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Role of HSCRP in Detecting Myocardial Infarction.

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Abstract:
Introduction: *Inflammatory markers, such as high-sensitivity C-reactive protein (hsCRP), are useful prognostic factors for cardiovascular events in several situations.Acute myocardial infarction (MI) triggers an acute phase response which is induced by proinflammatory cytokines, which stimulate the liver to synthesize C-reactive protein (CRP).The present study is to elucidate the significant link between plasma hsCRP levels and AMI.*
Objective: *Role ofAcute plasma hsCRP levels in predicting the Acute Myocardial infarction.*
Methodology: *The study was conducted in Maharajah's Institute of Medical Sciences (MIMS) . Fifty seven patients were initially included in the study later fifteen patients were excluded from the study . Finally ,forty two patients (n=42) with AMI for the first time enrolled for the study representing the guide lines of MI by European society of cardiology (ESC) and American college of cardiology (ACC) .The data obtained from the above patients is compared with control group (n=33) after matching .*
Results: *The mean age of male participants in infarct group is 58 years and in females it is 56 years.The values of different bio chemical variables like Urea,FBS,HDL,LDL,VLDL in the infarct group are significantly higher when compared to the normal group .The values of hsCRP showed a significant difference between the time frames of 6 ,12,24,48hours and the difference is increased with the normal as time progresses . Diagnostic criteria of hsCRPis established by ROC curves and it is increased more after 6 hours of infarction.*
Key Words: *Acute Myocardial Infarction, Cardiac markers, Diagnostic criteria, hsCRP.*

I. Introduction

Acute myocardial infarction (AMI) is a major public health problem among the non communicable diseases in the developing countries like India, inspite of progressive research in diagnosis and management over the last threedecades. Inflammation plays an important role in the AMI .An acute phase response C-reactive protein (CRP), is a marker of inflammatory activity and is associated with atherosclerosis and increased incidence of coronary events [1]. Acute myocardial infarction (MI) triggers an acute phase response which is induced by proinflammatory cytokines, which are released from the necrotic myocardium and they stimulate the liver to synthesize C-reactive protein (CRP) [2] .The purpose of this study was to assess the utility of hsCRP levels after acute myocardial infarction in risk stratification.

II. Methodology

A Single center- prospective observational study is conducted after getting approval from Institutional Ethical Committee of Maharajah's Institute of Medical Sciences (MIMS) .

Patients who were admitted in the OP Department between an age group ranging from 31 to 70 years complaining of chest pain and other ischemic symptoms for the first time without prior medication or treatment were included in the study. Fifty seven patients were initially included in the study later nine patients were excluded from the study whose clinical data is not completely obtained and six patients were died and finally 42 patients (n=42) with AMI for the first time with an age group of 31-70 were enrolled for the study representing the guide lines of MI by European society of cardiology (ESC) and American college of cardiology (ACC). To establish the diagnosis, blood samples were drawn from the individuals, later based on the advice of the treating cardiologist and physician they were subjected to TMT-ECG. On confirmation of ischemia on TMT-ECG based diagnostic criteria, a second blood sample was collected from these patients within one hour after TMT. The blood samples collected from the subjects before TMT, who do not show any abnormality pertaining to IHD were discarded and excluded from the study. Finallytheir data compared with control group (n=33) after matching.

Diagnostic Criteria of the Myocardial Infarction

After recording typical ECG changes showing of pathological Q-waves (or) S-T segment and T-wave changes and troponin I >0.1ng/ml , the subsequent blood samples were collected in plain vacutainers at different time intervals viz., 6, 12, 24, and 48hours . The samples were centrifuged and stored at -70 ^{0}C and assayed in batches.

Exclusion Criteria: The patients with renal failure, renal transplantation, aortic dissection ,cardiac contusion, chemotherapy, myocarditis, diabetes mellitus, sepsis, severe neurological disorders, pericarditis, old AMI cases, non atherosclerotic MI, left ventricular hypertrophy, muscular dystrophy were excluded from our study as these conditions lead to changes in some of the cardiac biomarkers.

Assay for Cardiac Markers

Blood was collected, centrifuged and the serum was recovered for all the markers which is stored at -70^{o}C and assayed batch wise. Standard controls were assayed regularly .hsCRP was assayed by ELISA . Serum hsCRP was assayed by using Acculite CLIA microwells supplied by MonobindInc (Lake Forest, CA, USA) with an upper limit of ≤1.3ng/ml.

Statistical Analysis: Statistical analysis was performed using SPSS version 17 .Arithmetic mean ,SD of variables were estimated and relevant statistical tests were performed like Unpaired t test, repeated measure ANOVAwere used. Differences were considered statistically significant at $p<0.05$.Ability of the diagnosis was based on the area under the curve (AUC) of receiver operator characteristic curve (ROC).

III. Results

Table 1 : Comparisons Of Means And Sd Of Age Between Infarct And Control Group

	Infarctgroup		**Controlgroup**	
	Mean	**SD**	**Mean**	**SD**
Male	**58.10**	**8.573**	**54.05**	**11.56**
Female	**56.36**	**7.103**	**45**	**11.68**

In the present study mean age in males in infarct group is 58 years and is ranging from 48 years to 70 years. In females it is 56 years ranging from 44 to 71.

Table 2: Laboratory Values Incontrol Group.

	n	Minimum	Maximum	Mean	Std. Deviation
UREA	33	18	44	34.58	6.750
FBS	33	80	415	107.97	37.140
T.CHOL	33	110	410	184.85	41.274
HDL	33	28	58	36.18	5.169
LDL	33	54	325	121.21	40.953
VLDL	33	15	47	29.55	8.345

The mean values of different variables in the control group are within the normal limits of actual values .

Table 3 : Laboratory Investigation Values In Infarct Group.

	N	Minimum	Maximum	Mean	Std. Deviation
UREA	42	22	61	41.14	7.618
RBS	42	98	225	148.83	34.196
T.CHOL	42	143	270	221.07	33.926
HDL	42	37	98	43.40	9.548
LDL	42	48	202	141.57	31.457
VLDL	42	24	46	35.57	8.019

The values of different bio chemical variables in the infarct group are significantly higher when compared to the normal group and this difference is found to be statistically significant.

Table 4: Hscrp Levels In Infarct Group At Different Time Frames.

	N	Mean	Std. Deviation
Admission	42	4.2381	1.76966
6thhr	42	6.8571	2.57124
12hr	42	9.5190	2.76355
24hr	42	12.1595	3.27005
48hr	42	17.2714	5.56142

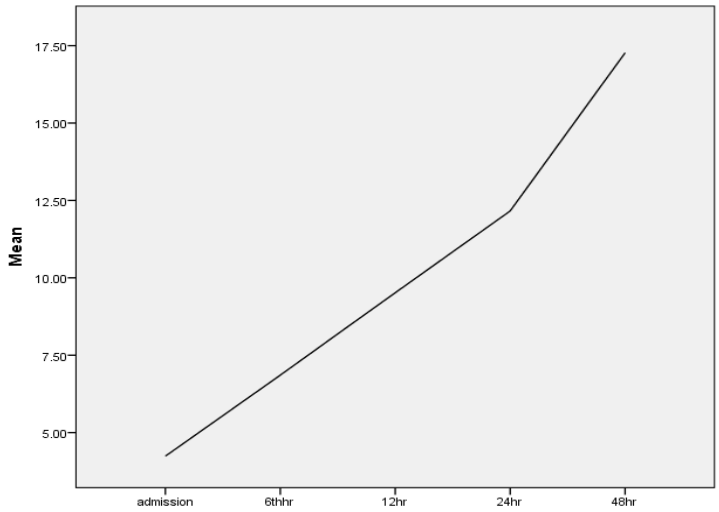

Figure1: Showing the hsCRP Values At Different Time Frames.

Above figure clearly states that with time after infarction the hsCRP values getting raised .
The mean values showed a significant difference between the time frames with Wilks' Lambda showing a F value of 109.06 which is statistically highly significant.

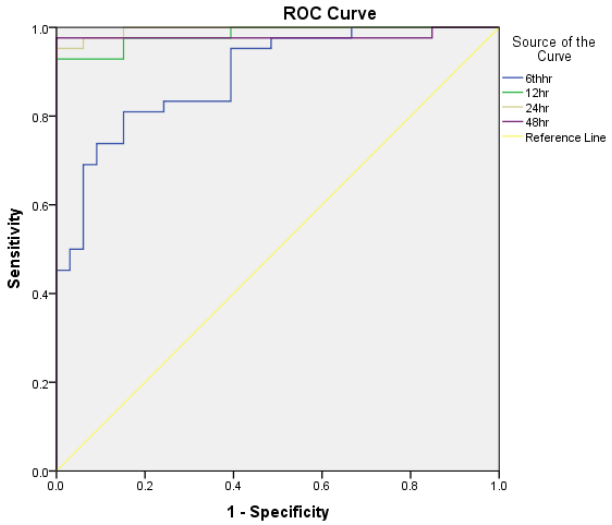

Figure 2: Rocs at Different Time Frames after Ami.

Table 5: Area Under The Curve

Test Result Variable(s)	Area	Std. Error[a]	Asymptotic Sig.[b]	Asymptotic 95% Confidence Interval	
				Lower Bound	Upper Bound
6thhr	.892	.036	.000	.822	.962
12hr	.983	.012	.000	.961	1.000
24hr	.995	.005	.000	.986	1.000
48hr	.980	.020	.000	.940	1.000

a. Under the nonparametric assumption
b. Null hypothesis: true area = 0.5

The AUC at and after six hours of infarct is more than 0.892 which clearly states that hsCRP plays an important role as diagnostic criteria from 6 hours of Acute myocardial infarction .

IV. Conclusion

There is a significant role of hsCRP in predicting Acute Myocardial Infraction and the prediction value is more predominant after 6 hours of infarction . Thus hsCRP can play a role in early identification of MI and can also be used as a confirmatory indicator in post MI patients.

V. Discussion

Many studies suggest that there is a role for baseline hsCRP in predicting AMI .HisatomiArima[3] clearly demonstrated that hsCRP levels were associated with future coronary events in a general population of Japanese. They concluded that serum hsCRP levels are potent predictors of prognosis in patients with AMI and elevated levels of hsCRP at the time of admission indicates a poor prognosis in patients with AMI. Studies conducted by AmrutDambal et al [4] concluded that significant high values of hsCRP in diabetics may signal a considerable damage to the vascular endothelium, whichcould play a role in the causation of CHD.

Studies conducted by SubodhVermaet al [5]says that CRP appears to play the role of a participant and not just that of anindicator of atherosclerosis, uncovering the molecular mechanismsbehind this interaction is more important.

M BASKURT et al [6]studied that serum levels of hs-CRP could not predict the occurrence of reversible myocardial ischaemia during exercise and they stated that large-scale clinical studies are needed to clarify the status of hsCRP, SAA and NT-proBNP with exercise.

Deepak Y et al [7] stated that the normal or basal values of hsCRP are likely higher in the Indian population and there is need to derive risk cut-off values for CVD in the Indian population.

Yip HKetal[8] in their prospective cohort study observed that patients with STEMI of onset < 6 h and were undergoing primary PCI showed that Prospective evaluation of the hsCRP in them helps in accurate risk stratification of individuals at risk of 30-day after primary PCI.

MetteRauheMouridsen et al [9]concluded that hsCRP was not independently associated with CAD. Exercise has the potential to cause unwanted variations in hsCRP and that exercise prior to hsCRP measurements in subjects included in epidemiological studies should be avoided.

Zebrack JS et al [10] showed that predischarge CRP levels are higher after AMI than after Unstable Angina Pectoris or Stable Angina Pectoris . CRP is strongly predictive of long-term AMI for patients presenting with SAP or UAP, it is not predictive shortly after AMI, suggesting that measurements should be delayed until the acute phase reaction is over and levels have returned to baseline.

Badiger RH etal [11]showed that the raised hs-CRP level in the majority of patients with AMI suggests involvement of inflammation in the etiopathogenesis of MI and has prognostic utility in AMI. Higher the serum hs-CRP levels on admission in patients of AMI the more the patient is prone for developing complications during their hospital stay.

IwonaSwiatkiewiczetal[12] in the their study on Usefulness of C-reactive protein as a marker of early post-infarct left ventricular systolic dysfunction concluded that measurement of CRP plasma concentration levels may be useful as a marker of early LVSD in patients after a first STEMI.

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