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Original Research Article

Evaluation of renal function tests and serum electrolytes in patients with acute myocardial infarction

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Abstract

Background: Acute myocardial infarction (AMI) is one of the leading causes of morbidity and mortality across the world. Very few studies were done to see the association of renal function tests and serum electrolytes with acute myocardial infarction.

Objectives: To study the renal function tests like blood urea and serum creatinine with serum electrolytes like serum sodium, potassium and chloride in patients with acute myocardial infarction.

Material and Methods: This case control study was carried on 50 AMI patients and 50 healthy controls. 5 ml of blood was drawn from each patient within 6 hrs. of AMI, to estimate blood urea, serum creatinine, serum sodium, serum potassium, serum chloride and cardiac marker CK-MB. The same biochemicals were also determined in 50 age and gender matched controls for comparison.

Results: We found significantly increased levels of blood urea and serum creatinine in AMI patients as compared to controls. We also found statistically significant decreased levels of serum sodium, serum potassium and non-significant difference in the levels of serum chloride in AMI cases as compared to controls.

Conclusion: We found altered changes in the blood urea and serum creatinine in AMI with hyponatremia and hypokalemia which may act as indicator of injury to myocardium. Further studies are needed to correlate all these parameters with the prognosis of AMI.

Keywords: Acute myocardial infarction, blood urea, creatinine, sodium, potassium, CK-MB.

1.Introduction

Acute myocardial infarction (AMI) incidence for those aged 30- 69 years is increased from 274 cases per 100,000 person years in 1999 to a peak of 287 cases per 100,000 person years in 2000, and then reduced every year thereafter, to 208 cases per 100,000 person years in 2008[1]. The prevalence of ischemic heart disease in Indian population was estimated at 96.7 per 1000 individuals in the urban and 27.1 percent in rural areas [2].

AMI is caused by interruption of blood supply to cardiac muscle resulting in ischemic necrosis of the myocardium.Several systemic metabolic changes occur in AMI which include increased plasma concentrations of glucose, free fatty acids, glycerol, cortisol, catecholamine and cyclic AMP. There are reduced levels of triglycerides concentration and an initial decrease in plasma insulin value, followed by an early return to normal level [3]

AMI is one of the manifestations of coronary heart disease which progresses to morbidity and mortality [4]. Hemodynamic abnormalities and arrhythmias in left ventricular dysfunction are the main reasons for mortality along with AMI. Predisposing factors for arrhythmias are autonomic nervous system dysfunction, left ventricular dysfunction, myocardial ischemia, electrolyte disorders and medications [5]. Coronary heart disease is caused by atherosclerosis and plaque formation on the surface of the coronary arteries [6]. These factors cause narrowing of the coronary arteries which lead to insufficient blood flow to heart and in severely compromised condition death is inevitable [7].

Urea is the end product of protein metabolism in humans and more than 90% is excreted by kidney. Creatinine is an endogenously formed substance produced by muscle from creatine and creatine phosphate, with a rate of formation proportionate to the muscle mass. Creatinine is mostly filtered freely by the renal glomerulus, not reabsorbed by the renal tubules, and secreted by the renal tubules only in small amounts. Urea and creatinine investigations are used for assessment of kidney functions [8]. The levels of blood urea and creatinine may increase in many conditions, including decreased cardiac output from cardiac failure [9]. Some studies also found that blood urea and creatinine could be used as an independent and important marker of mortality in patients with acute coronary syndromes forshort to long term of follow up [10-12].

Electrolytes play an important role in cellular function, intermediary metabolism, enzyme activities and electrical gradients [13]. Kidney function tests and serum electrolytes changes have not been studied extensively in AMI and there is less information in the literature in this regard, so the present study was conducted to evaluate serum sodium, potassium, chloride with urea and creatinine level in AMI patients.

2. Material and methods

We conducted this case control study on randomly selected 50 patients of AMI, hospitalized in the Intensive Care Unit (ICU) of Medicine Department, GMERS Medical College, Valsad only after taking informed consent from the study participants. The approval from the Institutional Ethics Committee was duly taken for this research study. The diagnosis of AMI was done by on duty ICU physician based on clinical presentation, electrocardiography (ECG) and biochemical investigation. Fifty, voluntarily willing, consent giving, age and gender matched healthy controls were selected on the basis of following criteria.

2.1 Inclusion criteria

(Different for AMI cases and healthy controls):

For cases:

Hospitalized in ICU with chest pain of at least 20 min duration

CK-MB level above 25 IU/L

ST-segment elevation of at least 2 mm in two or more successive leads of ECG

For controls:

Age and gender matched with cases.

CK-MB level within physiological limits.

No history or clinical/laboratory evidence of AMI.

2.2 Exclusion criteria

(Same for controls as well as cases):

Presence of renal diseases, pulmonary diseases, liver diseases, neoplastic diseases,

Presence of valvular heart disease

Diagnosed case of gout,

Diagnosed with any acute infections,

History of smoking,

Known case of hypertension

Known case of diabetes mellitus

Obesity with body mass index > 32 kg/m2.

Blood samples for the study were taken by venipuncture in plain vaccutainer. Hemolysed and lipemic samples were excluded from the study. All the samples were analysed by Microlab RX 50 diagnostic equipment. Creatine kinase-MB (CK-MB) was estimated by modified IFCC method. Blood urea was estimated by Urease GLDH method. Estimation of serum creatinine was done by Jaffes Kinetic method. Serum electrolytes were estimated by ion selective electrolyte analyzer.

2.3 Statistical analysis

The data of the study was analyzed by Graph pad prism software version 7. Students unpaired 't' test was applied for the comparison of variables between controls and cases. P value <0.05 was considered as statistically significant.

3. Results

Out of 50 cases of AMI, 27(54%) were males and 23(46%) were females as compared to 26(52%) males and 24(48%) females in controls. CK- MB level was significantly elevated in AMI patients as compared to controls. Blood urea and serum creatinine in AMI patients were increased significantly as compared to healthy controls. Statistically significant reduced levels of serum sodium, serum potassium and non-significant difference in the levels of serum chloride were observed in AMI as compared to controls

Table 1: Age and gender wise distribution of cases and controls

Variable	Control	Cases
Number of study subjects(n)	50	50
Mean age in years	46.56± 1.728	47.04± 1.873
Male: female ratio.	27:23	26:24

Table 2: Comparison of variables of controls and cases

Variable	Controls		Cases		't' value	P value	Significance
	Mean	SD	Mean	SD			
Age (years)	46.56	1.728	47.04	1.873	1.33	0.186	Non-significant
CK- MB(U/L)	17.62	3.516	155.1	20.25	47.3	<0.001	Significant
Blood Urea (mg/dl)	28.98	5.016	53.72	4.567	25.79	<0.001	Significant
Serum creatinine (mg/dl)	0.882	0.2624	1.794	0.2461	17.93	<0.001	Significant
Serum sodium (meq/L)	140.1	2.976	131.9	1.729	16.93	<0.001	Significant
Serum potassium (meq/L)	4.628	0.3124	3.7	0.203	17.61	<0.001	Significant
Serum chloride (meq/L)	102.7	2.037	103.3	2.325	1.51	0.1343	Non-significant

4. Discussion

In this research, we found significantly elevated levels of CK-MB in patients of acute myocardial infarction as compared to controls. Being a cardiac marker, increased level of CK-MB in AMI was very well correlated with the ischemic necrosis and infraction of myocardium.

In the present study we observed that blood urea level was significantly increased in AMI cases ($p<0.001$) in comparison with normal healthy controls. These observations were in consistent with that of other studies [10, 12]. Some studies have reported that, increasing urea predicts poor outcome and high mortality rates in subjects with AMI [14]. Increased values of urea act as indicator of renal response to systemic hypo perfusion with respect to decreased cardiac output in decompensated heart failure [15]. Similar type of results were obtained by experimental animal study, where rats have been induced of myocardial infarction by isoproterenol resulted in elevated urea level, probably due to low cardiac output state of ventricular dysfunction [16].

Similar to our results, few studies found increased levels of serum creatinine in AMI as compared to controls [11, 17]. After AMI because of direct leakage of creatine from infarcted myocardial cells, increase in creatine concentration in serum and urine can be predicted [18]. Elevated serum creatinine on admission was an independent predictor of poor myocardial perfusion which ultimately leads to systolic dysfunction in AMI, decreased renal blood flow and decreased GFR. All these phenomenons were responsible for reduced clearance of creatinine in urine and increased value of creatinine in serum [17-19].

We observed that serum sodium level was significantly reduced in AMI cases when compared with normal healthy controls. These findings were in consistent with that of other studies [20,21]. In AMI, vasopressin undergo non-osmotic release due to acute development of left ventricular dysfunction due to pain and stress or may be due to use of analgesics or diuretics resulting in reduced level of sodium.According to one study, decrease in serum sodium level was due to hypoxia,ischemia and infarction resulted in increased permeability of sarcolemma to sodium [22, 23].

The level of serum potassium found to be low ($p<0.001$) in AMI cases as that of healthy control groups. This is in accordance with other studies [20,21,]. Hypokalemia observed in our study may be due to stress induced Catecholamine, that resulting in increased potassium uptake by the cells [24]. This is an acute stress effect and is due to shift of potassium from extracellular to intracellular space and is a result of stimulation of beta2 adrenoceptor agonists linked to sodium-potassium ATPase pump [25].

5. Conclusion

Assessment of renal function tests and estimation of serum electrolytes could be important for AMI in terms of prognosis by evaluation of risk factors like renal dysfunction and electrolyte imbalance. This information could be useful for the treatment of AMI, for the assessment of short to long term mortality in AMI and to prevent further complications.

Conflict of interest: Nil

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