Association of Serum Electrolytes and Urea Levels with Cardiac Markers in Acute Myocardial Infarction

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ABSTRACT

Background: The alterations (increase or decrease) of intracellular and extracellular enzymes or components in developing phase of disease are called markers. Variations in biochemical markers like Cardiac Troponins-I (cTnI), Creatine kinase (CK) and its isoform Creatine kinase-MB (CK-MB) may correlate with the extent of myocardial damage in Acute Myocardial Infarction (AMI). Association of serum electrolytes and urea levels were undertaken in this study as adjunctive parameters along with cardiac markers, which probably helps in better prognosis of AMI.

Materials and Methods: 120 subjects were included in this study and divided equally as study and control groups. Estimation of cTnI, CK, CK-MB, Serum sodium, potassium, chloride and urea were done in all the participants. Data were compared by using student `t' test and chi-square test (SPSS version 17.0).

Results: There were statistically significant decreased levels of serum sodium ($p<0.001$), potassium ($p<0.001$) and elevated levels of urea ($p<0.001$) observed in AMI. But, the levels of serum chloride didn't show any significant correlation between case and control groups.

Conclusion: The present study was conducted to make an effort to find out association of abnormal levels of electrolytes and urea in AMI. Further studies are required to know the role of these correlations in prognosis of clinical condition.

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INTRODUCTION

Coronary heart disease is mainly caused by atherosclerosis and plaque formation on the surface of the coronary arteries[1,2]. These pathological factors causes narrowing of the coronary arteries which lead to inadequate blood flow to heart and if it severely compromised death is inevitable[3,4].

AMI is one the manifestations of coronary heart disease leading to morbidity and mortality. Cardiac biomarkers become detectable in the peripheral blood once the capacity of the cardiac lymphatics to clear the interstitium of the infarct zone is exceeded and spill over into venous circulation occurs[5]. Electrolytes play an important role in intermediary metabolism and cellular function, including enzyme activities and electrical gradients[6].

So, the present study was planned to evaluate serum sodium, potassium, chloride and urea in AMI cases to compare with normal healthy control subjects.

MATERIALS AND METHODS

Study group comprised 120 subjects between 35 to 70 years who were visiting hospital OPD, as well as admitted will be considered. Diagnosis and selection of the 60 cases were made on the basis of recent onset of chest pain, abnormal ECG pattern and by cardiac biochemical markers. The control group included 60 normal healthy subjects visiting hospital for routine check up without any history of chest pain or previous incidence of AMI, hypertension, diabetes, alcoholism and smoking.

Under aseptic conditions about 5 ml of venous blood was drawn and collected in plain vacutainer, after taking informed consent and study was approved by the ethical committee of the institution. Serum was separated by centrifugation at 3000 rpm for 10 minutes at room temperature and the following parameters were estimated.

Estimation of serum cardiac Troponin I (cTnI) was done by chemilumenescence immunoassay (CLIA) on Lumax hormone analyser. Serum Creatine kinase (CK) and its isoform Creatine kinase-MB (CK-MB) evaluated by kinetic methods, also serum urea by Diacetyl Monoxime using Hitachi 902 chemistry analyzer. Serum sodium, potassium and chloride were determined by direct ion selective electrode methods by using Roche AVL 9180 electrolyte analyser.

Statistical analysis were done by student `t' test and chi-square tests (SPSS version 17.0).

RESULTS

The sex wise distribution of AMI & healthy control groups are summarized in Table No. 1, which shows males have more risk than females. Troponin I (Table No. 2) was significantly higher in AMI cases (10.369 ± 9.32, p<0.001) in comparison with healthy control group (0.256 ± 0.2). CK & CK-MB levels are 10 fold higher (1737 ± 1597.29, p<0.001, 206.98 ± 135.93, p<0.001) than healthy control group (87.41 ± 39.43, 23.13 ± 4.74) respectively. The levels of cardiac markers between AMI cases & healthy control group, shows significant higher levels which point towards its significant diagnostic importance.

The electrolytes such as Na^+, K^+ levels in AMI cases (132.51 ± 4.5, 3.88 ± 0.57, p<0.001) shows significant difference when compared with healthy control group (138.96 ± 3.88, 4.43 ± 0.44 respectively). But, Cl^- levels didn't show any difference in both the groups. Moreover, urea values were significantly higher in AMI cases (55.58 ± 28.67, p<0.001) compared to healthy control group (29.66 ± 14.77).

Table No. 1

Group			
Healthy Control Group		AMI Group	
Male	Female	Male	Female
35	25	43	17

Table No. 2

Cardiac Markers	Healthy Control Group (n=60) Mean ± SD	AMI Group (n=60) Mean ± SD	P-Value
Troponin I	0.256 ± 0.2	10.369 ± 9.32	0.001
CPK	87.41 ± 39.43	1737 ± 1597.29	0.001
CKMB	23.13 ± 4.74	206.98 ± 135.93	0.001

Table No. 3

Cardiac Markers	Healthy Control Group (n=60) Mean ± SD	AMI Group (n=60) Mean ± SD	P-Value
Na^+	138.96 ± 3.88	132.51 ± 4.5	0.001
K^+	4.43 ± 0.44	3.88 ±0.57	0.001
Cl^-	103.22 ± 5.19	102.25 ± 4.17	NS
Urea	29.66 ± 14.77	55.58 ± 28.67	0.001

NS = Not Significant

DISCUSSION

Acute myocardial infarction occurs when there is an abnormal ischemic alteration of the myocardium due to an inability of the coronary perfusion to meet the myocardial contractile demand[7]. The diagnosis of a myocardial infarction relies heavily on clinical signs, symptoms, electrocardiogram changes, cardiac enzymes and radiological tests[8].

It is widely accepted that presence of cTnI in blood serum indicates myocardial damage, thus cTnI is considered specific biochemical marker for AMI[9]. CK and CK-MB were also employed in the diagnosis of AMI along with cTnI. In the present study all these marker levels were elevated significantly in AMI case groups and also rate of occurrence was more in males than females.

The high sensitivity and specificity of Cardiac Troponin assay confer great impact in the early diagnosis and risk stratification in patients presenting with chest pain. The majority of Cardiac Troponin is bound to myofilaments and the remainder is free in the cytosol in comparison to CK-MB which is fully cytosolic. In myocyte damage, the cytosolic pool is released which results in elevation of both of these markers[10].

In the present study we observed that serum sodium level was significantly decreased ($p<0.001$) in AMI cases when compared with normal healthy controls. These observations are in consistent with that of Hadeel Rashid Faraj[11]. In AMI, non osmotic release of vasopressin may occur due to acute development of left ventricular dysfunction due to pain and stress or may be due to use of analgesics or diuretics. This could result in low sodium level in blood[12, 13].

The level of serum potassium found to be low ($p<0.001$) in AMI cases as that of healthy control group. This is in accordance with Vinod Wali and singi yatiraj studies[14]. This observed hypokalemia may be due to stress induced Catecholamine, that resulting in increased potassium uptake by the cells[15]. 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 beta-2 adrenoceptor agonists linked to sodium/potassium ATPase[16].

Urea is the end product of protein metabolism. Rise of serum urea is observed in AMI cases of this study. Only few studies have been done about association of urea with AMI. Some studies have reported that, increasing in the urea predicts poor outcome and high mortality rates in subjects with AMI[17]. Elevated levels of urea indicate renal response to systemic hypoperfusion with respect to reduced cardiac output in decompensated heart failure[18]. Similar type of results are obtained by Heraldo Guedis Lobo Filho et al, where experimental rats have been induced of myocardial infarction by isopreterenol resulted in increased urea level, probably related to low cardiac output state of ventricular dysfunction[19].

CONCLUSION

The cTnI, CK, and CK-MB are the ideal markers for the diagnosis of AMI. Serum electrolytes and urea levels may also have important role to identify patients at high risk. Admission of serum sodium, potassium and urea levels could be used as supplemental information with regard to treatment and better prognosis of AMI patients.

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