

Original Article

Evaluation of biochemical parameters in acute myocardial infarction and angina patients

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Abstract

Background: Early and accurate detection of acute myocardial infarction (AMI) is an important unmet clinical requirement. The present study sought to evaluate the levels of N-terminal pro-brain natriuretic peptide (NTproBNP), cardiac troponin I (cTnI), fasting lipid profile, random blood sugar, and serum creatinine in AMI patients compared to angina patients.

Methods: In a single-center, hospital-based, cross-sectional, observational, prospective study conducted at Gandhi Medical College, Secunderabad, from October-2018 to September-2019. A total of 150 patients aged above 40 years with acute chest pain (within 2-6 hours) and who were clinically susceptible of AMI and angina were investigated. The patients were divided into group I (50 AMI patients) and group II (100 angina patients). Levels of all biochemical parameters of blood were assessed. The statistical analyses were performed using the SPSS statistical software, version 15. A student t-test was used to compare the continuous variables between the two groups.

Results: Out of 150 patients, higher male prevalence was found in both the groups (60% and 63%, respectively). Group I had higher levels of NTproBNP (2909±273pg/ml vs. 110±20.74pg/ml, P<0.01), cTnI (2.06±1.3ng/ml vs. 0.006±0.002ng/ml, P<0.01), and fasting lipid profile (total cholesterol:216±41.2mg/dl vs.201±32.5mg/dl, P<0.05, triglycerides:217.7±63.6 mg/dl vs. 175.3±48.8 mg/dl, P<0.01, low-density lipoprotein:141.7mg/dl±41.5 vs. 127.1±30.24 mg/dl, P<0.05, very low-density lipoprotein:43.4±12.8mg/dl vs. 35.1±9.8mg/dl, P<0.01) than group II, except low levels of high-density lipoprotein (31.2±3.83mg/dl vs. 38.9±4.32 mg/dl, P<0.01).

Conclusion: Assessment of NTproBNP, cTnI, and fasting lipid profile may aid in the early diagnosis of AMI and its management.

Keywords: Biomarkers, Diagnosis, Myocardial Infarction; Stable Angina, India

Background

Acute myocardial infarction (AMI) is the leading cause of death and morbidity worldwide. Recently, India has had the highest disease burden of AMI, with 6-9% in-hospital mortality rates [1,2]. AMI is characterized by reduced coronary artery reperfusion attributed to stenosis or distal embolization of the thrombus and abrupt total obstruction of a coronary artery caused by thrombosis. The main clinical manifestations of AMI include acute chest discomfort and prolonged ST-segment elevation on an electrocardiogram (ECG). Though, a small

percentage of patients have no symptoms or changes in their ECG [3]. As ECG has limited sensitivity and specificity for the diagnosis of AMI, the European Society of Cardiology (ESC) and the American College of Cardiology (ACC) established AMI criteria. To be diagnosed with AMI, a patient must exhibit at least two of the following traits: a) characteristic elevation in cardiac markers (such as creatinine kinase-MB (CK-MB) isoenzymes), particularly serum cardiac troponins I or T (cTnI or cTnT), and b) ECG changes with ST elevations and abnormal Q waves [4-6]. Though AMI causes significant morbidity and mortality, measuring a variety of cardiac biomarkers is of paramount importance for early diagnosis, risk classification, and tailoring appropriate treatment in the management of these high-risk patients. The optimal

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biomarkers for identifying myocardial injury are exhibited at significantly higher levels inside cardiac tissue, have good clinical sensitivity and specificity, and can be detected in the bloodstream early following the commencement of clinical manifestations such as chest pain [3]. Against this background, the present study was done to assess the levels of N-terminal prohormone of brain natriuretic peptide (NTproBNP), cTnI, fasting lipid profile, serum creatinine, and random blood sugar (RBS) in AMI patients as compared to angina patients.

Methods

Study design

A single-center, hospital-based, cross-sectional, observational, prospective study was conducted from October-2018 to September-2019 at Gandhi Medical College, Secunderabad. A total of 150 patients aged above 40 years with acute chest pain (within 2-6 hours) and who were clinically susceptible of AMI and angina were included in the study. Patients with known cases of heart failure and chronic kidney disease were excluded. Two-dimensional echocardiography was performed at admission to exclude cases of heart failure. The patients were segregated into group I (50 patients with AMI) and group II (100 patients with angina). The levels of NTproBNP, cTnI, fasting lipid profile, RBS, and serum creatinine were compared between both groups.

Sample Collection

After obtaining informed consent, 5ml of venous blood sample was collected within 6 hours of chest pain for NTproBNP, cTnI, RBS, and serum creatinine assays. Another 5ml of blood sample was collected after 10-12 hours of fasting to assay the fasting lipid profile by venipuncture into plain red-color vacutainer tubes under aseptic conditions. The collected blood sample was sent for analysis. Then, the blood was allowed to stand for about 30 minutes at room temperature. It was centrifuged at a rate of 3000 rpm, and serum was separated and analyzed for the following parameters: NTproBNP, cTnI, fasting lipid profile [total cholesterol, triglycerides, high-density lipoprotein (HDL), low-density lipoprotein (LDL), and very-low-density lipoprotein (VLDL)], RBS, and serum creatinine. NTproBNP was assayed by immunosandwich method with final detection by enzyme-linked fluorescent assay and analyzed in BiomerieuxVidas autoanalyzer. cTnI was assayed by immunometric immunoassay and analyzed in Vitros 5600 autoanalyzer. Total cholesterol was assayed by the cholesterol oxidase peroxidase method and analyzed in Beckman Coulter AU 5800 analyzer. Triglycerides were assayed by the Glycerol-3-Phosphate Oxidase method and analyzed in Beckman Coulter AU 5800. HDL was analyzed by Beckman Coulter AU 5800. LDL was assayed by the inbuilt calculated method by Friedewald equation and analyzed in Beckman Coulter AU 5800. RBS was assayed by the Hexokinase method and analyzed in Beckman Coulter AU 5800. Serum creatinine was assayed by the modified Jaffe method and analyzed in Beckman Coulter AU 5800.

Statistical analysis

Continuous variables are described as mean and standard deviation. Categorical variables are represented as numbers and percentages. Comparison of age and biochemical parameters

among the two groups were performed using the student t-test. A p-value of <0.05 was considered statistically significant. The statistical analyses were performed using the SPSS statistical software, version 15 (Statistical Package for the Social Sciences, Inc., Chicago, Illinois, USA).

Results

Out of 150 patients, 50 AMI patients were allocated to the group I, and 100 angina patients were assigned to group II. A higher male prevalence was found in both groups (60% and 63%, respectively). The majority of the patients in both groups were in the sixth decade of life (Figure 1). Duration of diabetes was 5-10 years in most patients of group I, while <10 years in group II (Figure 2). Non-diabetic patients were more prevalent in group II (15%) than in group I (4%).

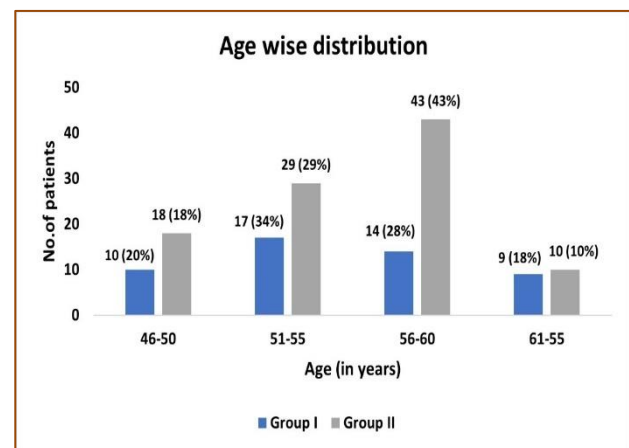


Figure 1: Distribution of patients by age between the two groups

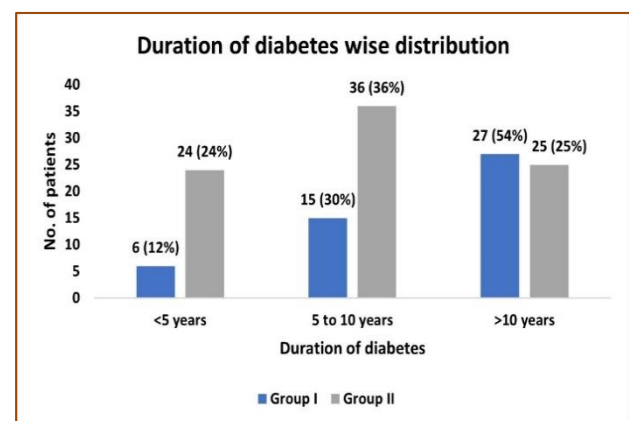


Figure 2: Distribution of patients by diabetes duration between the two groups

Table 1 represents the demographic characteristics of study groups. There was no significant difference found between the mean age of the study population (group I: 55.06 ± 5.01 vs. group II: 54.9 ± 4.24 years, $p=0.9084$). Group I had significantly higher levels of serum NTproBNP (2909 ± 273 pg/ml vs. 110 ± 20.74 pg/ml, $p=0.0001$), serum cTnI (2.06 ± 1.3 ng/ml vs. 0.006 ± 0.002 ng/ml, $p=0.0001$), and fasting lipid profile (total cholesterol: 216 ± 41.2 mg/dl vs. 201 ± 32.5 mg/dl, $p=0.0162$, triglycerides: 217.7 ± 63.6 mg/dl vs. 175.3 ± 48.8 mg/dl, $p=0.0001$, LDL: 141.7 mg/dl vs. 127.1 ± 30.24 mg/dl, $p=0.0001$, and VLDL: 43.4 ± 12.8 mg/dl vs. 35.1 ± 9.8 mg/dl, $p=0.0001$) than group II, except low levels of HDL (31.2 ± 3.83 mg/dl vs. 38.9 ± 4.32 mg/dl, $p=0.0001$).

Table 1: Demographic characteristics of study groups

Characteristics	Group I: AMI patients (n=50)	Group II: Angina patients (n=100)	P value
Age, years	55.06 ± 5.01	54.9 ± 4.24	0.9084
Serum NTproBNP, pg/ml	2909 ± 273	110 ± 20.74	0.0001
Serum troponin I, ng/ml	2.06 ± 1.3	0.006 ± 0.002	0.0001
Fasting lipid profile			
Total cholesterol, mg/dl	216 ± 41.2	201 ± 32.5	0.0162
High-density lipoprotein, mg/dl	31.2 ± 3.83	38.9 ± 4.32	0.0001
Triglycerides, mg/dl	217.7 ± 63.6	175.3 ± 48.8	0.0001
Low-density lipoprotein, mg/dl	141.7 ± 41.5	127.1 ± 30.24	0.0001
Very low-density lipoprotein, mg/dl	43.4 ± 12.8	35.1 ± 9.8	0.0001
Random blood sugar, mg/dl	202.2 ± 61.3	187.9 ± 39.3	0.0836
Serum creatinine, mg/dl	0.880 ± 0.191	0.892 ± 0.2	0.7212
† Data are presented as mean ± S.D.			
NTproBNP: N-terminal prohormone of brain natriuretic peptide			

Table 2: Comparison of NTproBNP levels of the present study with previous studies

Studies	Study patients	Serum NTproBNP, pg/ml
Present study	AMI vs. angina	2909 ± 273 vs. 110 ± 20.74
Ezekowitz et al.(2006) [9]	AMI patients with duration of symptoms <2 hours, 2-4 hours, >4hours	113 (50-245), 246 (83-711) and 464 (158 - 2174)
Kasap et al. (2007) [10]	AMI vs. healthy	1432.17 ± 140.64 vs. 93.23 ± 3.25
Khan et al. (2008) [11]	AMI (survivors vs. dead)	700.2(0.3–11485.3) vs.5781.3 (1.4–10835.9)
Haaf et al. (2011) [12]	AMI patients vs. other final diagnoses	886 vs.135
Drewniak et al. (2015) [13]	AMI patients	4773 ± 8807

Discussion

The quantitative data collected in this research has shown that the present study scrutinized the diagnostic value of NTproBNP, cTnI, fasting lipid profile, RBS, and serum creatinine in AMI patients compared to angina patients and reported the significance of NTproBNP, cTnI, and fasting lipid profile with the potential to improve early diagnosis in AMI patients.

Significance of NTproBNP in AMI patients

The release of NTproBNP into the bloodstream is triggered by increased wall stretching due to ventricular wall stress. This is directly related to the diameter of the chamber and the transmural pressure but inversely related to the thickness of the wall. The increase in chamber diameter and transmural pressure in AMI patients contributes to the elevation of natriuretic peptides. The level of NTproBNP rises soon after infarction and peaks after 24 hours, thereby relating to the extent of the infarct [7]. Furthermore, its level increases after five days in AMI patients with complications such as cardiac failure. After 24 hours of symptom onset, NTproBNP remains a prognostic factor in addition to cardiac troponin when assessed upon admission [8]. The present study reported significantly higher values of NTproBNP in AMI patients than in angina patients (2909 ± 273 pg/ml vs. 110 ± 20.74 pg/ml, p=0.0001). This result is satisfactory when compared to past studies. The comparison of values of NTproBNP among these studies is shown in Table 2 [9-13]. All these studies also substantiated the significance of higher levels of NTproBNP as a marker of poor cardiac function in AMI patients.

Significance of cardiac troponin I in AMI patients

The cardiac troponin increases at 3-8 hours, peaks at 12-24 hours, and remains elevated for about ten days. Though the use of troponin for diagnosing AMI and risk stratification to facilitate decision-making has transformed the treatment of patients with chest pain, the 12-hour delay for the levels to peak remains a problem for this biomarker [14]. The test approach employed in the present investigation was exceptionally sensitive troponin I with an AMI cutoff of 0.012 ng/ml. Statistical analysis of the acquired findings revealed that serum troponin I readings are significantly higher in acute AMI patients (2.06 ± 1.3 ng/ml) than in angina patients (0.006 ± 0.002 ng/ml). Keller et al. [15] reported that troponin I is a more sensitive assay that improves early identification of AMI and risk stratification, consistent with the present study's findings. The present study also supports the results of Reichlin et al. [16] and Daubert et al. [17]. They stated that troponin I is a highly sensitive marker of myocardial necrosis that is requisite for identifying MI in a clinical situation where ischemia is present. Significance of fasting lipid profile in AMI patients

Current prospective cohort studies have propounded that lipid abnormality is linked to an increased risk of cardiovascular events, and LDL, HDL, and triglycerides are all critical determinants of cardiovascular disease [18, 19]. However, there is a paucity of evidence of the significance of serum lipid assessments in AMI patients. Thus, in the present study, we have found a significant increase in total cholesterol (216 ± 41.2 mg/dl vs. 201 ± 32.5 mg/dl), triglycerides (217.7 ± 63.6 mg/dl vs. 175.3 ± 48.8 mg/dl), LDL (141.7 mg/dl ± 41.5 vs. 127.1 ±

30.24 mg/dl), and VLDL (43.4 ± 12.8 mg/dl vs. 35.1 ± 9.8 mg/dl), whilst there was a significant decrease in HDL (31.2 ± 3.83 mg/dl vs. 38.9 ± 4.32 mg/dl) in AMI patients than angina patients. This finding is concordant with those of other studies investigating fasting lipid profiles in an AMI population that found a direct association between total cholesterol, triglycerides, LDL, and VLDL and an inverse relationship between HDL and AMI disease, as delineated in Table 3 [18, 20, 21].

The present study's findings are expected to weigh in on the discussion about the management of AMI patients. There are several limitations to the study that need to be acknowledged. First and foremost, because this was a single-center study with small sample size, it is not representative of the general population. Furthermore, the conclusion of this study was constrained by an inadequate history and a shorter study period. Further large, well-designed studies, especially in different ethnic populations, are warranted.

Table 3: Comparison of fasting lipid profile of the present study with previous studies

Studies	Present study	Narayana et al. (2011) [18]	Shirafkan et al. (2012) [20]	Kumar et al. (2019) [21]
Study patients	AMI vs. angina	AMI vs. stable angina	AMI vs. normal controls	AMI (Within 24 hours and after 48 hours)
Total cholesterol, mg/dl	216 ± 41.2 vs. 201 ± 32.5	224.02 ± 14.92 vs. 202.39 ± 15.72	201.86 ± 44.24 vs. 181.37 ± 15.32	207.5 ± 30.5 vs. 192.4 ± 49.3
High-density lipoprotein, mg/dl	31.2 ± 3.83 vs. 38.9 ± 4.32	31.82 ± 4.49 vs. 44.03 ± 5.37	40.62 ± 5.18 vs. 61.57 ± 11.59	46.6 ± 9.9 vs. 40.7 ± 11.8
Triglycerides, mg/dl	217.7 ± 63.6 vs. 175.3 ± 48.8	165.03 ± 20.09 vs. 125.09 ± 20.02	168.69 ± 17.34 vs. 123.69 ± 31.71	153.8 ± 10.2 vs. 183.8 ± 14.8
Low-density lipoprotein, mg/dl	141.7 ± 41.5 vs. 127.1 ± 30.24	160.08 ± 18.27 vs. 137.02 ± 20.75	117.03 ± 22.67 vs. 87.57 ± 20.78	149.0 ± 41.2 vs. 133.4 ± 54.0
Very low-density lipoprotein, mg/dl	43.4 ± 12.8 vs. 35.1 ± 9.8	32.89 ± 4.12 vs. 24.83 ± 4.03	33.73 ± 3.46 vs. 24.73 ± 6.34	-

Conclusion

Higher levels of NT-proBNP, cTnI, and fasting lipid profile (total cholesterol, triglycerides, LDL, and VLDL), as well as lower levels of HDL, were found in AMI patients. Thus, measurement of NTproBNP, cTnI, and fasting lipid profile may provide crucial prognostic information in the evaluation of AMI patients.

Abbreviation

ACC: American College of Cardiology; AMI: acute myocardial infarction; CK-MB: creatinine kinase-MB; cTnI or cTnT: cardiac troponins I or T; ECG: electrocardiogram; ESC: European Society of Cardiology; HDL: high-density lipoprotein; LDL: low-density lipoprotein; NTproBNP: N-terminal prohormone of brain natriuretic peptide; RBS: random blood sugar; VLDL: very-low-density lipoprotein

Declaration

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Availability of data and materials

Data will be available by emailing cvsarada2@gmail.com

Authors' contributions

All authors equally contributed to the concept, design, literature search, data analysis and data acquisition, manuscript writing, editing, and reviewing. All authors have read and approved the final manuscript.

Ethics approval and consent to participate

We conducted the research following the Declaration of Helsinki. Ethical permission was granted by the Institutional Ethics Committee of Gandhi Medical College, Secunderabad (Ref No. IEC/GMC/2019/03/14) on 01st August 2019. All patients gave written informed consent.

Consent for publication

Not applicable

Competing interest

The authors declare that they have no competing interests.

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