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Utility Of High-Sensitivity C - Reactive Protein (Hscrp) & Lipid Profile In Myocardial Infarction

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ABSTRACT:

Background: Myocardial infarction also known as acute myocardial infarction (AMI) term, which is commonly used for an event of heart attack. The biomarker is possible of using different hsCRP & lipids biochemical marker for predicting risk of myocardial infarction (MI).

Objectives: Utility of high sensitivity C-reactive protein and Lipid Profile levels in Myocardial Infarction.

Patients and Methods: Evaluation of biochemical marker and examined by the cardiologist of confirming myocardial infarction patients and healthy control of all age groups from the period of January 2018 to December 2019. This study divided into two groups: group A: 55 Myocardial Infarction patients; group B: 55 healthy control subject. Comparison between lipid profiles & High sensitivity C Reactive Protein, including serum total cholesterol, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, and triglycerides, in 55 myocardial infarction patients. Patients included 29 (52.8%) STEMI patients, 4 (7.2%) NSTEMI patients and 22 (40%) patients with chest pain. Control group age and gender-matched normal subjects are included.

Results: The levels of lipid profile and hsCRP in case and control subjects were significant (p value= 0.0001**) high in the myocardial infarction patients. Myocardial infarction patients had significant higher levels of hsCRP, TC, LDL, VLDL, TG, LDL/HDL, TC/HDL and decreased level of HDL as compared to the control subjects.

Conclusions: Elevated hsCRP has a strong significant association with lipid profile in myocardial infarction. These data suggest that inflammatory processes play a self-regulating role in the pathogenesis of myocardial infarction.

Key Word: Lipid Profile, hsCRP, Chest Pain, Myocardial Infarction, Hypertension, Diabetes, Dyslipidemia.

INTRODUCTION:

Myocardial infarction (MI) is major cause of necrosis resulting from acute obstruction of a coronary artery. Myocardial infarction is one of the important reasons of mortality and unhealthiness in the world. Various risk factors for Myocardial infarction have been reported, including age, gender, race and family history and an another risk factors, like serum

cholesterol, smoking, diabetes and high blood pressure can be changed. [1] According to the Global Burden of Disease Report released on September 15, 2017, heart disease is the leading cause of death in India, killing 1.7 million Indians in 2016. [2]

About 50%-70% of ischemic heart disease is represented by myocardial infarction (MI) and MI continues to be the leading morbidity for hospital admissions. [3-4] many factors have been identified related to MI that includes smoking, diabetes, systemic arterial hypertension and dyslipidemia. [4-8]
Inflammatory Markers, high sensitivity C-receptive protein (hsCRP) can be used to identify patients with the most complicated coronary lesions and hsCRP is an independent predictor of future cardiovascular event, in addition to predicting the risk of incident hypertension and diabetes. [9] The pathophysiological of inflammation features of atherosclerosis is well established [10-11] as well as the prognostic usefulness of biomarker surrogates, such as hsCRP (high-sensitivity C-reactive protein), for predicting the risk of vascular events in primary cardiovascular prevention. [12] Lp (a) concentrations were closely correlated with hs-CRP concentrations in myocardial infarction patients, suggesting that Lp (a) may also act as an acute phase reactant. [13]

The donation of inflammation to the pathophysiological features of atherosclerosis is well recognized, [10-11] as well as the prognostic helpfulness of biomarker surrogates, such as hsCRP (high-sensitivity C-reactive protein), for predicting the risk of vascular events in primary cardiovascular prevention. [12]
Lp (a) concentrations were closely correlated with hsCRP concentrations in myocardial infarction patients, suggesting that Lp (a) may also act as an acute phase reactant. [13]
Suspected myocardial infarction (MI) is a common reason for emergency hospital attendance and admission. The development and acceptance of biomarker measurement as part of the diagnostic strategies for patients presenting with chest pain and suspected Myocardial Infarction (MI). [14] Inflammation is important in MI prognosis: elevations of inflammatory markers such as high-sensitivity C-reactive protein after MI are associated with an increased risk of recurrent cardiovascular events. [15-16] the researchers coincide in that they during admit hsCRP concentration reflects the baseline inflammatory status of the patient; thus, patients with MI and high hsCRP levels at admission usually experience more cardiovascular complications during follow-up. [17]
Elevated Interleukin-6 along with C reactive protein might be the sign of early risk of atherogenic risk progression [18] and CRP as a predictive biomarker of CVD risks in the patients suffering from Type 2 diabetes [19]. Increased lipoprotein ratios and Hs-CRP may promote as a cardiovascular risk can be developed [20]. Increasing concentration of C-reactive protein along with LDL-C may progress to cardiac abnormality [21]. It is significant to recognize that the immediate analyze of lipids, particularly Low density lipoprotein, and Interleukin- 6 develop the prediction of risk of future MI-coronary death compared with that correlated with lipids or Interleukin- 6 alone [22].Other past studied showed elevated level of glycated hemoglobin as significant risk factors in the progression of DM, atherosclerosis, myocardial infarction, renal disorder, hypertriglyceridemia, and obesity [23]. As elevated HbA1c and dyslipidemia are independent risk factors of very high risk group for CVD [24].
Current study was aimed to endothelial dysfunction circulation of Lipid level analysis in patients of Myocardial Infarction admitted.

PATIENTS AND METHODS:

Clinically diagnosed & confirmed cases of diabetic retinopathy in age group 35 to 74 years. The study was approved by the Institute Ethics Committee, and informed consent was obtained from all the case and control subjects from the OPD and IPD of Santosh University and Ganesh Shankar Vidyarthi Memorial Medical College, Kanpur. The study period was January 2018 to 1 December 2019.

This prospective study was conducted on 55 Acute Myocardial Infarction patients and 55 ages matched healthy controls. The MI patients were classified into STEMI (ST-elevated myocardial infarction, N = 29); NSTEMI (non-ST-elevated myocardial infarction, N = 04) and (Chest pain, N= 22) Mean ages of STEMI, NSTEMI, and chest pain patients were 29(52.8%), 4(7.2%), and 22(40%), respectively (Table 1).

Methodology: Retrospective cohort studies

Study sample: Case groups were confirmed as a study group, and distinguished from Control groups, that is, treat as healthy subjects by following the basic selection criteria prescribed by Physicians and expert's cardiologist. Prescribed selection criteria are as follows:

Inclusion Criteria:

- Patients more than 55 years of age, ECG findings and biochemical markers: Suggestive of acute myocardial infarction
- Elevated level of CK-MB and Trop T
- Chest pain lasting 24 hours, suggestive of myocardial ischemia of accelerated pattern, or a prolonged one (>20 minutes), or with recurrent episodes at rest, or at minimal excretion, in addition to at least one of the following:
    - (a) New or presumed ECG changes (any of the following three characteristics): ST-segment depression ≥ 0.5 mm, transient ST-segment elevation (< 20 minutes) ≥ 1 mm, T-wave inversion ≥ 3 mm in two or more contiguous leads;
    - Development of pathological Q waves in the ECG
    - (b) Raised levels of cardiac markers (CK ≥ 2X the upper limit of normal).

Exclusion Criteria:

- Known causes of elevated uric acid level (chronic kidney disease, gout, hematological malignancy, and hypothyroidism).
- Patients on drugs which increase serum uric acid e.g. salicylates (2gm/dl, hydrochlorothiazide, pyrazinamide).
- Chronic alcoholics.
- Acute phase of impaired subject of obesity (body mass index > 30) was excluded. In addition, patients receiving medications affecting lipid metabolism, such as lipid lowering drugs, beta-blockers, oral contraceptives, estrogen, thyroxin and vitamin E was also excluded.
- Present or past aspirin, statins or hormone replacement therapy, autoimmune diseases and malignancies smokers, Subjects with any chronic diseases or acute infections, antioxidant vitamin supplements, hepatic disease etc.
- Renal dysfunction, Myocarditis, Rhabdomyolysis, Cardiomyopathy, Cardiac Surgery, Stroke, etc.

Laboratory Methodology

Blood Samples and Biochemical Measurements

The fasting blood samples were collected from the study and control subjects for blood glucose, lipid profile (total cholesterol, triglyceride, high and low-density lipoprotein cholesterol), high-sensitivity C-reactive protein (hsCRP), and blood sugar fasting. The biochemical tests were carried out on a Merck® Microlab 300 analyzer.

Measurement of Blood sugar, Lipid Profile and High sensitivity C- reactive protein Concentration

The serum/plasma concentrations of estimation of blood glucose by GOD POD Oxidase [25] and lipid profile Cholesterol (TC) by CHOD-PAP METHOD [26], Triglycerides (TG) by GPO-TOPS method[27], HDL by selective inhibition method[28], LDL-C and VLDL will be calculated by Friedwald and Fredricson formula were determined using a commercially available kit (were purchased from Laboratory Erba Biochemistry Reagents, USA). Serum high sensitive C-reactive protein (hs-CRP) measurements by immunoturbidimetry method[29] were determined through turbidimetric immunoassay method using a commercially available kit (were purchased from Agappe Diagnostics Switzerland) according to the manufacturer's protocol. The biochemical tests were carried out on a Merck® Microlab 300 analyzer, Serum and plasma samples were stored at -80^{o}C until analyzed.

Body Mass Index (BMI) Assessment

Anthropometric, lifestyle, and dietary data were derived from the questionnaire administered to female and male group, with missing information substituted from previous questionnaires.
BMI calculated using the equation BMI = weight [Kg]/height[m] ^{2}

Blood Pressure Measurement

Systolic and diastolic blood pressure measured in a sitting position, after a 5 minute rest, using a mercurial sphygmomanometer instrument.

STATISTICAL ANALYSIS:

The results are presented in mean±SD and percentage. Chi-square test was used to compare the categorical variables between cases and controls. Unpaired t-test was used to compare the study parameters between cases and controls. The Pearson correlation coefficient was calculated among the study parameters. The p-value<0.05 was considered significant. All the analysis was carried out by using IBM SPSS 21.0 version (Chicago, Inc., USA).

RESULTS:

Clinical attribute, lipid profile of STEMI, NSTEMI & Chest pain patients. **Table1** show that a significant regarding age and body mass index between case and Control subjects age and body mass index was not significance. Differ between the two groups case group MI and Controls. MI severity, n (%) MI type STEMI 29(52.8%) and NSTEMI 4(7.2%) and 22(40%) Chest Pain patients.

Table 1: Sociodemographic characteristics of the Myocardial Infarction and control subjects

Characteristics	Myocardial Infarction (n=55)	Control (n=55)	SD Error	p-Value
Sociodemographics				

Age, Median	56.76±10.60	52.49±13.24	2.287	0.0231
Body Mass Index	12.35±2.22	11.93±1.92	0.396	0.2910
Diabetes mellitus	10(18.1%)	-	-	-
MI severity, n (%) MI type				
STEMI	29(52.8%)			
NSTEMI	4(7.2%)			
Chest Pain	22(40%)			
Co morbidities and MI risk factors, n (%)				
Hypertension	37(67.2%)	-	-	-
DM with HTN	15(27.2%)	-	-	-
Dyslipidemia	24 (43.6%)			
Smoker	30(54.5%)	-	-	-
Alcohol	11 (20%)	-	-	-

Two-tailed p- value <0.0001**= Statistically Significant, significant at the <0.05 level, Unpaired t-test

Data was expressed as ± SD. The continuous data was analyzed by using student's t-test. Abbreviations: Hypertension (HTN), Diabetes Mellitus (DM), Non ST-segment elevation myocardial infarction (NSTEMI), ST-segment elevation myocardial infarction (STEMI)

Table 2: Biomarkers of Myocardial Infarction in case and control subjects

Variables	Myocardial Infarction(n=55)	Control(n=55)	SD Error	p-Value
hsCRP (mg/L)	4.69±1.80	0.52±0.24	0.245	<0.0001**
FBS (mg/dl)	173.91±47.03	81.91±11.18	6.518	<0.0001**
Lipid profile				
TC (mg/dl)	220.97±68.75	144.71±43.93	11.001	<0.0001**
TG (mg/dl)	218.12±94.92	106.11±34.48	13.617	<0.0001**
HDL (mg/dl)	41.21±4.51	81.78±19.73	2.729	<0.0001**
LDL (mg/dl)	85.34±16.81	41.84±5.46	2.383	<0.0001**
VLDL (mg/dl)	43.47±21.27	21.37±6.97	3.018	<0.0001**
TG/HDL-c	5.37±2.51	2.11±0.58	0.347	<0.0001**
LDL/VLDL	2.42±1.24	1.31±0.34	0.173	<0.0001**
TC/HDL-c	5.44±1.81	1.80±0.46	0.252	<0.0001**

Two-tailed p- value <0.0001**= Statistically Significant, significant at the <0.05 level, Unpaired t-test

Data was expressed as ± SD. The continuous data was analyzed by using student's t-test.

Abbreviations: High sensitivity C-reactive protein (hsCRP), Fasting Blood Sugar (FBS), Total Cholesterol (TC), Triglyceride (TG), High-density lipoprotein (HDL), Low-density lipoprotein (LDL), Very low-density lipoproteins (VLDL)

Table 2 shows a comparison of clinical characteristics between Myocardial Infarction patients with Controls group subjects. The hsCRP, Fasting blood sugar and lipid profile levels of MI and controls group was significance (p<0.0001**).

Table 3: Biochemical parameter of ST segment elevated Myocardial Infarction with case and controls subjects

Variables	STEMI (n= 29)	Control (n=55)	SD Error	p-Value
hsCRP (mg/L)	5.57±1.91	0.52±0.24	0.260	<0.0001**
FBS (mg/dl)	196.12±54.23	83.65±11.33	7.470	<0.0001**
Lipid profile				
TC (mg/dl)	251.01±72.74	142.82±38.18	11.077	<0.0001**
TG (mg/dl)	277.89±92.07	105.93±33.37	13.205	<0.0001**
HDL (mg/dl)	41.57±4.20	80.96±21.08	2.898	<0.0001**
LDL (mg/dl)	85.91±20.19	42.27±5.92	2.837	<0.0001**
VLDL (mg/dl)	55.73±22.21	21.32±6.57	3.123	<0.0001**
TG/HDL-c	6.81±2.60	2.12±0.57	0.359	<0.0001**
LDL/VLDL	1.87±1.25	1.34±0.36	0.175	<0.0001**
TC/HDL-c	6.10±1.91	1.78±0.34	0.262	<0.0001**

Two-tailed p- value <0.0001**= Statistically Significant, significant at the <0.05 level, Unpaired t-test

Data was expressed as ± SD. The continuous data was analyzed by using student's t-test.

Abbreviations: High sensitivity C-reactive protein (hsCRP), Fasting Blood Sugar (FBS), Total Cholesterol (TC), Triglyceride (TG), High-density lipoprotein (HDL), Low-density lipoprotein (LDL), Very low-density lipoproteins (VLDL)

Table 3 shows a ST segment elevated Myocardial Infarction cases and Controls groups significant (p value <0.0001**) of Increase levels of cases hsCRP, FBS, TC, TG, VLDL, LDL, TG/HDL-c, LDL/VLDL, TC/HDL- c and Controversial HDL-c was significant and decrease in cases (p value <0.0001**).

Table 4: Correlation of biochemical parameter of ST segment elevated Myocardial Infarction with case

		FBS mg/dl	TC mg/dl	TG mg/dl	HDL mg/dl	LDL mg/dl	VLDL mg/dl	TG/HDLc	LDL/VLDL	TC/HDLc	hsCRP mg/L
FBSmg/dl	Pearson Correlation	1	.245	.626**	-.077	.019	.617**	.570**	-.461*	.250	.287
	Sig. (2-tailed)		.201	.000	.692	.921	.000	.001	.012	.192	.132
	N		29	29	29	29	29	29	29	29	29
TCmg/dl	Pearson Correlation		1	.577**	-.067	.382*	.577**	.529**	-.276	.949**	.816**
	Sig. (2-tailed)			.001	.731	.041	.001	.003	.147	.000	.000
	N		29	29	29	29	29	29	29	29	29
TGmg/dl	Pearson Correlation			1	-.284	.142	.964**	.967**	-.631**	.643**	.466*
	Sig. (2-tailed)				.136	.461	.000	.000	.000	.000	.011
	N			29	29	29	29	29	29	29	29
HDLmg/dl	Pearson Correlation				1	-.034	-.284	-.499**	.087	-.363	.079
	Sig. (2-tailed)					.860	.136	.006	.653	.053	.683
	N				29	29	29	29	29	29	29
LDLmg/dl	Pearson Correlation					1	.115	.129	.317	.374*	.422*
	Sig. (2-tailed)						.552	.505	.094	.046	.023
	N					29	29	29	29	29	29
VLDLmg/dl	Pearson Correlation						1	.935**	-.695**	.644**	.487**
	Sig. (2-tailed)							.000	.000	.000	.007
	N						29	29	29	29	29
TG/HDLc	Pearson Correlation							1	-.585**	.668**	.379*

	Sig. (2-tailed)								.001	.000	.042
	N							29	29	29	29
LDL/VLDL	Pearson Correlation								1	-.291	-.220
	Sig. (2-tailed)									.125	.251
	N								29	29	29
TC/HDLc	Pearson Correlation									1	.722**
	Sig. (2-tailed)										.000
	N									29	29
hsCRP mg/L	Pearson Correlation										1
	Sig. (2-tailed)										
	N										29

**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).

Table 4: Shows the correlation represents the quantitative measurements of degree of relationship among different variables. The FBS and hsCRP was not significant (r=0.287, p=0.132) in cases. The FBS and TG was significant (r=0.626**, p=0.00) hsCRP and TC was significant (r=0.816, p=0.00); hsCRP and TG (r=0.466, p=0.11); hsCRP and VLDL (r=0.487, p=0.00); hsCRP and TG/HDL (r=0.722, p=0.00). There is negative correlation between hsCRP and LDL/VLDL (r= -0.220 p=0.251); FBS and HDL (r=-0.77, p=0.692) in cases.

Table 5: Biochemical parameter of Non ST segment elevated Myocardial Infarction with case and controls subjects

Variables	Non STEMI (n=4)	Control (n=55)	SD Error	p-Value
hsCRP (mg/L)	4.81±2.33	0.41±0.24	0.316	<0.0001**
FBS (mg/dl)	169±58.17	80±7.87	7.915	<0.0001**
Lipid profile				
TC (mg/dl)	218.12±82.18	133±33.84	11.984	<0.0001**
TG (mg/dl)	199.25±106.21	76.75±10.99	14.398	<0.0001**
HDL (mg/dl)	43.50±9.33	83±16.30	2.532	<0.0001**
LDL (mg/dl)	84.65±5.30	38.25±1.25	0.734	<0.0001**
VLDL (mg/dl)	37.35±23.56	16.35±2.28	3.192	<0.0001**
TG/HDL-c	4.69±2.38	2.37±0.32	0.324	<0.0001**
LDL/VLDL	2.86±1.42	0.93±0.10	0.192	<0.0001**
TC/HDL-c	5.32±2.33	1.39±0.43	0.319	<0.0001**

Two-tailed p- value <0.0001**= Statistically Significant, Unpaired t-test
Data was expressed as ± SD. The continuous data was analyzed by using student's t-test.
Abbreviations: High sensitivity C-reactive protein (hsCRP), Fasting Blood Sugar (FBS), Total Cholesterol (TC), Triglyceride (TG), High-density lipoprotein (HDL), Low-density lipoprotein (LDL), Very low-density lipoproteins (VLDL)

Table 5: Shown the difference for Lp (a) levels was significant between the two groups Non STEMI vs Control of TC, TG, LDL, and HDL, VLDL, TG/HDL, LDL/VLDL, TC/HDL levels and hsCRP and FBS was significant between the two groups Non STEMI vs Control Subjects (p=0.0001**).

Table 6: Correlation of biochemical parameter of Non ST segment elevated Myocardial Infarction

Correlations

		FBS mg/dl	TC mg/dl	TG mg/dl	HDL mg/dl	LDL mg/dl	VLDL mg/dl	TG/HDLc	LDL/VLDL	TCHDLc	hsCRPmg/L
FBSmg/dl	Pearson Correlation	1	.694	.992**	.162	.662	.951*	.894	-.712	.391	.918
	Sig. (2-tailed)		.306	.008	.838	.338	.049	.106	.288	.609	.082
	N	4	4	4	4	4	4	4	4	4	4
TCmg/dl	Pearson Correlation		1	.771	-.527	.399	.881	.890	-.981*	.902	.861
	Sig. (2-tailed)			.229	.473	.601	.119	.110	.019	.098	.139
	N		4	4	4	4	4	4	4	4	4
TGmg/dl	Pearson Correlation			1	.039	.611	.980*	.941	-.793	.501	.958*
	Sig. (2-tailed)				.961	.389	.020	.059	.207	.499	.042
	N			4	4	4	4	4	4	4	4
HDLmg/dl	Pearson Correlation				1	.501	-.139	-.294	.566	-.837	-.232
	Sig. (2-tailed)					.499	.861	.706	.434	.163	.768
	N				4	4	4	4	4	4	4
LDLmg/dl	Pearson Correlation					1	.568	.375	-.275	-.026	.398
	Sig. (2-tailed)						.432	.625	.725	.974	.602
	N					4	4	4	4	4	4
VLDLmg/dl	Pearson Correlation						1	.976*	-.893	.654	.980*
	Sig. (2-tailed)							.024	.107	.346	.020
	N						4	4	4	4	4
TGHDLc	Pearson Correlation							1	-.936	.747	.998**
	Sig. (2-tailed)								.064	.253	.002
	N							4	4	4	4
LDLVLDL	Pearson Correlation								1	-.924	-.910
	Sig. (2-tailed)									.076	.090
	N								4	4	4
TCHDLc	Pearson Correlation									1	.700
	Sig. (2-tailed)										.300

	N									4	4
	Pearson Correlation										1
hsCRPmg/L	Sig. (2-tailed)										
	N										4

**. Correlation is significant at the 0.01 level (2-tailed).

*. Correlation is significant at the 0.05 level (2-tailed).

Table 6: Shows the correlation represents the quantitative measurements of degree of relationship among different variables. The hsCRP and HDL was not significant (r=0.287, p=0.132) in cases. The FBS and TG was significant (r=0.992, p=0.008); hsCRP and TG was significant (r=0.958, p=0.042); hsCRP and TG (r=0.466, p=0.11); hsCRP and VLDL (r=0.487, p=0.00); hsCRP and TG/HDL (r=0.722, p=0.00). There is negative correlation between hsCRP and HDL (r= -0.232 p=0.768); hsCRP and LDL/VLDL (r=-0.910, p=0.090), FBS and LDL/VLDL (r=-0.712, p=0.288) in cases.

Table 7: Biochemical parameter of Chest Pain with Myocardial Infarction in case and controls subjects

Variables	Non STEMI (n=29)	Control (n=55)	SD Error	p-Value
hsCRP (mg/L)	3.61±0.98	0.53±0.25	0.136	<0.0001**
FBS (mg/dl)	148.18±14.02	79.63±11.40	2.437	<0.0001**
Lipid profile				
TC (mg/dl)	183.24±40.05	153.22±50.86	8.729	0.0008
TG (mg/dl)	150.17±36.03	112.31±36.28	6.895	<0.0001**
HDL (mg/dl)	40.48±3.89	83.31±19.57	2.690	<0.0001**
LDL (mg/dl)	85.79±12.72	41.90±5.19	1.852	<0.0001**
VLDL (mg/dl)	29.90±7.40	22.46±7.25	1.397	<0.0001**
TG/HDL-c	3.74±0.97	2.01±0.60	0.154	<0.0001**
LDL/VLDL	3.03±0.86	1.35±0.28	0.122	<0.0001**
TC/HDL-c	4.58±1.19	1.87±0.56	0.177	<0.0001**

Two-tailed p- value <0.0001**= Statistically Significant, Unpaired t-test
Data was expressed as ± SD. The continuous data was analyzed by using student's t-test.
Abbreviations: High sensitivity C-reactive protein (hsCRP), Fasting Blood Sugar (FBS), Total Cholesterol (TC), Triglyceride (TG), High-density lipoprotein (HDL), Low-density lipoprotein (LDL),Very low-density lipoproteins (VLDL)

Table 7: Shown the difference for Lp (a) levels was significant between the two groups Chest Pain with Myocardial Infarction vs Control of TC, TG, LDL, and HDL, VLDL, TG/HDL, LDL/VLDL, TC/HDL levels and hsCRP and FBS was significant between the two groups Chest Pain with Myocardial Infarction vs Control Subjects (p=0.0001**).

Table 8: Correlation of biochemical parameter of Chest Pain with Myocardial Infarction in case subjects

		FBS mg/dl	TC mg/dl	TG mg/dl	HDL mg/dl	LDL mg/dl	VLDL mg/dl	TG/HDLc	LDL/VLDL	TC/HDLc	hsCRP mg/L
FBSmg/dl	Pearson Correlation	1	.300	.140	-.063	-.067	.130	.143	-.175	.274	.076
	Sig. (2-tailed)		.176	.534	.782	.766	.564	.527	.435	.218	.736
	N	22	22	22	22	22	22	22	22	22	22
TCmg/dl	Pearson Correlation		1	.196	-.234	.003	.201	.268	-.205	.934**	.544**
	Sig. (2-tailed)			.383	.294	.990	.369	.227	.360	.000	.009
	N		22	22	22	22	22	22	22	22	22
TGmg/dl	Pearson Correlation			1	.004	-.028	.998**	.923**	-.840**	.168	.118
	Sig. (2-tailed)				.985	.900	.000	.000	.000	.455	.602
	N			22	22	22	22	22	22	22	22
HDLmg/dl	Pearson Correlation				1	.417	.035	-.371	.217	-.561**	-.200
	Sig. (2-tailed)					.053	.879	.089	.332	.007	.373
	N				22	22	22	22	22	22	22
LDLmg/dl	Pearson Correlation					1	-.004	-.163	.514*	-.141	-.237
	Sig. (2-tailed)						.988	.469	.014	.533	.289
	N					22	22	22	22	22	22
VLDLmg/dl	Pearson Correlation						1	.911**	-.829**	.161	.123
	Sig. (2-tailed)							.000	.000	.473	.585
	N						22	22	22	22	22
TGHDLc	Pearson Correlation							1	-.840**	.366	.172
	Sig. (2-tailed)								.000	.094	.444
	N							22	22	22	22
LDLVLDL	Pearson Correlation								1	-.250	-.212
	Sig. (2-tailed)									.262	.344

	N								22	22	22
TCHDLc	Pearson Correlation									1	.549**
	Sig. (2-tailed)										.008
	N									22	22
hsCRPmg/L	Pearson Correlation										1
	Sig. (2-tailed)										
	N										22

**. Correlation is significant at the 0.01 level (2-tailed).
*. Correlation is significant at the 0.05 level (2-tailed).

Table 8: Shows the correlation represents the quantitative measurements of degree of relationship among different variables. The hsCRP and TC was positive correlation and significant (r=0.544, p=0.009) in cases. There is negative correlation and not significance between hsCRP and HDL (r= -0.200 p=0.373); hsCRP and LDL (r=-0.237, p=0.287), hsCRP and LDL/VLDL (r=-0.212, p=0.344) in cases.

DISCUSSION:

The characteristics of myocardial infarction and healthy controls are summarized in Table 1. The Socio-demographic characteristics of the Myocardial Infarction of associated diseases was well-known for hypertension, Dyslipidemia, Smoker and Diabetes mellitus 29/40 (52.8%) STEMI, 4/40 (7.2%) NSTEMI and 22/40 (40%) chest pain.

Our data indicated that: (1) myocardial infarction patients had age & sex-related division in lipid profiles; (2) dyslipidemia was increasingly predominant among the myocardial infarction; (3) myocardial localized necrosis were inclined to more significant levels of LDL; and (4) decrease level of HDL was the most common in dyslipidemia with myocardial infarction. Compared to these previous studies, our sample was much larger, recruited nationwide and included only patients with a recent MI. Currently the fact that there was no established method or predicting long-term risk of recurrence post-Myocardial infarction, we put forth an attempt by receiving indicators of a remotely approved model produced for patients with ongoing Myocardial infarction and a coordinating essential composite outcome [30]. Our outcomes support the conflict that the all around recorded predictive role lipid levels for the risk of a first event of ASCVD can't be extrapolated to the secondary prevention setting post-MI [31-32].
Our result showed significance increase in case control total cholesterol (220.97±68.75 vs 144.71±43.93, p=0.0001**), triglyceride (218.12±94.92 vs 106.11±34.48, p=0.0001**), low density lipoprotein (85.34±16.81 vs 41.84±5.46, p=0.0001**), Very low density lipoprotein lipoprotein (43.47±21.27 vs 21.37±6.97, p=0.0001**), and high sensitivity C reactive protein (4.69±1.80 vs 0.52±0.24, p= 0.0001**) and decrease level of High density cholesterol levels

(41.21±4.51 vs 81.78±19.73, p= 0.0001**) in MI patients (Table 2). This is comparable with those announced in past investigations expanded spotlight on focusing on and treating low serum levels of HDL with an effort to additionally increase risk for cardiovascular event, including myocardial infarction [33].

Haseeb A et al [34] observed elevated levels of Total Cholesterol and Low Density Lipoprotein in patients with complicated versus those with non-complicated clinical course of infarction, proposing more significant levels of these biomarkers elevated in the during initial 24 hours of MI have a solid negative prognostic value. In our investigation, it is vital that in spite of the fact that the TC/HDL proportion had demonstrated to be related with a higher number of vessels affected, it was not correlated with the level of stenosis in MI patients with non-ST- segment elevation ACS (NSTEACS) , unlike the other lipid variables. This can be clarified by the fact that the lesions that are all the more potentially unstable and prone to rupture are frequently non-occlusive and not diagnosed by angiography [35] Then again, these lesions have an enormous lipid nucleolus signs of active inflammation and macrophage accumulation at the site of plaque rupture [23] In any case, the nonappearance of a relationship between serum LDL cholesterol and oxidized LDL-cholesterol in the plaque has been exhibited in ACS [36]. We find out the hsCRP is positively correlated with resting myocardial infarction status. Also, hsCRP and lipid profile are correlated with level of myocardial injury. Diabetes, smoking history, hypertension etc, and hsCRP ≥6 mg/L are important predictors of significant myocardial infarction. Tanveer et al. [37] analyzed 190 patients with STEMI to examine the association between hs-CRP level and complications of myocardial infarction. However, there were no significant differences in hs-CRP values between patients who died and who survived during hospitalization.

Our outcome of the result was significant association among hs-CRP level on admission and in-hospital mortality after STEMI. This is comparable to previous studies (3.2-20.6%) [38-39]. Our results also demonstrated that in-hospital mortality after STEMI was strongly associated with elderly patients. Past studies have examined the correlation between age and death in patients with myocardial infarction and showed that age is a significant indicator of mortality in these cases [40].

CONCLUSION :

Our findings demonstrate that hsCRP has strong significant association with lipid in myocardial infarction cases. Therefore, a decrease in serum HDL and elevated level of hsCRP strongly significance incline the dangerous risk individuals to the occurrence of Myocardial infarction. We underline the significance of HDL and hsCRP estimations in the assessment of a consolidated inflammatory risk factor for the screening of high risk individuals and the diagnostic & prognosis of Myocardial infarction. We recommend that Lipid profile must be assessed in all patients conceded for Myocardial infarction to comprehend the evolving pattern, start way of changing trend, initiate lifestyle measures to reach target lipid levels, and predict the choice of lipid lowering therapy. Therefore, hsCRP on admission of patients with STEMI is a strong univariate predictor of mortality.

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Review concept – P K & MKV
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Conflict of Interest
The authors confirm that there are no conflicts of interest.

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