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Evaluation of Adjuvant Biomarkers in Acute Myocardial Infarction

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Abstract:

Background: *Acute myocardial infarction (AMI) is one of the dreadful complications of cardiovascular disease causing increasing mortality worldwide. 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 - T (ctnl) creatine kinase may correlate with the extent of myocardial damage in Acute myocardial infarction (AMI).* ***Aim and Objectives:*** *This study was undertaken to evaluate serum sodium, potassium, urea and creatinine as adjunctive parameters along with cardiac markers, which probably help in better prognosis of AMI.* ***Material Methods:*** *100 subjects were included in this study with confirmed diagnosis of recent AMI by the physicians and 100 healthy persons visiting hospital for routine checkup. Blood samples of both group were analyzed for serum urea by Diacetylmonoxime, creatinine by Jaffe's, &sodium & potassium by flamephotometry uing Bio- Lab Diagnostic Kit Methods. Whereas cardiac troponin - T was done by chemiluminescence immunoassay (CLIA) on Lumax hormone analyser and CKMB by kinetic kit method.* ***Results:*** *There were statistically significant decreased levels of serum sodium (P<0.0001) potassium (p<0.0001), and elevated levels of urea (p<0.0001) and creatinine (0.0001) observed in AMI. Both established cardiac markers Trop- T and CKMB were extremely statistically significantly increased (p<0.0001) as compared to control group.* ***Conclusion:*** *This study showed association of routine blood tests such as urea, creatinine, sodiumand potassium hence can be used as supplement information with regard to treatment and better prognosis of AMI patients or these could be cost effective time saving adjuvant markers in management of myocardial* infarction.

Keywords: ***Adjuvant, Acute myocardial infarction, electrolytes, urea, creatinine, sodium, potassium.***

Introduction

Acute myocardial Infarction (AMI) is one of the complications of cardiovascular diseases causing increasing mortality worldwide. This disease occurs when blood flow stops to part of heart causing damage to heart muscle and cardiac biomarkers become detectable in peripheral blood.[1]

A good biomarker is something that is easily measured and can be used as a surrogate marker for diagnose or predict risk accurately (high specificity and sensitivity), promptly provide affordable but meaningful results, and should provide this incrementally over existing markers or clinical characteristics. Medications and treatments also come at a cost, therefore simple and cheap tests have become increasingly necessary to decide how to target treatment.[2]

Two well known biomarkers in use of diagnosis of acute myocardial infarction are creatine kinase MB isoform and Cardiac troponin T. In 2000, cardiac Tropnin replaced CK MB as the biomarker of choice for diagnosing a myocardial infarction.[3]

Several systemic metabolic changes occur in AMI.[4] Electrolytes (sodium and potassium) play an important role in intermediary metabolism and cellular function, including enzyme activities and electrical gradients.[5]

So, this study was planned to evaluate serum sodium, potassium, urea, and creatinine along with established markers viz, trop T and CKMB.

Material & Methods:

After obtaining the clearance from Ethical Committee of the institution, the study was carried out on 100 patients with recent onset of acute myocardial infarction admitted in the department of Medicine during the period between May 2015 and October 2017 at Government Medical College & Hospital, Aurangabad, Maharashtra, India.

Study group: 100 patients in study group were selected based on age, sex, duration of symptoms of AMI and history of vascular complications with help of physician's opinion.

Control group: For control group, 100 normal person non-hypertensive with no renal failure, no diarrohea, no vomiting and with no history of AMI were selected.

Informed consent: Informed consent was taken from both subject and control group after explaining the purpose and procedure of study.

Collection of samples:
Collection of 5ml venous blood samples in plain vacutainerwere collected from both the groups on the day of admission within 12 hours under aseptic conditions. Blood was allowed to clot at room temperature for half an hour and then separated at 3000 rpm by using a remi-clinical centrifuge. Serum separated was used for the estimations.

Estimation of serum cardiac Troponin T as done by chemiluminescence immunoassay (CLIA) on Lumax hormone analyser. Creatine kinase (CKMB) was evaluated by kinetic kit method,urea by diacetymonoxime and creatinine by Jaffes kit method, sodium and potassium were measured by flame photometry (Bio-Lab Diagnostic Kit).

The comparative study between controls and AMI patients was carried out.

Results

Table No 1. Shows that the male and female ratio in control group and AMI group,

Group			
Healthy Control group n=100		**AMI Group n=100**	
Male	Female	Male	Female
65	35	72	28

Table No.1: Shows the sex wise distribution of AMI and healthy control groups are summarized and it shows male have more risks than females.

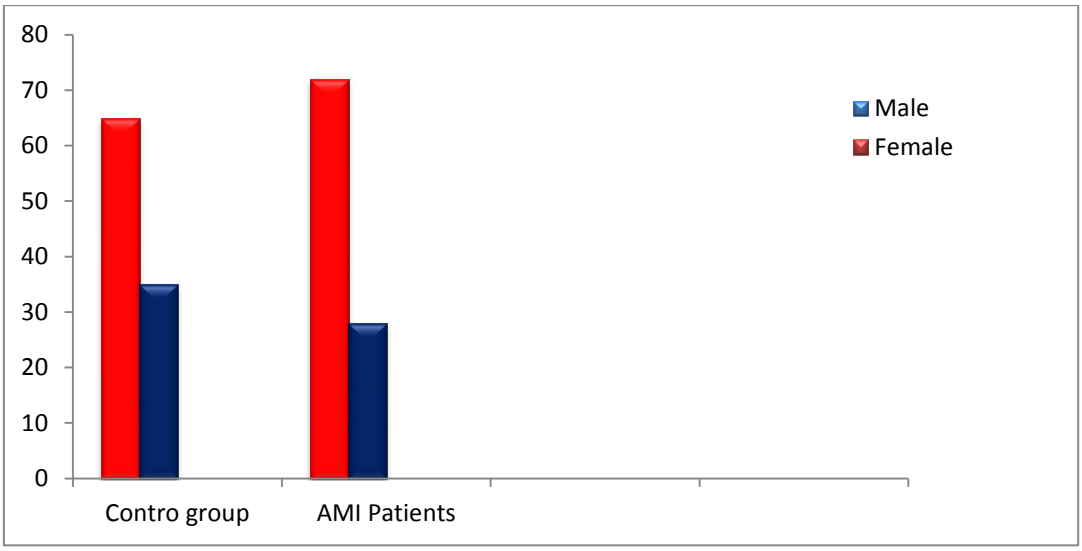

Table No. 2: Shows the activity Troponin and CKMB in AMI patients and control group.

Established Cardiac Markers	Healthy control group (n=100) mean ± SD)	AMI group. (n=100) mean ± SD	P-Value	Std. error
Troponin T	0.256 ± 0.20	12.83 ±4.19	<0.0001	0.41
CKMB	5.99 ± 4.60	234.14± 80.96	<0.0001	0.81

Table No. 2: Shows that the established markers such as Troponin T& CKMB are extremely significant higher (12.83 ± 4.19) and (234.14+ 80.96) with P<0.0001, than healthy control group (0.256 ± 0.20) and (5.99 ±4.60) for Troponin T and CKMB respectively.

Table No. 3: Shows the activity serum urea, creatinine, sodium and potassium in AMI patients and control group.

Established Cardiac Markers	Healthy control group (n=100) mean ± SD)	AMI group (n=100) mean ± SD	P-Value	Std.error
Urea	18.3 ± 2.8	33.18 ± 18	P < 0.0001	1.8
Creatinine	0.67 ± 0.12	1.1 ± 0.92	P<0.0001	0.28
Sodium	141 ± 2.5	133 ± 7.5	P<0.0001	0.79
Potassium	4.37 ± 0.15	3.9 ± 0.80	P<0.0001	0.28

Table no 3 Shows the level of Serum Urea, Creatinine, Sodium and Potassium in AMI patients compare with normal healthy control groups. It shows the activity of serum urea and creatinine was highly significantly increased found in AMI patients compare to control group and found that the serum electrolyte (Sodium and potassium) activity were significantly decreased in AMI patients. Also Shows mean, SD, P -value & Standard error. The routinely done tests such as urea, creatinine, Sodium& Potassium shows significant difference between AMI cases and healthy control group.

The Sodium and Potassium levels are low (133 ± 7.5) and (3.9 ± 0.80) in AMI as compared to healthy group (141 ± 2.5) and (4.37 ± 0.15). Moreover urea and creatinine were significantly higher in AMI cases (P< 0.0001) as compared to healthy group.

Discussion

In the present study, the established markers, Trop. T& CKMB were significantly elevated in AMI as in previous studies.[6&7] This study showed serum sodium levels significantly decreased (P<0.0001) in AMI cases compared to normal healthy group. These observations are in consistent with that of Hadeel Rashid Faraj.[8] RakeshMudarraddi[9] and Flear and Singh.[10] In AMI non osmotic release of vasopression may occur due to acute development of left ventricular dysfunction due to pain & stress or may be due to use of analgesics or diuretics.

The level of serum potassium found to be low (P<0.0001) in AMI cases than that of healthy group. This is in accordance with VinodWali and SingiYatirajstudies.[11] Such lowering of potassium is an acute stress effect and is due to shift of potassium from extracellular to intracellular space and is a result of stimulation space and is a result of stimulation of beta -2 adrenoceptoragonis linked to sodium - potassium ATPase.[12]

Urea is the end product of protein metabolism. Rise of serum urea is observed in AMI cases of this study. Only few studies are done about association of urea with AMI. Elevated levels of urea indicate renal response to systemic hypoperfusion with respect to reduced cardiac output in decompensated heart failure.[13] The elevated levels of serum creatinine are associated with impaired myocardial flow[14] as seen in this study. From this study we concluded in patients with AMI hyponatremia was evident with statistical significance. The routine measurement of renal function test (Urea & Creatinine) along with electrolytes (Sodium& Potassium) thus can be used as adjuvant, affordable markers in diagnosis and treatment of AMI.Further studies are needed to establish the utility of these markers as effective cardiac biomarkers.

References

[1] Oliver MF. Diet and coronary heart disease, Br. Med. Bull, 37 (1):49-58; 1981.
[2] Tunstall - Pedoe H, Vanuzzo D, Hobbs M, Mahonen M, Cepaitis Z, Kuulasmaa K, Keil U: Estimation of contribution of changes in coronary care to improving survival, event rates, and coronary heart disease mortality across the WHO MONICA project populations, Lancet 2000, 355, 688-700.
[3] Alpert JS, Thygesen K, Antman E, Bassand JP: myocardial infarction redefined –a consensus-document of The Joint European Society of Cardiology / American college of cardiology committee for the redefinition of myocardial infarction. J Am coll Cordial 2000, 36: 959-969.
[4] Oliver M P, Metabolic response during impending myocardial infarction, II clinical implications, Circulation, 1974; 45: 491.
[5] Lobo D N: Fluid electrolytes and nutrition: Physiological and Clinical aspects. ProcNutr. Soc. 2004, 63 (3): 453-466.
[6] Ralf Labugger, Lenny organ, et al.: Extensive Tropinin I and T modification detected in serum of patients with AMI. Circulation 2000: 102; 1221-1226.
[7] Mohammed A, Al-Otaiby, Hussein S., Al - Amri, Abdulrahaman M. Al- Moghairi; Journal of Saudi Heart Association (2011) 23, 3-11.
[8] Haded Rashid Faraj- Clinical study of some electolytes with patients in acute syndrome (ACS) in Thi-Qur Governorate, Iraq. Int. J. Curr. Microiol. App. Sci. (2015): 4 (3); 700-705.
[9] RakeshMudarraddi, ShrirangPralhadKulkarni, DhirajJayantilalTrivedi- Association of serum electrolytes and urea levels with cardiac markers in AMI. International Journal of Clinical Biochemistry & Research 2015; 2 (4): 233-235.
[10] Flear CTG, Singh C M. - Hyponatremia and sick cells. Br. J. Anesth. 1973: 45: 976.
[11] VinodWali and SingiYatiraj, - Study of serum sodium & potassium in Acute Myocardial infarction, Journal of clinical and Diagnostic Research, 2014 Nov. Vol-8 (11); cc07- cc09.
[12] Diuretics, Hypokalemia & Cardiac arrhythmias, A critical analysis, VasiliosPapademetrious, Am Heart Journal 1986; 111: 1217-24.
[13] Doron Aronson, HaimHammerman, et al. Serum blood urea nitrogen & long term mortality in Acute ST- elevation myocardial infarction, International Journal of Cardiology; 127(2008) : 380-385.
[14] Zhao L, et al. Elevated admission serum creatinine predicts poor myocardial blood flow and one-year mortality in ST- segment elevation myocardial

infarction patients undergoing primary percutaneous coronary intervention.

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