	
Male (without diabetic)	Pre (Day 1)	139.09	2.95	0.210	3.58	0.65	0.001	99.7	4.03	0.755	1.98	0.16	0.000
	Post (Day 5)	140.9	0.95		4.05	0.45		99.9	2.37		2.45	0.24	
Female (diabetic)	Pre (Day 1)	139.56	3.51	0.239	4.07	0.69	0.220	97.8	4.12	0.755	1.83	0.20	0.000
	Post (Day 5)	140.45	1.18		4.33	0.37		98.05	2.90		2.33	0.31	
Female (without diabetic)	Pre (Day 1)	139	1.39	0.000	3.66	0.39	0.001	98.25	2.42	0.002	1.98	0.17	0.000
	Post (Day 5)	140	1.14		4.06	0.52		99.74	2.88		2.40	0.28	

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

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 nondiabetics.

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.

		Na			K			CL			Mg		
Gender	**Duration**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Valve**
Male (cardiac failure)	Pre (Day 1)	135.5	4.48	0.004	4.08	0.89	0.335	98.4	5.04	0.061	1.95	0.25	0.000
	Post (Day 5)	140.4	1.10		4.27	0.43		99.9	2.76		2.35	0.33	
Male (without cardiac failure)	Pre (Day 1)	139.6	0.92	0.001	3.68	0.39	0.001	98.2	1.02	0.010	2.06	0.16	0.000
	Post (Day 5)	139.3	0.67		4.08	0.44		99.0	1.03		2.53	0.21	
Female (cardiac failure)	Pre (Day 1)	137.67	2.66	0.010	3.90	0.88	0.486	99.82	3.24	0.037	1.82	0.23	0.000
	Post (Day 5)	140.9	0.98		4.02	0.60		97.04	3.26		2.23	0.33	
Female (without cardiac failure)	Pre(Day 1)	139.5	1.31	0.011	3.75	0.18	0.000	98.09	3.42	0.227	2.04	0.13	0.000
	Post(Day 5)	141.67	1.05		4.29	0.29		99.08	2.64		2.55	0.23	

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

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.

		Na			K			CL			Mg		
Gender	**Duration**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Value**	**Mean**	**SD**	**P Valve**
Male (with cardiogenic shock)	Pre (Day 1)	134.56	2.67	0.000	4.07	0.83	0.471	99.6	3.19	0.001	1.94	0.35	0.000
	Post (Day 5)	140.7	1.17		4.22	0.42		96.9	2.17		2.31	0.46	

Male (without cardiogenic shock)	Pre (Day 1)	139.9	2.48	0.529	3.57	0.45	0.000	98.07	1.14	0.018	1.95	0.11	0.000
	Post (Day 5)	140.7	0.96		4.12	0.39		98.95	1.02		2.45	0.19	
Female (with cardiogenic shock)	Pre (Day 1)	135.57	3.16	0.038	3.85	0.84	0.400	96.09	2.74	0.291	1.02	2.74	0.291
	Post (Day 5)	139.09	0.40		4.11	0.48		97.09	1.90		1.03	1.90	
Female (without cardiogenic shock)	Pre (Day 1)	140.5	0.28	0.035	3.68	0.18	0.027	99.7	4.28	0.614	1.99	0.21	0.000
	Post (Day 5)	141.45	0.80		4.12	0.43		98.09	2.67		2.47	0.27	

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

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.[9] 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.[11] Our study also supported the hypothesis that the alterations in the electrolyte levels could prompt the pathological events in coronary heart diseases.[11] Fall in serum electrolyte levels on day of admission in AMI is similar to finding was observed by Shah et al[12] 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.[13] Significant decrease in serum sodium concentration in both sexes was also reported by Flear and Singh,[14] 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.[15] 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[16] (n=50) was 135 ± 5.17, while in Vinod Wali et al[17] (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. Solmon et al and Hulting J et al also observed hypokalaemia[18] 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.[19] Highest incidence of ventricular tachycardia was observed in hypokalaemic group by Dyckner and Erik J. and Solmon 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.[20] 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 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.[21] 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[22] 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

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 predicator of life-threatening arrhythmias occurring in acute myocardial infarction and should be treated.

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