To Assess the Study of Pro- BNP, CPK -MB and Troponin Level in Acute Myocardial Infarction

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Abstract

Introduction: One of the greatest causes of death and morbidity in the world is acute myocardial infarction (AMI). Atherosclerotic coronary artery disease (CAD) with plaque erosion or breakup, causing transient, partial or total arterial occlusion, is the most common cause of AMI. Without appropriate blood flow, the heart cannot continue to function, and if it is severely compromised, death is inevitable. Simple and objective measures of cardiac function are brain natriuretic peptide (BNP) levels. These measurements can be used to diagnose heart failure, including diastolic dysfunction, and have been shown to save money by using them in the emergency room setting. For the diagnosis of acute myocardial infarction, the most sensitive and most precise measure available is creatine phosphokinase (CPK-MB). The main biomarker for AMI diagnosis is cardiac troponin I. The level of troponin may also be elevated with important prognostic value in many other disorders, including heart failure.

Methods: This study was a case-control study, conducted in the Medicine and cardiology Department at DMMC & SMHRC, Nagpur in collaboration with ABVRH, Sawangi (Meghe) during Nov 2020 to Jan 2020. Total 80 individuals were considered for the study, 40 each in case and 40 in control groups.

Results: As present study show the Pro-BNP Levels were increased significantly (P<0.001) in the AMI (2200±601.30) as compared in controls (90.20±3.20). The mean values of serum CK-MB and troponin I were significantly (P<0.001) higher in the AMI (165±47.21, 1.48±0.45) as compared to those in the healthy controls (11.50±0.80± 3.250, 0.03±0.01) respectively.

Conclusions: These findings therefore indicate that the combined detection of CK-MB, Troponin I and NT-Pro-BNP levels will contribute significantly to the early diagnosis of AMI. It can also provide the clinic with diagnostic evidence and thus reduce AMI mortality in the acute phase.

Key words: *AMI, N-terminal Pro-BNP, CPK-MB, Troponin I.*

Introduction

In the acute phase of myocardial infarction, post-infarction heart failure is a growing medical and epidemiological issue, especially in the community of patients over 65 years of age, despite the increasingly successful treatment available.[1]

Easy and objective measurements of cardiac activity are brain natriuretic peptide (BNP) levels. These measurements, including diastolic dysfunction, can be used to detect heart failure and have been shown to save money by using them in the emergency room setting. To rule out heart failure, the high negative predictive value of BNP tests is especially helpful. Treatment with angiotensin-converting enzyme inhibitors, angiotensin-

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II receptor blockers, spironolactone and diuretics decreases the level of BNP, which means that BNP testing can play a role in tracking heart failure patients. Patients with chronic, stable heart failure, however, can have levels above the normal range (i.e. less than 100 pg/mL of BNP and less than 125 pg/mL of Pro-BNP N-terminal for patients younger than 75 years). Increases in BNP levels may be due to intrinsic heart disease or may be caused by secondary causes, such as pulmonary or renal disorders (e.g., chronic hypoxia). Other tests of cardiac status, such as the New York Heart Association ranking, are associated with BNP examinations. In patients previously diagnosed with heart failure or heart dysfunction, the BNP level is a good predictor of the risk of death and cardiovascular events.[2]

In order to maintain stable blood pressure and plasma volume, and to prevent excess salt and water retention, the heart secretes natriuretic peptides as a homeostatic signal. In the atrial myocardium of rats, an auric natriuretic peptide (ANP) was initially identified.[3]

Due to their stretching, volume overload and elevated fi ling pressure, BNP is released from cardiac myocites Both of these activities result in high wall stress that initiates the release of the BNP precursor Pre-Pro-BNP- it first cleaves to pro-BNP, then to the biologically active BNP and the inactive amino terminal fragment, BNP-NT-Pro-BNP N-terminal prohormone. BNP release is part of a compensatory operation in the failed heart, such as renin-angiotensin-aldosterone system (RAAS) activation and sympathetic nervous system activation. In addition to its function as an intense mechanical pump, the heart has now become a new endocrine organ—the heart communicates its pain by releasing BNP. In various trials in clinical and epidemiology. The direct association between the reduction of the left ventricle's systolic function and the elevation of natriuretic peptides has been shown, allowing for a potential biochemical diagnosis of heart failure. BNP levels are up to 100 pg/ml in the heart with good systolic activity. If the level of BNP is 100-500 pg/ml, further diagnostic evaluation is required.[4] For the diagnosis of acute myocardial infarction, the most sensitive and most precise measure available is creatine phosphokinase-MBB (CPK-MB). With the exception of post-cardiac surgery, the degree and duration of serum CPK-MB elevation approximates the level of acute myocardial infarction, although a number of factors can undermine the reliability of such an index. Conflicting documentation concerning the presence of CPK-MB in tissues other than myocardium and the release of CPK-MB under conditions other than acute myocardial infarction has been provided by differences in fractionation and assay methods for creatine phosphokinase isoenzymes. Also worthy of consideration is the embryological growth of the CPK-MB isoenzymes, as well as the different conditions involving increased serum CPK-BB activity.[5]

While CPK-MB is of great benefit in the environment of a coronary care unit (ccu), following cardiac surgical procedures, it is less useful. CPK-MB is normally elevated postoperatively for procedures including coronary bypass grafting, valve replacement or reconstruction of congenital defects. The release of CPK-MB occurs intraoperatively, and in 74 percent of cases, CPK-MB has been reported back to normal levels 18 hours after surgery.[6,7]

The creation of delicate and precise biochemical markers of myocardial injury (cardiac troponin) has helped to diagnose and treat acute myocardial infarction in recent years (MI). The diagnostic criteria for acute MI were redefined by the European Society of Cardiology (ESC) and the American College of Cardiology (ACC) to include the measurement of cardiac troponin as the gold standard.[8] As a result of these recommendations and the increased availability of troponin assays, the diagnosis rate of MI may increase,[9] Which is likely to influence the treatment and epidemiology of patients.[10]

In addition to its diagnostic application, the ACS setting is prognostic, clinically actionable, and can be tracked as a proxy for progress during therapy. Troponin is a valuable biomarker of prognosis in acute decompensated heart failure (AHF), but it does not help in the diagnosis of AHF, and its function as an actionable biomarker is uncertain. Guidelines suggest that troponin levels be assessed in patients presenting with AHF for evaluation of the event of ACS as well as for assessment of prognosis and seriousness of disease.[11]

Materials and Methods

This study was a case-control study conducted between Nov 2020 and Jan 2020 in the Department of Medicine and Cardiology at DMMC & SMHRC, Nagpur

in collaboration with ABVRH, Sawangi (Meghe). For the analysis, a total of 80 participants were considered, 40 in each case and 40 in the control groups.

Inclusion criteria for myocardial infraction:

Patient suffering for Myocardial infraction (AMI).

Exclusion criteria for myocardial infraction:

Patients with chronic diseases such as tuberculosis, HIV, cirrhosis of the liver, acute & chronic kidney failure, etc.

Sample Collection:

5ml Blood sample was collected from each subject by venipuncture with standard blood collection technique in a plain vial for serum separation.

Biochemical Analysis

Pro-BNP, CK-MB, and Troponin I were detected by chemiluminescence immunoassay (CLIA)in 40 AMI patient (AMI group) and 40 Non-AMI Subject (control group).

Result

Table 1: Percentage wise distribution of subjects in Control Group & Study Group.

Sr. No	Group	No of patient	Percentage (%)
1	Control group (Healthy Individuals)	40	50%
2	Study group (AMI Patients)	40	50%
	Total	80	100%

Table 1: Shows percentage wise distribution of control group and study group. The present study includes total 80 subjects. Total 80 subjects were divided into two groups: Control Group consists of 40 (50%)) Healthy Individuals and study group consists of 40 (50%) (AMI Patients)

Table No 02: The BNP, CPK-MB, Trop I Levels

Parameter	Study group (MI patient)	Control group (Healthy subject)
Pro-BNP (pg/ml)	2200±601.30	90.20±3.20
CPK-MB(IU/L)	165±47.21	11.50±0.80
Trop I(ng/ml)	1.48±0.45	0.03±0.01

As shown in table no. 2 The BNP Levels were increased significantly ($P<0.001$) in the AMI (2200±601.30) as compared in controls (90.20±3.20). The mean values of serum CK-MB and troponin I were significantly increase ($P<0.001$) higher in the AMI (165±47.21, 1.48±0.45) as compared to those in the healthy controls (11.50±0.80± 3.250, 0.03±0.01) respectively.

Discussion

Natriuretic hormones are a family of vasoactive peptides that, as their name implies, promote natriuresis and diuresis, which serve as healthy arterial and venous vasodilators. The ability to measure the circulating concentration of these hormones has led to an interest in enhancing the diagnostic and prognostic evaluation of cardiovascular disease patients through the use of these

levels.[12,13]

Sabatine et al. It has been shown to be closely associated with the severity of an acute ischemic insult and the amount of Pro-BNP circulating.[14]

In this study, we observed increased levels of Pro-BNP relative to healthy populations in patients with AMI. Among the patient classes, Pro-BNP levels were found to be the highest in the cardiovascular patient population,

In interpreting myocardial infarction, CK and, more specifically, its isoenzyme CK-MB also have a formal function. The new definition, however, is not very beneficial because studies have shown that patients with myocardial infarction and unstable angina have similar results, as currently described.[15,16]

As expected, in patients with AMI, CK-MB levels were higher than in the healthy population in the current study. According to this study, CK-MB therefore appears to be a better predictor of MI compared to Troponin, especially within the first few hours of MI. There were significantly higher CK-MB levels comparison with the control group ($p<0.05$). Troponin I is a complex troponin-regulatory protein involved in contractility of the heart. Troponin I have a very high myocardial tissue specificity, is not detectable in the blood of healthy people, and, compared to a mixture of ECG and traditional biochemical markers, provides improved sensitivity and specificity for AMI. Cardiac-specific troponin are highly sensitive and reliable markers of myocardial injury.[17] and therefore cardiac troponins are the preferred markers for the diagnosis of myocardial infarction.[18] In this study, AMI patients were found to have increased levels of troponin I as compared to healthy controls, as predicted.

Conclusion

These findings have therefore indicated that the combined detection of CPK MB, Troponin I and NT-Pro-BNP levels will contribute significantly to the early diagnosis of AMI. It can also provide clinical diagnostic evidence and thus lower AMI mortality in the acute stage.

Conflict of Interest: Nil

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Ethical Clearance: taken from institutional ethics committee

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