# Correlation of Inflammatory Marker with Glycogen Phosphorylase BB (GPBB) in Patients of Acute Myocardial Infarction

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## ABSTRACT

**Introduction:** Acute myocardial infarction (AMI) is a significant cause of morbidity and mortality worldwide, which results from occlusion of coronary artery. Glycogen phosphorylase BB (GPBB) is a new marker invented for early diagnosis of AMI. AMI is associated with profound systemic inflammatory response and inflammatory marker C-reactive protein (CRP) has been found to increase after AMI. The objective of the present study was to investigate the correlation between inflammatory marker i.e. C-reactive protein (CRP) and cardiac marker i.e. glycogen phosphorylase BB (GPBB) in AMI patients.

**Material and Methods:** The present study includes 150 AMI patients and 100 normal healthy individuals as controls. In all the cases and controls, GPBB levels were measured by ELISA where as CRP levels were measured by diagnostic kit supplied by ERBA.

**Results:** The mean levels of both CRP and GPBB were significantly increased in AMI cases as compared to controls. Also, there was significant positive correlation between GPBB and CRP in AMI cases.

**Conclusions:** The positive correlation between CRP and GPBB in AMI indicates that there occurs inflammation in acute myocardial infarction patients. Hence it is concluded that CRP is a risk marker of AMI and can be used for diagnostic purpose in patients of AMI.


**Keywords:** Acute Myocardial Infarction, Glycogen Phosphorylase BB, C-reactive Protein

## INTRODUCTION

Acute myocardial infarction (AMI) is the significant cause of morbidity and mortality worldwide.[1] Acute Myocardial infarction, more commonly known as heart attack, is a common presentation of ischemic heart disease or coronary artery disease. It is a medical condition that occurs when a coronary artery is severely blocked by vulnerable plaque and as a result of that there is a significant reduction or break in the blood supply.[2] It remains a leading cause of death in India and represents an enormous cost to health care system.[3] The mortality rate of myocardial infarction is approximately 30% and for every 1 in 25 patients who survive the initial hospitalization, dies in the first year after AMI. Indians are four time more prone to AMI as compared to the people of other countries due to a combination of the genetic and lifestyle factors that promote metabolic dysfunction.[4]

Cardiac marker i.e. GPBB is a fundamental enzyme in the regulation of carbohydrate metabolism by mobilization of glycogen. Three different isoenzymes exist; GPMM (present in muscles), GPLL (liver) and GPBB (brain and heart muscles).[5] Glycogen phosphorylase-BB appears to be released into the circulation 2 - 4 hours after myocardial injury.[6]

AMI is associated with profound systemic inflammatory response including elevated levels of circulating inflammatory mediators and activation of peripheral leukocytes and platelets. The excessive inflammatory response in AMI could be caused by a deregulated immune system.[7] C-reactive protein (CRP) is a phylogenetically highly conserved plasma protein that participates in the systemic response to inflammation and its plasma concentration increases during inflammatory states, a characteristic that has long been employed for clinical purposes.[8] Tissue necrosis is a potent acute phase stimulus, following AMI, there is a major CRP response, the magnitude of which reflects the extent of myocardial necrosis[9] and predicted that AMI may be associated with systemic inflammatory response. To our knowledge, no previous study has reported the relationship between CRP and GPBB. Therefore, we aimed to investigate the correlation between inflammatory marker i.e. C-reactive protein (CRP) and cardiac marker i.e. glycogen phosphorylase BB (GPBB) in AMI patients.

## MATERIAL AND METHODS

The present study was carried out in the Department of Biochemistry and Department of Cardiology, Gajra Raja Medical College and J. A. Group of Hospitals, Gwalior. The study included 250 subjects (both sex) of age group 35-75 years. Out of them, 100 were normal healthy controls and 150 were patients of AMI admitted to the Cardiology Department of J.A. Group of Hospitals. Each patient undergone clinical and laboratory evaluation, which included the detailed clinical history, clinical examination, ECG, chest X-ray, routine blood investigations and cardiac biomarkers [CK-MB and cardiac troponin T (Card test)] as a part of routine assessment and diagnosis of AMI was made after review of all the above information by a cardiologist. Patients with diabetes mellitus, chronic muscle disease, renal disease, recent surgery, implanted pacemaker, autoimmune disease, arthritis, any inflammatory disease and any other disease were excluded from the study.

About 3 ml of venous blood sample was taken from AMI patients (within 4 hours of chest pain) and controls under all aseptic precautions. Blood samples were collected in plain vials and incubated at $37^0$ C for 30 minutes. After incubation, samples were centrifuged at 3000 rpm for about 10 minutes. Serum was separated and kept at $-20^0$ C until the analysis was carried out.

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CRP was estimated by standard biochemical kit supplied by ERBA using Mindray-BS 400 fully autoanalyzer. Normal level of CRP was accepted upto 6 mg/L. Cardiac marker i.e. GPBB was measured by ELISA using QAYEE-BIO for life sciences kit. Normal reference level of GPBB was accepted as 7-18.47 ng/ml (established according to the values observed in control subjects with the help of StatsDirect 3).

This study was approved by institutional ethical committee and written consents were also obtained from the patients prior to study

## STATISTICAL ANALYSIS

Data are presented as mean ± SD values. Data analysis was performed with the Statistical Package for the Social Sciences, version 21.0 (SPSS, Chicago, Illinois, USA). The statistical differences between cases and controls were determined by student independent sample t-test. Relationship between variables was detected by Pearson's correlation coefficient.

## RESULTS

A total of 250 subjects were included in the present study. Of these, 150 were cases of AMI and rest 100 were controls**.** Table 1 and figure 1 show the mean levels of CRP and GPBB in AMI cases and controls. The mean levels of CRP and GPBB were increased in AMI cases as compared to controls and were statistically highly significant ($p<0.001$). Table 2 and figure 2 show the correlation between CRP and GPBB in AMI cases. There was positive correlation between CRP and GPBB in AMI cases.

## DISCUSSION

In the present study, cardiac marker i.e. GPBB was evaluated in AMI subjects and correlated with inflammatory marker i.e. CRP. In our study, we found significantly increased levels of both GPBB and CRP in AMI cases as compared to controls ($p<0.001$).

Glycogen phosphorylase BB is bound to glycogen in sarcoplasmic reticulum and catalyses the first step of glycogenolysis after activation, which involves the separation of glucose-1-phosphate from glycogen.[10] During myocardial ischemia, activation of glycogen phosphorylase BB results in an increase in glycogen degradation. Glycogen phosphorylase isoenzyme BB is released into bloodstream via the T-tubules system with the peak value within the first 4 hours after the onset of chest pain.[10,11]

CRP is a marker of systemic inflammation that has been associated with increased risk of incident myocardial infarction.[10] In the early phase of myocardial infarction, proinflammatory cytokines directly interfere with the myocardial contractility, the vascular endothelial function and recruitment of other inflammatory cells.[13] An association between sustained high values of CRP following AMI and its adverse outcomes was first reported in 1982.[14] Atherosclerotic process is characterized by a low-grade inflammation altering the endothelium of coronary arteries and is associated with an increase in the level of markers of inflammation.[1]

In our study, we found significant positive correlation between CRP and GPBB levels in patients of acute myocardial infarction. Inflammatory marker measurement in acute myocardial infarction, prior to the tissue necrosis, is significantly related with cardiac enzyme and prognostic significance. CRP has been studied mostly in acute myocardial infarction as a marker of proinflammatory state and plaque instability.[15] CRP is produced in response to inflammation (interleukin 6 released locally and acting on the liver) at the site of plaque rupture and then by the ischemic myocardium.[16]

| Parameters | Controls (n=100) (Mean±SD) | AMI Cases (n=150) (Mean±SD) |
|---|---|---|
| CRP (mg/L) | 4.31±0.99 | 8.14±1.83** |
| GPBB (ng/ml) | 11.79±2.82 | 61.58±33.84** |
| **Highly Significant ($p<0.001$), CRP = C-reactive protein, GPBB= glycogen phosphorylase BB. | | |

**Table-1:** Showing mean levels of CRP and GPBB in AMI cases and controls.

| Parameters | GPBB | CRP |
|---|---|---|
| CRP | 0.401** | 1 |
| GPBB | 1 | 0.401** |
| **Highly Significant at $p<0.001$, CRP= C-reactive protein, GPBB= glycogen phosphorylase BB | | |

**Table-2:** Showing correlation coefficient "r-value" between CRP and GPBB in patients of AMI.

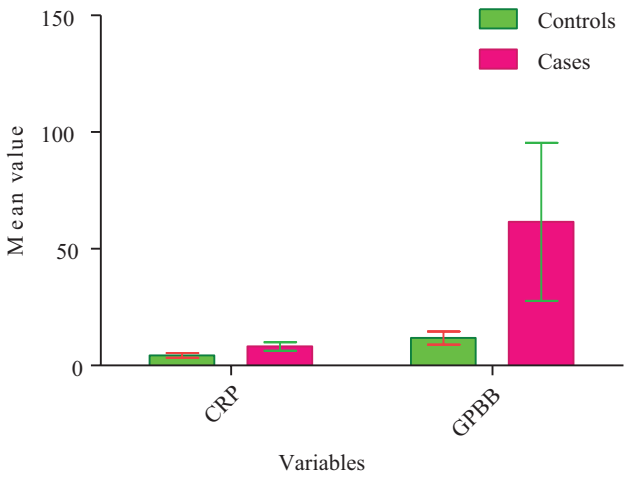


**Figure-1:** Showing status of CRP and GPBB in AMI cases and controls

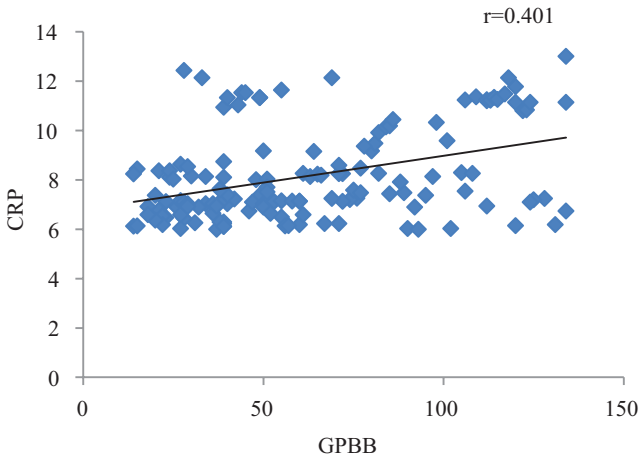


**Figure-2:** Showing the correlation between CRP and GPBB in AMI patients.

## CONCLUSION

We found increased levels of inflammatory marker CRP along

with cardiac marker GPBB and significant positive correlation between CRP and GPBB in AMI patients. It indicates that there occurs inflammation in acute myocardial infarction patients. Hence, it is concluded that CRP is a risk marker of AMI and can be used for diagnostic purpose in patients of AMI. However, further studies are needed to accept the concept.

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