Biochemical Study For Evaluation of some Clinical Parameters as Predictors of Myocardial Infarction in Patients With Type 2 Diabetes Mellitus Undergoing Elective Percutaneous Coronary Intervention

[*]Rasha Hasan Jasim and Sara Sattar Jabbar
e-mail: rasha.alfahham@uokufa.edu.iq
***Department of Chemistry-Factually of Education for Girls-University of Kufa-Iraq**

Abstract

When blood flow to the heart muscle is impeded, fat and cholesterol build up on the inner wall of the heart, causing coronary heart disease (CHD), myocardial infarction (MI), and sudden cardiac death are just a few of the diseases that can occur in CAD as a result of atherosclerosis. A catheter is used during Percutaneous Coronary Intervention (PCI), also known as angioplasty, a non-surgical procedure to squeeze a balloon or stent into a narrowing area to restore blood flow to the myocardium. Therefore, it is treated with medication to widen or open up the veins in the heart muscle that contain plaque accumulation that results in stenosis in order to avoid the coronary artery blockage from occurring again. The key CVD risk factor is lipid profile and some biochemicals parameters, and each kind these parameters are associated with an increase in the incidence of MI. Anabolic processes, energy metabolism, and blood sugar levels are all controlled by the hormone insulin. Glycosylated hemoglobin (HbA1c) is frequently used to measure the average blood glucose levels of a person based on the age. The resulting values also help to diagnose diabetes mellitus by determining the blood sugar level. Elevated aminotransferase levels indicating liver function, have attracted great concern as potential novel markers of cardiovascular risk assessment. Although blood urea nitrogen (BUN), creatinine (Cr), and electrolytes is not the mainstay of diagnosis in MI patients, they may have a role in providing a more detailed view of the complications and mortality rates.

Subjects: *100 individuals were collected to contribute in the present study. These cases were divided into two groups, the first included 70 patients (their age ranged between 30-66 years) with MI who underwent to elective PCI. The second group involved 30 healthy individuals (with the age range 30-55 years) were enrolled in the present study as a control group*

Results: *The results showed significant increase ($p=0.000$) for lipid profile, glucose, insulin, HbA1C% and HOMO-IR, while a significant decrease ($p<0.05$) in HDL, urea, creatinine levels and ALP was recorded when the two study groups were compared together.*

Keywords: **Insulin, Glucose, CVD,** *coronary heart disease* **CHD,** *coronary artery disease* **CAD,** *Percutaneous Coronary Intervention* **PCI,** ischemic heart disease **IHD,**

Glycosylated hemoglobin **HbA1C%, HAMO-IR, Lipid profiles, liver function test , ACS,** *blood urea nitrogen* **BUN &** *creatinine* **Cr,** *Myocardial infarction* **MI,** *triglycerides* ***TG,*** *, low-density lipoprotein* **LDL,** *high-density lipoprotein* **HDL,** *Non protonic nitrogen* **NPN,** *Total protein* **TP,** *Glutamine pyruvate transaminase* **GOT,** *Glutamine oxaloacetate transaminase* **GOT,** *Alkaline phosphatase* **ALP,**

Introduction

Myocardial Infarction (MI) it is commonly called heart attack when there is a lack in the blood supply or a complete interruption of oxygenated blood to the myocardium, due to the partials or complete blockage of coronary artery with the accumulation of atherosclerotic plaque with unstable collection of lipids (cholesterol and fatty acids) and white blood cells in artery walls in the coronary artery. Blockage of coronary artery is considered the most common cause of heart attack, if the condition left untreated it can cause damage or death (infarction) to the heart muscle tissue (myocardium) finally leading to heart failure [1]. A catheter is used during Percutaneous Coronary Intervention (PCI) also known as angioplasty, a non-surgical procedure to squeeze a balloon or stent into a narrowing area to restore blood flow to the myocardium **[2].** Lipids are the basic and necessary materials for the needs to the body of human exemplified by total cholesterol, phospholipids, fatty acids, and triglycerides (TG), all of them are involved in the formation of cell membranes and some of them are originator to steroid hormones, as lipids are the most powerful source of energy in the body . Where increase total cholesterol, triglycerides, low-density lipoprotein (LDL) levels, and decrease high-density lipoprotein (HDL) levels in serum. **[3-5]**. Profiling lipids in samples might advance the discovery of diagnostic, prognostic, and monitoring disease biomarkers. The understanding of homeostatic and inflammatory processes related to chronic inflammatory systemic diseases such as CVDs including MI **[6] .** A lack in insulin function has been linked to changes in metabolic pathways, all of which lead to an increased risk of CVDs people with diabetes who have high levels of free fatty acids that need to consume a lot of oxygen for metabolism, which can affect the function of the myocardium and lead to the intracellular accumulation of toxic mediators **[7, 8]**. Liver plays a crucial role in the synthesis of vital components; such as coagulation proteins, albumin, immune complement system proteins and transport proteins, also has an important role in nutrients metabolism functions such as lipids metabolism and lipoproteins through the biosynthesis of cholesterol, fatty acids, apolipoproteins, and proteins involved in lipoprotein homeostasis **[9].** Urea is the one of nitrogenous chemicals created primarily by the liver's protein metabolism, It is carried by the blood to the kidneys where it is eliminated with urine Creatinine, also a NPN waste product, is produced by the breakdown of creatine and phosphocreatine and can serve as an indicator of renal function. Arginine, Glycine, and Methionine are transaminated to form creatine in the liver, pancreas, and kidneys **[10, 11].**

Subjects and Methods

One hundred samples were enrolled and divided into two main groups, the first of which was 70 (patients with age ranged between 30-66 years) the current study essential the elimination of a group of patients (Patients with Covid-19 virus who have undergone elective PCI, people with cancer, patients who are undergoing cancer treatments or who have been cured, patients with thyroid diseases, patients with liver diseases, patients with diabetes complications added to cardiopathy, patients who underwent any surgical intervention during 5 years of MI occurring, whether or not the surgery involved heart disease, patients who are on the keto diet during the period of the onset of symptoms of heart attack and alcohol drinkers were excluded, 70 patients were divided to two subgroups 44 male patients with and 26 female patients. While the second involved 30 healthy individuals were divided into 16 healthy male and 14 healthy female with the age range (30- 55 years). A subjective perception of good health as determined by health questionnaire; healthy controls should have no medical history of heart disorders; control individuals are non-smokers and not alcohol drinkers. individuals should not take any medication during at least one year before study carrying out; they have not undergone surgical intervention or any illness requiring hospitalization, and the control group might be at an approximate age range with the patients group. Five milliliters of intravenous blood samples were taken from patients (after subjecting them to the PCI procedure) and healthy subjects while the participants were fasting for at least 8 hours. after it was divided into two portions: **(1)** Two mL were placed in an EDTA tube to determination HbA1c% level. **(2)** Three mL were placed in a plane tube at laboratory room temperature for measuring glucose and insulin, TC, TG, HDL, LDL ,vLDL, urea ,creatinine, urea/creatinine ratio and liver function test levels. The samples were left to coagulate at laboratory temperature, analyzers then separated using a centrifuge at 3000 xg for 15 minutes.

Result and Dissection

Evaluation of lipid profile levels, Glucose, Insulin and HbA1c% levels in the sera of the two main study groups (patients with elective PCI and healthy individuals are shown in **Table 1.** Noted that there are significant increases (**p=0.000**) in the TG , Cholesterol and LDL, vLDL, glucose, insulin, HbA1c% ,HOMO-IR,urea ,creatinine, urea/creatinine ratio the patients group comparison to controls group, while there is a significant decrease (**p<0.05**) in the sera HDL and liver enzymes levels of patients comparing to controls.

Table 2: in the current study illustrates significant elevation (p=0.000) in the lipid profiles, urea and creatinine and the vital ratios when males subgroups compared together, the same results were recorded when the females subgroups compared together. Lipid profiles, urea and creatinine showed that there are no statistical

differences comparing subjects in the same groups either in the patients nor healthy groups, Also a high levels of TC, TG, LDL-C and a low levels of HDL-C, in male PCI patients compared to their peers in the healthy group, in contrast high levels of urea showed in female patients. On the other hand non-significant differences ($p>0.05$) between the two genders in the same group when glucose, insulin, HOMO-IR and HbA1C%, were tested in the four subgroups of the study. On the other hand, the current study has demonstrated that significant differences were recorded when the comparison between the same genders in the study groups were carried out using ANOVA test, as demonstrated. On the other hand, the current study has demonstrated non-significant differences were recorded between the same gender, either healthy nor patient in the GOT,GPT and TP, except in ALP which illustrates a significant differences between females together in the both main groups.

Table3 showed positive relationships when TGs correlated to LDL-C, vLDL-C, Chol./HDL, TG/HDL and LDL/HDL in the sera of PCI patients, while this relationship seemed to be insignificant negatively with HDL-C ($r=-0.124$ at $p=0.308$).In the same way the results recorded negative correlations among HDL-C and LDL-C, v LDL-C, as well as; the vital lipid profiles ratios (Cholesterol/HDL, TG/HDL and LDL/HDL). In contrast, the outcomes of the study recorded positive correlations with statistical significance between LDL-C and vLDL-C as well as the vital lipid profiles ratios. Same result was observed when vLDL-C was correlated to vital lipid profiles ratios. Outcomes of healthy individuals does not show reliable significant statistical variations between cholesterol and all of TGs, HDL-C, vLDL-C, and TGs/HDL. While, high significant positive correlations were noticed when cholesterol correlated to LDL-C, Cholesterol/HDL and LDL/HDL in the control group. The result of the present work indicates an insignificant correlation between TGs and HDL-C, LDL-C, Cholesterol/HDL as well as LDL/HDL ratio in the group of healthy individuals. On the other hand, it was noted that there is a strong positive link between TGs and vLDL-C in addition to LDL/HDL.Also data of the healthy group exhibited negative relationships when HDL-C correlated to all of LDL-C, vLDL-C, Cholesterol/HDL, TG/HDL and LDL/HDL; respectively. Significant high positively relationship between LDL-C and Cholesterol/HDL in addition to LDL/HDL in the sera of healthy group, while the relationships seemed to be non-significant when the correlations were carried out between LDL and vLDL as well as TG/HDL.

Table 1: Levels (Mean ± S.D.) of Lipid Profiles, Glucose, Insulin, HbA1C%, urea, creatinine, urea/creatinine ratio and liver function test levels in The Sera of The Study Individuals

Test	The Study Groups		*P value*
	Patients (70) Mean ± S.D. Min.- Mix. Range	Controls (30) Mean ± S.D. Min.- Mix. Range	
TG (mg/dL)	241.556 ± 44.727 128-345 217	98.327 ± 10.436 75-116.8 41.8	0.000
Cholesterol (mg/dL)	268.560 ± 43.871 151.7-361.6 209.9	115.695 ± 11.845 96.13-138.53 42.4	0.000
HDL (mg/dL)	29.501 ± 9.582 7.4-52 44.6	50.773 ± 2.636 44.5-56.5 12	0.000
vLDL (mg/dL)	48.439± 8.975 25.6-69 43.4	19.664 ± 2.088 15-23.36 8.36	0.000
LDL (mg/dL)	190.602 ± 38.545 92-273.5 181.5	44.957 ± 11.244 28.1-63.15 35.05	0.000
Cholesterol/HDL	10.712±6.986 0-46.1 46.1	2.279±0.251 1.9-2.7 0.8	0.000
TG/HDL	9.771±6.253 3.8-40.9 37.2	1.938±0.239 1.5-2.4 0.9	0.000
LDL /HDL	7.890±5.626 2.5-37 34.4	0.381±0.299 0.1-1.3 1.2	0.000
Glucose (mg/dL)	154.096 ±73.065 69-381 312	87.600 ± 15.732 60.6-120 59.4	0.000
HbA1C %	7.919 ± 2.252 4.15-13.88 9.72	5.297 ± 0.781 3.79-6.99 3.21	0.000
Insulin (mIU/L)	21.681 ± 6.427 13.89-68.82 54.93	12.560 ± 2.416 8.98-17.93 8.95	0.000
HOMO-IR	8.275 ± 4.585 2.9-27.6 24.7	2.747 ± 0.726 1.5-4.3 2.8	0.000
Urea (mg/dL)	32.720±12.547 18.3-93.8 75.5	18.950±3.214 13.1-26.7 13.6	0.001
Creatinine (mg/dL)	1.124±0.399 0.6-3.4 2.8	0.757±0.114 0.5-1 0.5	0.003
Urea/Creatinine ratio	27.886±8.895 14.2-73 58.8	25.253±3.858 16.6-30.9 14.3	0.041
GPT (U/L)	4.622±4.583 1-29 28	4.778±2.926 1-14 13	0.366
GOT (U/L)	23.849±11.999 4-65 61	23.185±9.523 10-60 50	0.418
ALP (U/L)	176.403±67.251 18-386 368	128.556±36.656 75-203 128	0.017
TP (mg/dL)	6.106±2.051 2.1-10.3 8.2	6.807±1.162 3.9-9 5.1	0.000

Table 2: Levels (Mean ± S.D.) of Lipid Profiles, Glucose, HbA1c and Insulin in Sera of T2D Patients underwent to PCI and Healthy Groups

Parameters	Subjects (N)				
	Patients with PCI 70		Controls 30		*P value*
	Males (44)	Females (26)	Males (16)	Females (14)	
	Mean ± S.D. Min-Max Range	Mean ± S.D. Min-Max Range	Mean ± S.D. Min-Max Range	Mean ± S.D. Min-Max Range	
Cholesterol (mg/dL)	267.573±43.665 200.45-361.6 161.15	270.230±45.034 151.7-354.7 203	115.970±10.85 101.06-138.53 37.47	115.49±12.88 96.13-135.32 39.19	1*vs*2: 0.777, 3*vs*4: 0.972, 1*vs*3: 0.000, 2*vs*4: 0.000
Triglycerides (mg/dL)	240.402±34.246 177.4-322.8 145.4	243.508±47.94 128345 217	95.856±10.092 81.96-116.8 34.84	100.216±10.595 75-112.9 37.9	1*vs*2: 0.744, 3*vs*4: 0.758, 1*vs*3: 0.000, 2*vs*4: 0.000
HDL-C (mg/dL)	27.531±8.828 7.40-42 34.6	32.834±10.048 9.7-52 42.30	51.630±3.149 45.1-56.5 11.4	50.117±2.025 44.5-54.3 9.8	1*vs*2: 0.008, 3*vs*4: 0.606, 1*vs*3: 0.000, 2*vs*4: 0.000
LDL-C (mg/dL)	191.941±37.958 118.77-273.5 154.73	188.336±40.173 92-267.6 175.6	44.476±10.67 28.1-62.93 34.83	45.325±11.977 28.28-63.15 34.86	1vs2:0.662, 3vs4:0.945 1vs3:0.000, 2vs4:0.000
vLDL-C (mg/dL)	48.079±8.649 35.48-64.56 29.08	49.047±9.644 25.6-69 43.4	19.170±2.019 16.39-23.36 6.97	20.042±2.12 15-22.58 7.58	1vs2:0.612, 3vs4:0.759 1vs3:0.000, 2vs4:0.000
Chol./HDL-C	11.295±7.752 0-46.14 46.14	9.724±5.871 4.35-30.94 26.59	2.250±0.261 1.88-2.65 0.77	2.301±0.249 1.9-2.7 0.8	1vs2:0.283, 3vs4:0.981 1vs3:0.000, 2vs4:0.000
TG/HDL-C	10.379±6.714 4.55-90.94 36.39	8.743±5.349 3.75-28.53 24.78	1.861±0.251 1.57-2.35 0.78	1.996±0.219 1.54-2.44 0.9	1vs2:0.212, 3vs4:0.945 1vs3:0.000, 2vs4:0.000
LDL-C/HDL-C	8.457±6.033 2.96-36.95 33.99	6.93±4.82 2.54-24.24 21.7	0.837±0.264 0.39-1.28 0.89	0.825±0.331 0.08-1.28 1.2	1vs2:0.195, 3vs4:0.995 1vs3:0.000, 2vs4:0.000
Glucose (mg/dL)	147.295±72.356 70-381 310.3	165.603±74.225 69-336.4 267.4	91.364±13.981 72.2-120 47.8	82.676±17.062 60.6-109 48.4	1*vs*2: 0.236, 1*vs*3: 0.000 3*vs*4: 0.705, 2*vs*4: 0.002
Insulin (mU/L)	22.404±7.568 16.8-68.8 52	20.458±3.606 13.9-26.8 12.9	12.001±2.249 9.1-17.9 8.9	13.291±2.519 8.9-16.9 7.9	1*vs*2: 0.159, 1*vs*3: 0.000 3*vs*4: 0.529, 2*vs*4: 0.000
HOMA-IR	8.239±4.99 3.3-27.6 24.3	8.335±3.898 2.9-17.9 15	2.688±0.583 1.8-3.9 2.1	2.8231±0.9 1.5-4.3 2.8	1*vs*2: 0.920, 1*vs*3: 0.000 3*vs*4: 0.926, 2*vs*4: 0.000
HbA1C%	7.828±2.32 4.2-13.9 9.7	8.074±2.168 5.1-13.6 8.4	5.046±0.744 3.8-6.8 3	5.624±0.729 4.5-7 2.5	1*vs*2: 0.610, 1*vs*3: 0.000 3*vs*4: 0.424, 2*vs*4: 0.000
Urea (mg/dL)	29.925±7.459 19.6-53.3 33.7	37.45±17.31 18.3.93.8 75.5	19.369±3.881 13.1-26.7 13.6	18.629±2.678 13.70-23.2 9.50	1vs2: 0.004, 3vs4: 0.846 1vs3: 0.000, 2vs4: 0.000
Creatinine (mg/dL)	1.165±0.295 0.61-1.85 1.24	1.296±0.528 0.59-3.4 2.81	0.827±0.125 0.58-1 0.42	0.703±0.067 0.57-0.84 0.27	1vs2: 0.120, 3vs4: 0.322 1vs3: 0.000, 2vs4: 0.000
Urea/Creatinine Ratio	26.811±6.309 14.2-58 43.8	29.703±11 16.6-73 56.4	23.615±4.37 16.6-30.6 14	26.505±2.965 19.9-30.9 11	1vs2: 0.132, 3vs4: 0.311 1vs3: 0.022, 2vs4: 0.890
GPT (U/L)	4.861±4.923 1-29 28	4.118±3.855 1-15 14	4.067±3.218 1-14 13	5.667±2.348 2-11 9	1*vs*2: 0.542, 3*vs*4: 0.319 1*vs*3: 0.322, 2*vs*4: 0.533
GOT (U/L)	24.944±12.097 12-65 53	21.529±11.806 4-55 51	24.200±12.131 10-60 50	21.917±4.889 16-32 16	1*vs*2: 0.307, 3*vs*4: 0.603 1*vs*3: 0.928, 2 *vs*4: 0.831
ALP (U/L)	179.361±76.876 18-386 368	172.308±52.262 87-277 190	127.933±30.450 77-174 97	129.333±44.661 75-203 128	1*vs*2: 0.651, 3*vs*4: 0.952 1*vs*3: 0.045, 2*vs*4: 0.007
TP (mg/dL)	6.128±2.018 2.1-10.3	6.069±2.144 2.2-9.1	6.500±1.210 3.9-8.3	7.208±1.001 6-9	1*vs*2: 0.898, 3*vs*4: 0.300 1*vs*3: 0.072, 2*vs*4: 0.481

8.2	6.9	4.4	3		

Table 3: Correlation of Evaluated Lipid Profiles in The PCI Patients Group

	Criteria	Cholesterol	TGs	HDL	LDL	vLDL	Chol./HDL	TG/HDL	LDL/HDL
r p	Cholesterol	1	0.910** 0.000	0.158 0.190	0.964** 0.000	0.920** 0.000	0.361** 0.002	0.418** 0.000	0.414** 0.000
	TG		1	-0.124 0.308	0.837** 0.000	0.993** 0.000	0.331** 0.005	0.417** 0.000	0.365** 0.002
	HDL			1	-0.398* 0.001	-0.134* 0.270	-0.80** 0.000	-0.795** 0.000	-0.804** 0.000
	LDL				1	0.849** 0.000	0.532** 0.000	0.576** 0.000	0.585** 0.000
	vLDL					1	0.335** 0.005	0.416** 0.000	0.369** 0.002
	Chol./HDL						1	0.978** 0.000	0.982** 0.000
	TG/HDL							1	0.992** 0.000
	LDL/HDL								1
Correlation of Evaluated Lipid Profiles in The PCI Healthy Group									
	Criteria	Cholesterol	TGs	HDL	LDL	vLDL	Chol./HDL	TG/HDL	LDL/HDL
r p	Cholesterol	1	0.170 0.368	0.093 0.627	0.951** 0.000	0.170 0.368	0.876** 0.000	0.103 0.587	0.689** 0.000
	TG		1	-0.054 0.777	0.016 0.933	1.00** 0.000	0.184 0.332	0.899** 0.000	-0.040 0.834
	HDL			1	-0.145 0.446	-0.054 0.777	-0.396 0.030	-0.481 0.007	-0.348 0.060
	LDL				1	0.016 0.932	0.946** 0.000	0.070 0.712	0.789* 0.000
	vLDL					1	0.184 0.331	0.899** 0.000	-0.040 0.835
	Chol./HDL						1	0.330 0.075	0.804** 0.000
	TG/HDL							1	0.114 0.549
	LDL/HDL								1

A frequent risk factor for atherosclerotic cardiovascular disease is hyperlipidemia. High levels of LDL-C and lower HDL-C levels have been linked to an increased risk of coronary heart disease (CHD) and other cardiovascular disorders. The morbidity of acute myocardial infarction (AMI), the most serious kind of CHD, is definitely connected with greater lipid levels. Obesity, hypertension, DM and old age are all risk factors for cardiovascular disease that can raise the chance of mortality. The results of the current work support the findings of previous studies show that hyperlipidemia is an independent influencing factor for myocardial infarction, or at least in the formation of the partial or total plug inside the blood vessels coinciding with the other risk factors. In addition, it can cause hyperlipidemia, with continuity and without changing the lifestyle to overlap one or more catheters, especially after a thrombosis forms inside the damaged vessel **[11-13].**

Table 4 shows highly positive correlations with the statistical significance levels ($r=0.452$ at $p<0.015$) and ($r=0.939$ at $p=0.000$), exclusively of glucose with each

HOMA-IR and HBA1C%. With the same way, the finding indicated to positively significant relationships for the insulin levels with HbA1C%, similar results between HOMA-IR and HbA1C% in the patient group. Besides a significant positive correlations were recorded when HOMA-IR with all of other parameters in the healthy group. Furthermore, the results indicated to significant positive correlations among insulin and HOMA-IR in addition to HbA1C%. The observed results in the current study revealed that there was no significant correlation between glucose and insulin in the control individuals.

Table 4: Correlation of Glucose Related Parameters in The PCI Patients Group

	Parameters	Glucose	Insulin	HOMA-IR	HbA1C%
r p	Glucose	1	0.018 0.882	0.825** 0.000	0.939** 0.000
	Insulin		1	0.572** 0.000	0.026 0.834
	HOMA-IR			1	0.781** 0.000
	HbA1C%				1
Correlation of Glucose Related Parameters in The Healthy Control Group					
	Parameters	Glucose	Insulin	HOMA-IR	HbA1C%
r p	Glucose	1	0.057 0.765	0.660** 0.000	0.306 0.100
	Insulin		1	0.728** 0.000	0.409* 0.025
	HOMA-IR			1	0.486** 0.006
	HbA1C%				1

There is an increasing incidence of T2DM worldwide. The risk of microvascular and macrovascular complications in diabetic patients increases as the disease develops. It is important to prevent diabetes and its macrovascular complications early and diagnose them as early as possible. Cardiovascular disease is one of the most common macrovascular complications in diabetic patients. It is complex process involving multiple factors that leads to diabetic cardiovascular disease. There is a strong link between atherosclerosis and MI with T2D. Several studies have demonstrated that many T2D patients suffer from vascular atherosclerosis resulting from hyperglycemia, insulin resistance, and hyperlipidemia, which should be taken into consideration as macrovascular complications are likely to occur **[14-16].** Hyperglycemia is a persistent risk factor for CVD when modifications in the vascular epithelium caused by T2D have already been done and cannot be undone by simply reducing blood glucose**[17]**.In epidemiological and pathophysiology studies, hyperglycemia has been identified as a key contributor to CVD **[18]**. The increase in blood sugar (more than 150 mg/dL) also leads to insulin resistance and gluconeogenesis, so hyperglycemia is a result of inflammation **[19]**.The combination of hyperglycemia and inflammation starts a vicious cycle that increases the risk of death in patients with segment elevated myocardial

infarction as well as the burden of atherosclerosis, carotid artery atherosclerosis, and plaque rupture **[20]**.

The results of the existing study which recorded high positive significant correlations (r=0.669 at p=0,000) and (r=0.489 at p=0.004), respectively among urea with creatinine and urea/creatinine ratio in the PCI patient, but a negative relationship was recorded (r=-0.282 at p=0.018) when creatinine linked to urea/creatinine ratio. Outcomes of the present study have recorded a positive insignificant correlation between urea and creatinine; and positive significant relationship (r=0.589 at p=0.001) between urea and urea/creatinine ratio, in contrast a negative correlation between creatinine and urea/creatinine ratio in the control group **(Table 5).**

Table 5: Correlation of Urea and Creatinine in The Elective PCI Patients Group

	Criteria	Urea	Creatinine	Urea/Creatinine Ratio
r p	Urea	1	0.669** 0.000	0.489** 0.004
	Creatinine		1	-0.282* 0.018
	Urea/Creatinine Ratio			1
Correlation among Urea, Creatinine and Urea/Creatinine Ratio in The Healthy individuals				
	Parameters	Urea	Creatinine	Urea/Creatinine Ratio
r p	Urea	1	0.507 0.331	0.586** 0.001
	Creatinine		1	-0.393* 0.032
	Urea/Creatinine Ratio			1

Renal insufficiency has long been associated with poor outcomes in cardiovascular diseases. In patients with AMI, The risk of death in patients with raised BUN levels is not explained only by renal function-either initially impaired or reduced as a result of hypoperfusion but has some other explanation. This may be due to increase age, large body mass, increase catabolic rate, haemolysis, pre-renal & renal causes **[21]**.According to Harita′s study, low serum creatinine levels are associated with higher survival rates. A higher risk of T2D was associated with it. Because skeletal muscle is one of the target tissues for insulin, skeletal muscle mass might be associated with T2D. Serum creatinine is a possible surrogate marker of skeletal muscle mass **[22]**. BUN reflects not only renal function but also neuro-hormonal activation. One study confirmed that high level of BUN was a powerful indicator for in-hospital mortality in patients with AMI. In another study Aronson and BUN was strongly associated with long-term mortality in ST-elevation MI. Albumin, a main component of plasma proteins, plays a key role in maintaining vascular osmotic pressure, transporting endogenous and exogenous compounds, and regulating pharmacokinetics of drugs. Hypoalbuminemia predicts a poorer outcome in HF, stroke, and CAD. Zhao found that a decrease in albumin at admission might be an independent predictor of long-term mortality in discharged

patients with AMI .The present study aimed to explore the prognostic value of blood urea nitrogen and creatinine ratio in predicting the long-term mortality **[11, 22]** .

Table 6 demonstrates Correlation between criteria of liver functions (GPT,GOT,ALP and TP) in the study groups .The results of the statistical analysis of person's correlation test refers to a significant positive correlation relationship for GPT and GOT enzyme levels in the PCI Patients group. On the other hand, it is noted in the absence significant correlation between other parameters. Also the present study has recorded a positive insignificant correlation between GOT and GPT (r=0.522 at p=0.005), in contrast exhibited a negative correlation between parameters in the control group.

Table 6: Correlationships of Criteria for Assessing Liver Functions in TheSera of PCI Patients Group

	Parameters	GPT(U/L)	GOT(U/L)	ALP(U/L)	TP(mg/dL)
r p	GPT(U/L)	1	0.540** 0.000	-0.092 0.514	-0.137 0.327
	GOT(U/L)		1	0.007 0.962	0.084 0.549
	ALP(U/L)			1	0.042 0.745
	TP(mg/dL)				1
Correlationships of Criteria for Assessing Liver Function in The Sera of Controls Group					
	Parameters	**GPT(U/L)**	**GOT(U/L)**	**ALP(U/L)**	**TP(mg/dL)**
r p	GPT(U/L)	1	0.522** 0.005	-0.198 0.322	0.311 0.114
	GOT(U/L)		1	-0.228 0.253	-0.70 0.728
	ALP(U/L)			1	0.169 0.398
	TP(mg/dL)				1

The parameters GPT, GOT and ALP are used as a predictive marker not only for liver disorders but also probably when there are signs indicative of a defect in the myocardium. AST or GOT is found in the liver, heart (cardiomyopathy), skeletal muscle, kidneys, brain and blood **[23]** .GPT cells are primarily distributed in the liver, and GPT is closely associated with endothelial dysfunction caused by atherosclerosis and inflammation **[24]**. Where it was found that the levels of these enzymes are directly proportional to the number of dead cells in the damaged myocardium tissue, where the myocardium contains a number of enzymes and proteins that once these tissues are damaged **[25]** ,these enzymes are released into the extracellular fluid into the blood, especially the GOT at GPT due to degeneration and necrosis of myocardium cells causes a series of metabolic disturbances in addition to the breakdown of aerobic glucose, which made it an excellent biomarker for measuring myocardial performance after infarction **[26]**.

Conclusion:

Cardiovascular complications are directly associated with combination of risk factors which are; hyperlipidemia, hyperglycemia, hypertension, and obesity. Lipids profiles,

Urea, Creatinine, Urea/Creatinine ratio Glucose, Insulin, HOMA-IR, and HbA1C%, as well as Liver functions tests are good prognostic indicators for predicting complications of T2D that cause MI.

References

[1]X. Zhao, D. Wang, and L. Qin, "Lipid profile and prognosis in patients with coronary heart disease: a meta-analysis of prospective cohort studies," *BMC Cardiovascular Disorders*, vol. 21, no. 1. 2021. doi: 10.1186/s12872-020-01835-0.

[2]N. Waheed *et al.*, "Sex differences in non-obstructive coronary artery disease," *Cardiovasc. Res.*, vol. 116, no. 4, pp. 829–840, 2020, doi: 10.1093/cvr/cvaa001.

[3]D. P. Kelley, A. Chaichi, A. Duplooy, D. Singh, M. R. Gartia, and J. Francis, "Labelfree mapping and profiling of altered lipid homeostasis in the rat hippocampus after traumatic stress: Role of oxidative homeostasis," *Neurobiol. Stress*, vol. 20, no. April, p. 100476, 2022, doi: 10.1016/j.ynstr.2022.100476.

[4]B. Fletcher *et al.*, "Managing abnormal blood lipids: A collaborative approach," *Circulation*, vol. 112, no. 20, pp. 3184–3209, 2005, doi: 10.1161/CIRCULATIONAHA.105.169180.

[5]A. Wolska, M. Reimund, D. O. Sviridov, M. J. Amar, and A. T. Remaley, "Apolipoprotein mimetic peptides: Potential new therapies for cardiovascular diseases," *Cells*, vol. 10, no. 3, pp. 1–19, 2021, doi: 10.3390/cells10030597.

[6]P. H. G. Sanches, A. A. R. Silva, and A. M. Porcari, "Plasma lipid profiles differ among chronic inflammatory diseases," *EBioMedicine*, vol. 70, pp. 10–11, 2021, doi: 10.1016/j.ebiom.2021.103526.

[7]Q. G. Karwi, Q. Sun, and G. D. Lopaschuk, "The contribution of cardiac fatty acid oxidation to diabetic cardiomyopathy severity," *Cells*, vol. 10, no. 11, pp. 1–18, 2021, doi: 10.3390/cells10113259.

[8]P. Kanikarla Marie *et al.*, "The Provocative Roles of Platelets in Liver Disease and Cancer," *Front. Oncol.*, vol. 11, no. July, pp. 1–22, 2021, doi: 10.3389/fonc.2021.643815.

[9]O. A. (2020 Oyawaluja, A.A. ; Oiseoghaede, J.O. ; Ndu, C.C. ; Odukoya, "NJP.pdf."

[10] J. H. Salazar, "Overview of urea and creatinine," *Lab Med.*, vol. 45, no. 1, pp. e19–e20, 2014, doi: 10.1309/LM920SBNZPJRJGUT.

[11] M. Wang, Y. Li, S. Li, and J. Lv, "Endothelial Dysfunction and Diabetic Cardiomyopathy," *Front. Endocrinol. (Lausanne).*, vol. 13, no. April, pp. 1–12, 2022, doi: 10.3389/fendo.2022.851941.

[12] L. Liu, X. Liu, X. Ding, H. Chen, and H. Li, "Body Mass Index and New-Onset

Atrial Fibrillation in Patients with Acute Myocardial Infarction," *Int. J. Gen. Med.*, vol. Volume 15, pp. 5717–5728, 2022, doi: 10.2147/ijgm.s367868.

[13] A. Tas, "Description of the characteristics , epidemiology , diagnosis , and risk factors of presbycusis disorder," vol. 2, no. 2, pp. 46–56, 2022.

[14] M. S. Kuchay, N. S. Choudhary, and S. K. Mishra, "Pathophysiological mechanisms underlying MAFLD," *Diabetes Metab. Syndr. Clin. Res. Rev.*, vol. 14, no. 6, pp. 1875–1887, 2020, doi: 10.1016/j.dsx.2020.09.026.

[15] J. A. M. J. L. Janssen, "Hyperinsulinemia and its pivotal role in aging, obesity, type 2 diabetes, cardiovascular disease and cancer," *Int. J. Mol. Sci.*, vol. 22, no. 15, 2021, doi: 10.3390/ijms22157797.

[16] I. Ahn *et al.*, "CardioNet: a manually curated database for artificial intelligence-based research on cardiovascular diseases," *BMC Med. Inform. Decis. Mak.*, vol. 21, no. 1, pp. 1–15, 2021, doi: 10.1186/s12911-021-01392-2.

[17] L. Nibali, N. Gkranias, G. Mainas, and A. Di, "Periodontitis and implant complications in diabetes," pp. 1–18, 2022, doi: 10.1111/prd.12451.

[18] S. Salaramoli, S. Mehri, H. Hosseinzadeh, and S. I. Hashemy, "The effects of ginger and its constituents in the prevention of metabolic syndrome : A review".

[19] J. Kesavadev *et al.*, "Blood glucose levels should be considered as a new vital sign indicative of prognosis during hospitalization," *Diabetes Metab. Syndr. Clin. Res. Rev.*, vol. 15, no. 1, pp. 221–227, 2021, doi: 10.1016/j.dsx.2020.12.032.

[20] M. Kaldirim *et al.*, "Modulation of mTOR Signaling in Cardiovascular Disease to Target Acute and Chronic Inflammation.," *Front. Cardiovasc. Med.*, vol. 9, no. June, p. 907348, 2022, doi: 10.3389/fcvm.2022.907348.

[21] N. Harita *et al.*, "Lower serum creatinine is a new risk factor of type 2 diabetes: The kansai healthcare study," *Diabetes Care*, vol. 32, no. 3, pp. 424–426, 2009, doi: 10.2337/dc08-1265.

[22] D. Zhao *et al.*, "Predictive Value of Blood Urea Nitrogen to Albumin Ratio in Long-Term Mortality in Intensive Care Unit Patients with Acute Myocardial Infarction: A Propensity Score Matching Analysis," *Int. J. Gen. Med.*, vol. 15, no. March, pp. 2247–2259, 2022, doi: 10.2147/IJGM.S349722.

[23] E. E. Jabbar and W. T. Mahdi, "An enzymatic and biochemical study of patients with beta thalassemia," *Int. J. Health Sci. (Qassim).*, vol. 6, no. March, pp. 11611–11628, 2022, doi: 10.53730/ijhs.v6ns1.7849.

[24] G. Ndrepepa and A. Kastrati, "Alanine aminotransferase—a marker of cardiovascular risk at high and low activity levels," *J. Lab. Precis. Med.*, vol. 4, pp.

29–29, 2019, doi: 10.21037/jlpm.2019.08.01.

[25] W. M. Merza, Z. H. Abdul-Qahar, and Z. Alsharifi, "Transaminase and creatinine predict diastolic and systolic dysfunction in patients with myocardial infarction," *Int. J. Pharm. Qual. Assur.*, vol. 10, no. 1, pp. 178–185, 2019, doi: 10.25258/ijpqa.10.1.30.

[26] D. S. Uthirapathy, "Cardioprotection effects of diosgenin from Dioscorea bulbifera against isoproterenol-induced myocardial infarction," *Drugs Cell Ther. Hematol.*, vol. 10, no. 1, pp. 887–896, 2021, [Online]. Available: https://www.dcth.org/index.php/journal/article/view/182