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Original Research Paper

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SERUM ADENOSINE DEAMINASE ACTIVITY IN MYOCARDIAL INFARCTION:A NEW DIMENSION IN MODERN DAY DIAGNOSTICS.

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ABSTRACT

Objective:-Myocardial infarction is characterised by widespread irreversible cellular necrosis & death due to ischaemic injury.Atherosclerotic plaques & their role in bringing about ischaemic injury forms an integral component of the pathogenesis of myocardial infarction.An immune link in the formation of atherosclerotic plaques has been reported by few researchers.The enzyme adenosine deaminase is a key player in the immune system.This study intends to estimate adenosine deaminase(ADA) activity in serum,Troponin I & other cardiac enzymes in patients with myocardial infarction.

Methods:-The study comprised of 40 patients with myocardial infarction alongwith 20 age & sex matched healthy controls.Serum ADA,Troponin I & cardiac enzymes [CPK-MB,LDH,AST] were estimated.

Results:- An appreciable rise in serum ADA activity was observed alongwith elevated titres of cardiac enzymes & Troponin I in patients with myocardial infarction.

Conclusion:- ADA is a key player in immune system & is a marker of T-lymphocyte activation.Raised titres of ADA in myocardial infarction patients as was observed in this study provide evidence in favour of T-lymphocyte activation & proliferation in myocardial infarction thereby opening a new dimension in understanding of the pathogenesis of myocardial infarction.This finding also suggests possible use of serum ADA activity as a marker in elucidating the pathogenesis of myocardial infarction.

KEYWORDS :

INTRODUCTION

Myocardial infarction (MI) is characterised by death of myocytes due to an imbalance in myocardial oxygen supply & oxygen demand[1].Coronary atherosclerosis alongwith superimposed coronary thrombosis is almost an invariable feature associated with all cases of MI[2].It is a major cause of mortality in elderly poulation[2].Thrombus & atheroscletic plaque obstruct the flow of the coronary artery resulting in a diminished myocardial oxygen supply & increased oxygen demand[2].The atherosclerotic lesion is comprised of macrophages & activated T lymphocytes which secrete cytokines[2]. The inflammatory response leads to plaque rupture & thrombosis Mi[2].

MI is diagnosed on the basis of specific ECG changes & characteristic pattern in change of titre of certain serum enzymes such as creatinine kinase[CK], creatinine kinase MB isoenzyme [CK-MB], lactate dehydrogenase [LDH] & certain cardiac specific proteins such as troponin[3].

Clinicians primarily rely on ECG changes while making a diagnosis of MI as clinical features vary widely from person to person[3].This variation in clinical features amongst patients puts the clinician in a dilemma & hence the importance of biochemical markers in diagnosis.Apart from the established markers,several new upcoming biomarkers are being monitored to asses myocardial injury.Availability of new biomarkers with markedly enhanced sensitivity for myocardial damage enables clinicians to diagnose MI in an additional one third of patients who would not have fulfilled criteria for MI in the past[4].This study was therefore directed towards studying the relationship between established markers such as CK-MB,AST,LDH ,Troponin-I with a lesser established marker such as serum adenosine deaminase activity in myocardial infarction cases.

MATERIAL & METHODS

The study was carried out from July 2020 to October 2020 at Deben Mahata Govt Medical College & Hospital, Purulia, West Bengal..The study was approved by the institutional ethical committee at Deben Mahata Govt Medical College & Hospital, Purulia, West Bengal. Study subjects were recruited from patients diagnosed with acute myocardial infarction admitted in intensive care unit. The study group comprised of 40 patients (age 30-50 years) diagnosed with myocardial infarction.The control group comprised of 20 age & sex matched healthy subjects with no history of any known disease.Details pertaining to age,sex,habits,health etc were recorded in a special proforma.

With full aseptic precautions 5 ml of venous blood was drawn from patients with myocardial infarction as well as the healthy control subjects & allowed to clot.The clotted samples were then centrifuged at 2000 rpm for 10 minutes so as to separate the serum. Autoanalyser was used for estimating creatinine kinase (CK)[5], aspartate transaminase(AST)[6] & Lactate dehydrogenase(LDH)[7] using IFCC kinetic method. Instant view Troponin I cards based on the principle of immunoassay[8] were used for estimating Troponin I. A kit manufactured by Tulip diagnostics based on colourimetric method as explained by Giusti & Galanti[9] was used for estimating ADA. SPSS version 11 was used for statistical analysis. Student T test & Pearson correlation coefficient were employed for assessing the data. A "p" value <0.01 was considered significant.

RESULTS

The study comprised of two groups. Group I consisted of 20 age & sex matched healthy individuals (controls) & group II comprised of 40 patients diagnosed with acute myocardial infarction. Mean of CK-MB for group I was 18.43 ± 2.92 ,whereas for group II it was 107.8 ± 19.9 .CK-MB was significantly elevated in group II as compared to group I with a pearson coefficient("p" value) <0.001. Mean AST levels in group I was 21.2 ± 4.75 & in group II was 141.3 ± 21.01. The elevated AST levels in group II as compared to group I was significant with a "p" value<0.001.Group I demonstrated a mean LDH value of 328.53 ± 48.6 whereas mean LDH value of group II was 798.53 ± 60.2 . The comparison of LDH values in both groups revealed a significant elevation in group II with "p" value <0.01. A significant increase in CK-MB, LDH & AST was observed in group II as compared to group I. Mean$\pm$SD of ADA in group I & II was 17.49 ± 3.83 & 52.06 ± 7.77 respectively. A significant increase in ADA values was therefore noticed in

group II as compared to the controls in group I ($p<0.01$). Troponin I was negative in group I amongst the healthy controls as compared to MI patients in group II.

Table 1: Comparative data of various parameters between the two groups.

S.No	Parameters	Group I	Group II	p Value
1	CK-MB	18.43±2.92	107.8±19.9	<0.001
2	AST	21.2±4.75	141.3±21.01	<0.001
3	LDH	328.53±48.6	798.53±60.2	<0.01
4	ADA	17.49±3.83	52.06±7.77	<0.01

DISCUSSION

Myocardial infarction is an acute medical emergency & therefore an early diagnosis is a necessity to reduce mortality. Diagnosis of myocardial infarction as well as monitoring the course and size of infarction is largely dependent on laboratory parameters. Troponins are considered as most promising amongst all the markers available presently[3].CK-MB is detectable within 4 hours of onset of chest pain[4]. AST levels tend to rise 6-8 hours after onset of chest pain,reach a peak within 24 hours & subsequently return to normal after 36 hours[4].LDH values tend to rise 8 hours after onset of chest pain while attaining a peak at 24 hours.[4] Troponins are detectable 2-8 hours post MI & attains peak at 18-24 hours. In our study we found CK-MB, LDH, AST & Troponin-I values grossly elevated in patients with MI as compared to the controls.

ADA is an enzyme involved in purine metabolism.It is essential for maturation & proliferation of T lymphocytes.It is a mediator of cellular immunity in the body[8].

Coronary blood flow during stress & hypoxia is increased by adenosine to balance the supply & demand of oxygen. The advantage thus provided by adenosine to myocardial tissue will therefore be lost if its rapidly metabolised by ADA.Adenosine is metabolised to inosine which produces superoxide radicals & exaggerate ischaemic injury[6].

The results of our study showed a significant elevation in ADA levels in patients with MI as compared to controls. This is in agreement with A.Jyoti etal[2].

CONCLUSION

Our study clearly revealed significant elevation of all the cardiac markers alongwith elevation of ADA levels in patients with MI.It might therefore be suggested that ADA can serve as an inflammatory marker which has been studied poorly in respect of MI.

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