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OBSERVATIONAL STUDY OF CARDIAC BIOMARKERS (TROP I, CKMB, AST, LDH) IN ACUTE MYOCARDIAL INFARCTION PATIENTS WITH RAISED CRP AND D-DIMER LEVELS.

Dr. Thambiraj Richa Lily[1], Dr. Ganesh S. Manoorkar[2], Dr. Humaira N. Khan[3], Dr. A. M. Siddiqui[4]

[1]Senior Resident Department of Biochemistry, Byramjeey Jeejeebhoy Government Medical College, Pune, Maharashtra, India.
[2]Professor & HOD Department of Biochemistry,Government Medical College Hingoli, Maharashtra, India.
[3]Professor & HOD Department of Biochemistry, Dr. Shankarrao Chavan Government Medical College, Nanded, Maharashtra, India.
[4]Associate Professor Department of Biochemistry, Government Medical College, Dharashiv, Maharashtra, India

Correspondence:
Dr. Thambiraj Richa Lily

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Abstract

Introduction: Acute Myocardial infarction (AMI) refers to death of heart muscle myocardium caused by ischemia, the lack of oxygen delivery to myocardial tissue. In addition to being aninflammatory marker, CRP has apro-inflammatory effect causing expression of adhesion molecules and inflammatory cell. D-dimer is a protein fragment (small piece) that's made when a blood clot dissolves in your body. To study the rise of Cardiac BioMarkers (S. Creatinine Kinase- Myoglobulin Binding/ Troponin I / Lactate Dehydrogenase / Aspartate Aminotransferase) in AMI patient with corelation to CRP and D-DIMER levels. ***Material & Methods:*** Present analytical, Cross-sectional study was conducted in Dr Shankar Rao Chavan Government Medical College, Nanded, Maharashtra. In present study 120 patients admitted in ICU with AMI with raised D Dimer and CRP levels were included. ***Results and Observations:*** The correlation coefficient for S.AST was 0.19, suggesting only a weak positive correlation with raised CRP levels, which may reflect the less specific nature of S. AST concerning cardiac injury. D-dimer is associated with thrombotic processes; its relationship with biomarkers of myocardial injury can vary significantly. Stronger correlations with highly cardiac-specific markers like troponins could reflect the interplay between thrombosis and myocardial necrosis in the setting of AMI. ***Conclusion:*** The correlations were identified, particularly between CRP, D-dimer, and myocardial damage biomarkers, hint at the potential for more personalized treatment strategies. For instance, AMI patients with elevated CRP and D-dimer levels might benefit from targeted anti-inflammatory or anticoagulant therapies, respectively, in addition to standard AMI management protocols.

Keywords: Acute Myocardial Infarction, ischemia, D-dimer levels

INTRODUCTION

Acute Myocardial infarction (AMI) refers to death of heart muscle myocardium caused by ischemia, the lack of oxygen delivery to myocardial tissue. It is a type of acute coronary syndrome**,** which describes a sudden or short-term change in symptoms related to blood flow to the heart [1]. Ischemia first affects the subendocardial region, and tissue begins to die within 15–30 minutes of loss of blood supply. The dead tissue is surrounded by a zone of potentially reversible is chemiathat progresses to become a full-thickness transmural infarct [2]. The initial "wave" of infarction can take place over 3–4 hours. That is when the cardiac biomarkers like Troponin I, Creatinine Kinase- myoglobin binding, Lactate Dehydrogenase, Aspartate Transaminase are released in the blood stream by the dying myocytes [3]. A rise in Trop I occurs within 2–3 hours of injury to the heart muscle, and peaks within 1–2 days [4]. CK-MB as troponins rises within 4–8 hours and returns to normal within 2–3 days [5]. AST starts increasing in the blood 3 to 4 hours after an AMI, peak levels are seen at 15 to 28 hours and returns to baseline within 5 days [6].

CRP is an acute phase protein secreted by the hepatocytes during an inflammatory stimulus. In addition to being an inflammatory marker, CRP has a pro-inflammatory effect causing expression of adhesion molecules and inflammatory cell [7]. D-dimer is a protein fragment (small piece) that's made when a blood clot dissolves in your body. Therefore, it is used for the risk estimation and management response of the patients of AMI [8].

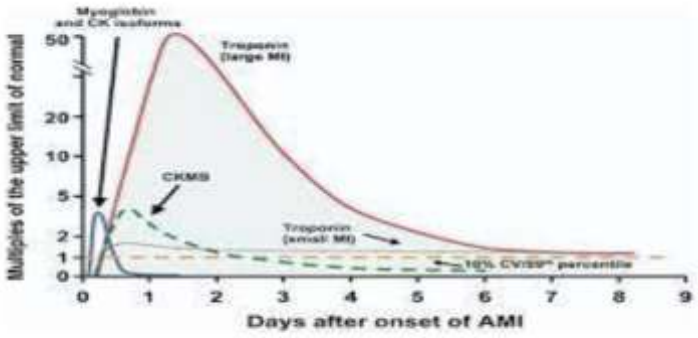

Figure 1: Days after onset of AMI

AIMS & OBJECTIVES:

To study the rise of Cardiac BioMarkers (S. Creatinine Kinase- Myoglobulin Binding/ Troponin I / Lactate Dehydrogenase / Aspartate Aminotransferase) in Acute Myocardial Infarction (AMI) patient with correlation to CRP and D-DIMER levels.

MATERIALS AND METHODS

Study design: Analytical, Crossectional study.

Study centre: Dr Shankar Rao Chavan Government Medical College, Nanded, Maharashtra

Study population: Patients admitted in the Medical in Patient Dept, Medical ICU with AMI with raised D Dimer and CRP levels in Dr Shankar Rao Chavan Government Medical College, Nanded.

Sample Size: 120 cases

Study Period: 18 – 22 months ()

Inclusion Criteria: Total population agreed to participate in the study (irrespective of gender), All patients admitted in the Medicine wards with raised D- Dimer and CRP levels.

Outcome Parameters: Serum estimation & correlation of TROP I, CkmB, LDH, AST with D-dimer, CRP in Acute MI patients Admitted in Medicine IPD ward, HDU & ICU. Done by Colorimetric kit methods & CLIA.

Method:

Non-Fasting Blood samples (4 ml) will be collected immediately after hospital admission from each patient as well as control using disposable syringes in plain bulb for estimation of Cardiac Markers (CK MB/TROP I/ LDH/ AST) included in the study. All blood samples will be allowed to clot at room temperature and then centrifuged at 4000 RPM to obtain the serum. The clear serum will be taken immediately for analysis or stored at 2 – 8 deg Celsius for 24 hrs for further use.

1. CHEM 7 used for estimation of CKMB (working principle: Classical colorimetry)
2. Fully automated MAGLUMI 2000 used for the measurement of TROP I. (Working principle: Chemilumini scence immunoassay)
3. ERBA XL 640 used for the estimation of S.LDH and AST. (Working principle: Spectrometry)
4. Reagents kits used:

   For CKMB: biolabs (r1 and r2)
   For LDH: biolabs (r1 and buffer)
   For AST: Erba (r1)
   For TROP I : Maglumi Trop I J

Estimation of Creatine Kinase-MB: 5-25 IU/L

A specific antibody inhibits CK-M subunits but it does not affect to the CK-B subunits. CK-B catalytic concentration, which corresponds to half of CK-MB concentration, is determined from the rate of NADPH formation, measured at 340 nm, by means of the hexokinase (HK) and glucose-6- phosphate dehydrogenase (G6P-DH) coupled reaction

Estimation Aspartate Amino transferase: 8-33 IU/L

Aspartate aminotransferase (AST/GOT) catalyzes the transfer of the amino group from aspartate to 2- oxoglutrate, forming oxalacetate and glutamate. The catalytic concentration is determined from the rate of decrease of NADH measured at 340 nm, by means of the malatedehydrogenase (MDH) coupled reaction.

Estimation of LDH: 105 – 333 IU/L
Activity can be determined by measurement of the rate at which DPNH is oxidized (decrease in optical density at 340 ταμ) or DPN reduced (increase in optical density at 340 mμ) , depending on whether the reaction is studied from the pyruvate or lactate side.

Estimation of TROP I CRP & D-Dimer: CLIA, no reagents used.
Trop I: 0- 0.04 ng/L
CRP: 0.3 – 1.0 mg/dl
D-dimer :< 0.50 mg/L

RESULTS

Table 1: Raised CRP correlation with other Biomarkers of Acute Myocardial Infarction Patients.

	Mean	Standard Error	Standard Deviation	Sample Variance	Range	Confidence Level (95.0%)	Correlation coefficient
S.CKMB	21.64	0.68	7.37	54.32	30	1.34	0.51
S.TROP I	20.30	0.92	9.95	98.98	49.70	1.81	0.53
S.LDH	334.99	11.62	126.77	16070.48	598	23.01	0.40
S.AST	48.18	2.06	22.51	506.64	203	4.09	0.19

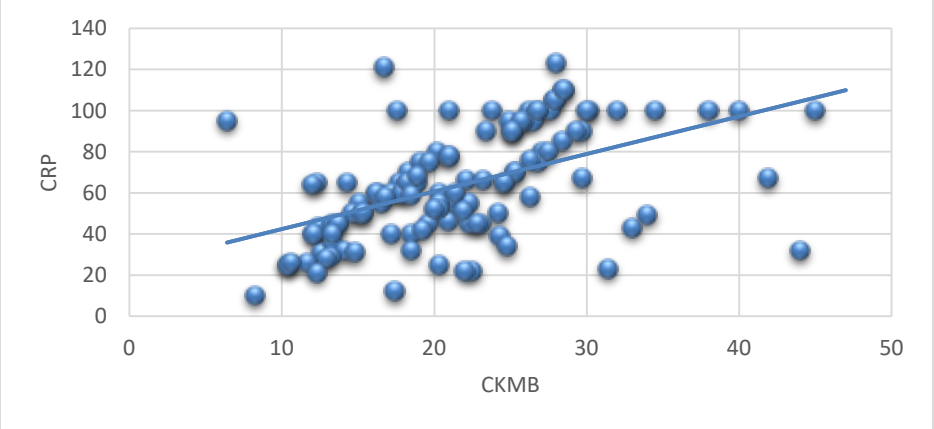

Fig 2: Scatter diagram showing correlation of CRP with CK-MB

The analysis of the correlation between raised C-reactive protein (CRP) levels and other cardiac biomarkers provides insight into the interrelation among these indicators in the context of myocardial injury.

For S.CKMB, the mean value across patients with raised CRP was 21.64, with a standard error of 0.68 and a standard deviation of 7.37, indicating a moderate variation among patient levels. The sample variance was 54.32, and the range of S.CKMB levels was 30. The 95% confidence level of the mean was 1.34, which reflects the precision of the mean estimate. A correlation coefficient of 0.51 suggests a moderate positive correlation between raised CRP levels and S.CKMB, indicating that as CRP levels increase, S.CKMB levels tend to increase as well.

The mean S.TROP I level was 20.30, with a standard error of 0.92 and a higher standard deviation of 9.95, pointing to a slightly greater variability in levels compared to S.CKMB. The sample variance was substantial at 98.98, and the range was 49.70. The confidence level of 1.81 denotes a reasonable estimate of the mean with a 95% certainty. The correlation coefficient for S.TROP I was 0.53, suggesting a moderate positive correlation with raised CRP, possibly indicating a common pathophysiological link between troponin I release and inflammatory processes.

S.LDH had a mean of 334.99 with a standard error of 11.62 and a standard deviation of 126.77, which is quite high and indicative of significant variability among the patients' levels. The sample variance was 16070.48, and the range was 598, signifying broad variability in S.LDH levels. The confidence level was 23.01, reflecting the greater spread of values. The

correlation coefficient of 0.40 indicates a weaker, yet positive, correlation with raised CRP levels compared to S.CKMB and S.TROP I.

Lastly, S.AST presented with a mean level of 48.18, a standard error of 2.06, and a standard deviation of 22.51, indicating variability but less so than S.LDH. The sample variance was 506.64, and the range was 203. The 95% confidence level of 4.09 is larger than that for S.CKMB and S.TROP I, denoting less precision due to greater data spread. The correlation coefficient for S.AST was 0.19, suggesting only a weak positive correlation with raised CRP levels, which may reflect the less specific nature of S.AST concerning cardiac injury.

Table 2: Raised D-DIMER correlation with other Biomarkers of Acute Myocardial Infarction Patients.

	Mean	Standard Error	Standard Deviation	Sample Variance	Range	Confidence Level (95.0%)	Correlation Coefficient
S.CKMB	21.64	0.68	7.37	54.32	39	1.34	0.45
S.TROP I	20.30	0.92	9.95	98.98	50	1.81	0.57
S.LDH	334.99	11.62	126.77	16070.48	598	23.01	0.01
S.AST	48.18	2.06	22.51	506.64	203	4.09	0.05

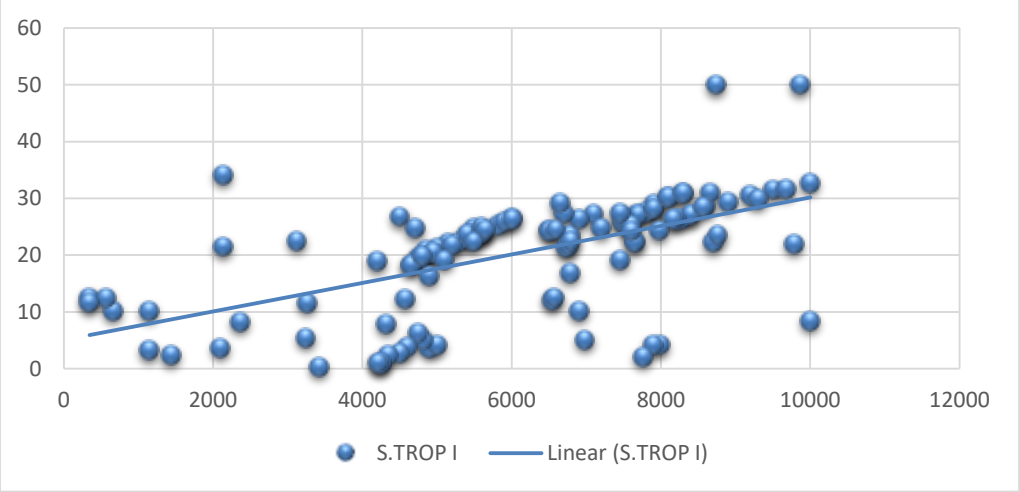

Fig 3 : Scatter diagram Showing correlation of D-dimer with TROP I

For S.CKMB, the mean value when D-dimer is elevated is 21.64, with a standard error of 0.68 and a standard deviation of 7.37, suggesting a moderate dispersion around the mean. The sample variance is 54.32, and the range is 39. The confidence level at 95% is 1.34, which offers a reasonable degree of certainty about the mean value. The correlation coefficient of 0.45 indicates a moderate positive correlation with raised D-dimer levels. This suggests that as D-dimer levels increase, which is indicative of fibrinolysis and possibly thrombotic activity, there may be a concomitant rise in S.CKMB, reflecting myocardial damage.

S.TROP I has a mean of 20.30, with a standard error of 0.92 and a standard deviation of 9.95. This greater variability is also reflected in the sample variance of 98.98 and a range of 50. The 95% confidence level is 1.81, denoting a credible mean estimate. A correlation coefficient of 0.57 indicates a stronger positive correlation with raised D-dimer levels compared to S.CKMB. This could be due to the high cardiac specificity of troponins, which may be released into the circulation in the setting of myocardial infarction, often associated with increased thrombotic activity.

S.LDH has a mean level of 334.99 with a standard error of 11.62 and a large standard deviation of 126.77, pointing to considerable interpatient variability. The sample variance is quite large at 16070.48, and the range is 598. The confidence level at 95% is 23.01, which reflects the broad spread of values across patients. However, the correlation coefficient is only 0.01, indicating virtually no correlation with raised D-dimer levels. This might suggest that LDH, being less specific to cardiac tissue, does not show a consistent pattern with thrombotic markers such as D-dimer.

Finally, S.AST shows a mean of 48.18, a standard error of 2.06, and a standard deviation of 22.51. The sample variance is 506.64, and the range is 203, with a 95% confidence level of 4.09. The correlation coefficient of 0.05 indicates a

negligible positive correlation with raised D-dimer levels, supporting the idea that AST, which can be released from various tissues, does not consistently reflect changes associated with thrombosis and myocardial injury indicated by D-dimer.

These findings may indicate that while D-dimer is associated with thrombotic processes, its relationship with biomarkers of myocardial injury can vary significantly.Stronger correlations with highly cardiac-specific markers like troponins could reflect the interplay between thrombosis and myocardial necrosis in the setting of AMI, while weaker or absent correlations with less specific markers like LDH and AST may reflect their wider tissue distribution and involvement in various pathological processes.

DISCUSSION

Our investigation into acute myocardial infarction (AMI) patients with elevated CRP levels revealed interesting correlations with other cardiac biomarkers. Specifically, we observed a moderate positive correlation between CRP and S.CKMB (r = 0.51) and S.TROP I (r = 0.53), suggesting that inflammatory processes, as indicated by CRP, are associated with the extent of myocardial damage reflected by these biomarkers. However, the correlation between CRP and S.LDH (r = 0.40) and S.AST (r = 0.19) was weaker, indicating that these latter biomarkers may not be as closely linked to the inflammatory processes in the context of AMI.

Zhang et al found that higher D-dimer levels, associated with inflammatory and coagulation processes, showed a positive correlation with troponin and NT-proBNP levels. This supports our findings of a relationship between inflammation (as measured by CRP) and myocardial damage markers. The absence of a direct comparison with CRP in their study leaves an opportunity to explore this relationship further [9].

Kim et al review emphasized the role of CRP as an inflammatory marker that may correlate with the degree of myocardial damage. This aligns with our findings, suggesting that the inflammation captured by CRP levels does indeed relate to the severity of myocardial injury as reflected by specific biomarkers [10].

In the context of acute ischemic stroke, Yao et al.[11] did not find CRP to be an independent predictor of outcomes after multivariate adjustment, which contrasts with our findings where CRP showed a moderate correlation with key cardiac biomarkers in AMI. This discrepancy might be attributed to differences in the pathophysiological mechanisms between ischemic stroke and AMI .

The positive correlation between elevated CRP levels and cardiac biomarkers such as S.CKMB and S.TROP I in our study underscores the intertwined nature of inflammation and myocardial injury in AMI. These findings highlight the potential utility of CRP, not just as a marker of inflammation but also as an adjunct in assessing the severity of myocardial damage and possibly guiding therapeutic decisions.

The weaker correlation between CRP and enzymes like S.LDH and S.AST may reflect the broader tissue expression and less cardiac specificity of these enzymes, diluting their association with the inflammatory processes specific to myocardial injury.

Clinically, these correlations suggest that in AMI patients with elevated CRP, a heightened alertness to the potential for severe myocardial damage may be warranted, particularly when S.CKMB and S.TROP I levels are also elevated. This could influence both the acute management and long-term treatment strategies for these patients, potentially guiding more aggressive anti-inflammatory and cardioprotective interventions.

In present cohort of acute myocardial infarction (AMI) patients, we observed notable correlations between raised D-dimer levels and several cardiac biomarkers. Specifically, there was a moderate positive correlation between D-dimer and S.CKMB (r =0.45) and a stronger correlation with S.TROP I (r =0.57), indicating that the coagulation process reflected by D-dimer is closely associated with the extent of myocardial injury. Interestingly, the correlation between D-dimer and S.LDH was negligible (r =0.01), and similarly minimal with S.AST (r =0.05), suggesting that these biomarkers might not be directly linked to the coagulation pathway activated in AMI.

Zhang et al highlighted the association of higher D-dimer levels with adverse outcomes in AMI, underscoring the prognostic significance of coagulation markers. While they did not specifically report on the correlation between D-dimer and other cardiac biomarkers, their findings on D-dimer's prognostic value are consistent with our observations of its correlation with markers of myocardial damage [10].

While focusing on ischemic stroke, Yao et al. [11] found that elevated D-dimer levels predicted worse outcomes, similar to our findings in AMI. This parallel suggests that D-dimer's role as a reflection of coagulation and thrombotic processes may have implications across different cardiovascular diseases.

The correlations we observed between raised D-dimer levels and specific cardiac biomarkers in AMI patients underscore the multifaceted nature of AMI pathology,

involving both myocardial injury and coagulation processes. The strong correlation with S.TROP I, in particular, highlights the potential utility of D-dimer as a marker for assessing the severity of myocardial damage in the context of AMI.

The minimal correlations with S.LDH and S.AST might indicate that these enzymes, which can be released from various tissues and are less specific to cardiac injury, do not directly participate in or reflect the coagulation and thrombotic processes marked by elevated D-dimer levels.

Clinically, these findings suggest that in AMI patients with elevated D-dimer, a careful assessment of myocardial injury severity using S.CKMB and S.TROP I is warranted. Elevated levels of these biomarkers in conjunction with high D-dimer could indicate a more severe myocardial injury and a higher risk of thrombotic complications, guiding more aggressive therapeutic interventions.

CONCLUSION

The moderate to strong correlations between elevated CRP and D-dimer levels with markers of myocardial injury (S.CKMB and S.TROP I) illuminate the intertwined roles of inflammation and coagulation in AMI pathology. These relationships underscore the multifactorial nature of AMI, where inflammatory and coagulative pathways contribute to the overall clinical picture and may influence patient outcomes.

Our findings advocate for a multi- dimensional approach in the assessment and management of AMI, integrating the consideration of age, the acknowledgment of similar biomarker implications across sexes, and the insightful use of biomarker correlations to guide clinical decisions.

Furthermore, our study paves the way for future research aimed at unraveling the nuanced interplay of biomarkers in AMI, potentially leading to the development of more precise diagnostic tools and tailored therapeutic interventions.

The correlations we identified, particularly between CRP, D-dimer, and myocardial damage biomarkers, hint at the potential for more personalized treatment strategies. For instance, AMI patients with elevated CRP and D-dimer levels might benefit from targeted anti-inflammatory or anticoagulant therapies, respectively, in addition to standard AMI management protocols.

Efforts should be made to enhance healthcare professionals' understanding of the implications of various cardiac biomarkers in AMI, ensuring that the latest research findings are translated into clinical practice effectively. Additionally, patient education regarding the significance of biomarkers and the importance of timely medical intervention in the event of cardiac symptoms can improve outcomes.

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