**Ministry of Higher Education** 



and Scientific Research

## **Journal of Kufa for Chemical Sciences**

A refereed

### **Research Journal Chemical Sciences**

Vol.2 No.9

Year 2022

ISSN 2077-2351

#### Study of Lipid Profiles Levels and Some Parameters of Glucose Monitoring As Predictive Indicators of Recurrent MI in Patients Undergoing Elective Percutaneous Coronary Intervention (PCI)

#### Rasha Hasan Jasim and Ahssan Ali Lefta

Department of Chemistry-Factually of Education for Girls-University of Kufa-Iraq e-mail: rasha.alfahham@uokufa.edu.iq

#### **Abstract**

**Background :** Coronary heart disease (CHD) also known as coronary artery disease (CAD) or ischemic heart disease (IHD), where it is caused by hindrance of blood transit to muscle of heart cause to collect the lipid and cholesterol on the inner wall of the heart. Because of atherosclerosis, CAD contains many types of disease: stable angina, unstable angina, myocardial infarction (MI), and sudden cardiac death. Percutaneous Coronary Intervention (PCI) is known as angioplasty is a non-surgical operation performed using a catheter to cram either inflate a balloon in the narrowing place or a stent (a very small fin grid tube made of either plastic or metal of stainless steel). So it is covered with medicine to prevent the coronary artery blockage again), to expand or open up the vessels in the muscle of the heart that contain plaque buildup that cause stenosis. Lipid profile is main factor of danger to CVD; each formed of lipid profiles is linked with rise in happening of coronary artery disease (CAD). Insulin is a protein hormone that regulates blood glucose levels, energy metabolism and anabolic. Glycosylated hemoglobin (HbA1c%) is a type of hemoglobin that is generally used to determine the average glucose values of blood that attached to hemoglobin of an individual according to the past 12 weeks that is red blood cells age for also the resulting values determine the level of blood sugar and aid in the diagnosis of diabetes mellitus. Subjects: One hundred individuals were collected to contribute to 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 elective PCI, were divided into two subgroups; 30 patients with the first PCI and 40 patients who underwent more than one PCI. The second group involved 30 healthy individuals (with the age range 30-55 years) who were enrolled in the present study as a control group. Where spectral methods, ELISA and some mathematical equations were used to find the results of the current study. Results: Using the statistical program SPSS to find the statistical indications. The results showed significant increases (p=0.000) for each of TG, Cholesterol, LDL, vLDL, glucose, insulin, HbA1c% and HOMA-IR, while a significant decrease (p=0.000) in HDL levels was recorded when the two study groups were compared together. Therefore, it is necessary to use the HOMA-IR analysis as a basic parameter to identify and determine the incidence of coronary heart disease

**Keywords:** Insulin, Glucose, CVD, CHD, PCI, HbA1c%, HAMA-IR & Lipid profile.

#### **Introduction**

**Coronary heart disease (CHD)** so known as coronary artery disease (CAD) or ischemic heart disease (IHD) [*Virani*,,2021], where is result from hindrance of blood transit to muscle of heart cause to collect the lipid and cholesterol on the inner wall of the heart. Because of atherosclerosis, CAD contains many types of disease: stable angina, unstable angina, myocardial infarction, and sudden cardiac death [*Dayana*, 2021]. **Percutaneous** 

**Coronary Intervention** (**PCI**) is known as angioplasty, is a non-surgical operation performed using a catheter (a tube that is flexible and thin) to cram either inflate a balloon in the narrowing place or a stent (a very small and fin grid tube that is made of either plastic or metal or stainless steel). So it is covered with medicine to prevent the coronary artery blockage again), to expand or open up the vessels in the muscle of the heart that contain plaque buildup that cause stenosis [*Debra & Brian, 2018; Mansoor, et al., 2021*]. The physician accesses the damaged arteries through a small incision in the thigh or arm, where the catheter is inserted through this opening, then delivered to the ascending aorta. Visualize the sites of damage in the arteries by using X-ray fluoroscopy after coronary arteries are injection by IV contrast (Radiopaque dye is a colorless liquid and it's based on iodine) to determine the damage site [*Manda & Baradhi, 2021*].

**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 [*Tareq, et al., 2020*].

**Lipid profiles** are the main factors of danger to CVD; each formed of lipid profiles is linked with a rise in the happening of coronary artery disease (CAD). Where increase total cholesterol, triglycerides, low-density lipoprotein (LDL) levels, and decrease high-density lipoprotein (HDL) levels in serum [*Laltesh, et. al.,2018*]. TG: The sources of TG are either animal, which are saturated and solid fats, or plant. TG are base lipids transferred to the blood stream with cholesterol. It is considered the main energy source that is utilized and stocked in the body. There are two

pathways to the synthesis of TG in both liver and adipose tissue [*Emine, et al., 2021*]. Hypertriglyceridemia is considered elevated in TG or TG, chylomicrons and vLDL, that caused by high anabolic and low catabolism [*Charlotte, 2020*].

**Cholesterol** is formed by substantially each tissue, though firstly *via* the liver, adrenal cortex, reproductive tissues and the intestine. Cholesterol is created in the cytoplasm, and can get rid of cholesterol *via* converting it into bile salts, or via excretion into the bile; bacteria can lessen cholesterol to cholesterol and coprostanol, both cholesterol generating neutral fecal sterols. The regulation of the cholesterol levels in the body is carried out by the liver, where it can be synthesized by de novo pathway or cholesterol from a many of sources like food, also excreted through the liver into bile or transformed into bile salts, which are secreted to the intestines. Cholesterol is deposited on the inner of the vessels due to the imbalance of the inside of it with the penetration. [ Emine, et al., 2021]. There are two kinds of cholesterol, one of which is salutary and the other is harmful to health if it increases [Charlotte, 2020]. A healthy cholesterol level in adult humans is dependent on other factors like gender, age, race, obesity, family history, smoking, physical inactivity, diabetes and high blood pressure [Małgorzata, et al., 2020]. The cholesterol level in healthy human plasma is less than 200 mg/dL. Lipids are created in the liver and then transferred to the plasma by the intestines in the form of complex molecules known as lipoproteins. Lipoproteins are spherical bodies with neutral lipids (nonpolar) that are triglycerides and cholesterol esters in the center, while on the surface polar lipids are cholesterol and phospholipid molecules [Nader, 2018]. Lipoprotein units types are chylomicrons (CM), high-density lipoproteins

(HDL), low-density lipoproteins (LDL), and very-low-density lipoproteins (vLDL); this variation causes lipid and protein ratio, density, size and origin site. The function of lipoproteins is both to save their section of lipids soluble as they transport them in the plasma, and to transport an effective mechanism for moving their lipid contents to or from the tissues [Denise, 2017]. vLDL is formed by nearly 60% triacyglycerol and its job is to transport TG from the liver to the tissues of the peripheral. There, the TG is degraded by lipoprotein lipase (LPL); vLDL are excreted into the stream of blood *via* the liver as nascent units include apo-B-100. They must get apo CII and E from HDL circulate. vLDL in the circulation, TAG dissolution via LPL, affecting the vLDL to reduce in the size and convert to more density. The components of surface components, counting the C and E apolipoproteins, are regained to HDL, but the units keep apo. B-100. With these alterations, vLDL is transformed to LDL in plasma. The reference value of vLDL is 2 - 38 mg/dL. [*Nader*, 2018]. LDL particles comprise high cholesterol and cholesteryl esters concentrations nearly cholesterol of plasma 70% is in LDL, while fewer TAG than their vLDL. The main function of LDL is to deliver cholesterol to the tissues of peripheral tissues [Denise, 2017]. The reference value of LDL below 100 mg/dL. [Nader, 2018]. Particles of HDL are made in the blood by the count of apo-A-1, an apolipoprotein complete and secreted by the intestine and liver. HDL of Nascent comprises mostly phospholipids (largely PC) and apo E, A, and C. They move cholesterol to the liver from tissues and form as cholesteryl esters [Emine, et. al., 2021].

**Insulin** is a protein hormone that regulates blood glucose levels, energy metabolism and anabolic. When the sugar level rises insulin is secreted into

the blood circulation by beta-cells located in the Langerhans islets in the pancreas. In the cell membrane, insulin receptors are activated that increase the transport of glucose into the intercellular to reduce its level in the extracellular [Vargas, et al., 2022]. The Golgi apparatus in  $\beta$ -cells receives proinsulin, where cleaved to active form, and C-peptide and active form sent to the granules in the cytosol where it is stored. Insulinase is an enzyme in the liver that is released to degrade insulin [Ming, et al., 2018; Emine, et al., 2021]. Insulin resistance is associated with many serious medical conditions such as type 2 diabetes mellitus, hypertension, atherosclerosis, and metabolic syndrome. Insulin resistance is the impact of insulin to reduce the blood glucose level because of a defect in insulin receptors on target cells; thus the sugar levels remain high with a rise or normal insulin levels [Zaman, 2019; Emanuel, et al., 2021]. Glucose is a major source of energy for cells body, especially for humans and animals [Fernandes, 2021]. The normal level of glucose in the blood of an adult is 60 to 110 mg/dL (3.3-6.1 mmol/L). When the level of blood sugar rises above 126 mg/Dl, there may be diabetes; this elevation may cause damage to vessels of blood, cells and nerves [Mouri & Badireddy, 2021; American Diabetes, 2022].

**Glycosylated hemoglobin percentage (HbA1c%)** is a type of hemoglobin that is generally used to determine the average glucose values of blood that attached to hemoglobin of an individual according to the past 12 weeks that is red blood cells age for also the resulting values determine the level of blood sugar and aid in the diagnosis (diabetes mellitus) [*Huang, et. al.,2021*]. Glycosylated hemoglobin is created via non-enzymatic glycation of the  $\beta$ -chain of hemoglobin A by the glucose of plasma; the glycation is synthesis continuously over the life of red blood cells and its creation

irreversibly. World healthy defined the assessment of HbA1c% high 6.5% as investigative of DM and (5.7- 6.4)% as pre-diabetes. [*Bo, et. al., 2021*].

#### **Subjects and Methods**

A hundred samples were selected 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 who underwent PCI on condition that they take clopidogrel therapy as a result of the diagnoses made by doctors in cardiology, they were divided to two subgroups (30 patients with one PCI and 40 patients with more than one underwent PCI. While the second involved 30 healthy individuals with the age range(30- 55 years). A subjective perception of good health as determined by a 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 determine HbA1c% level. (2) Three mL were placed in a plane tube at laboratory room temperature for measuring glucose and insulin, TC, TG, HDL, LDL, and vLDL levels. The samples were left to coagulate at laboratory temperature, analyzers then separated using a centrifuge at 3000 xg for 15 minutes.

#### **Result and Discussion**

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

In **Table 2**, the statistical results showed an increase (p=0.000) of all parameters in the two patients subgroups (those who carried out to the elective PCI for the first time and the others who underwent to this strategy at least for twice) compared to healthy individuals, while the results of the comparison of the two disease groups did not illustrated significant differentiation for lipid profiles and glucose, on the other side significant increase of insulin (p=0.008), HbA1c% (p=0.013), and HOMA-IR (p=0.002) were noted in the second patients subgroup comparison to the first.

When the three study subgroups were subdivided into six mini subgroups according to the sexes (**Table 3**). Outcomes of the statistical analysis demonstrated significant increases (p<0.005) in each of TG , Cholesterol, vLDL, and LDL, but are not in other parameters that evaluated in the present work, when the comparison between genders in the same subgroup was done (patients who underwent to the first elective PCI). While the comparison between sexes in the second subgroup (patients who underwent to two PCI at least) was illustrated a significantly raise in the female samples (p<0.005) for each of TG , Cholesterol and vLDL, while the results were not recorded acceptable changes when the other parameters were measured. In healthy individuals group, there were not significant variations between the males and females at all parameters were evaluated.

When the comparing was carried out among the two subgroups of patient females as well as healthy female subgroup, a significant increase (p < 0.005) was observed in each of the lipid profiles, glucose, HbA1c%, insulin, and HOMA-IR in the females patients subgroups comparison to healthy. Exception of the results of glucose, the results of the comparison among the males in the two patients subgroups and subgroup of healthy males were similar to those in the females subgroups.

One of the main risk factors for coronary heart disease is dyslipidemia, which results from an increase in cholesterol, TG and LDL levels and a decrease in the level of HDL in the serum of patients with CAD [*Hedayatnia*, 2020]. The effect of the lipid profiles on the work of the coronary arteries depends mainly on some mechanisms of the physiological functions of the lipid profiles. The origin of the development of atherosclerosis is the accumulation of LDL on the inner wall of the vessels,

where lipoprotein of atherogenic that include apo-B (LDL), is support collection of cholesterol in macrophages and responses of inflammatory inner well of the vessel, the transfer of LDL from the blood to the vessels walls results start of lipid deposition. Where the deposited lipids either return to the bloodstream and thus reduce the lipids deposited on the vessels and reduce the development of the lesion or oxidize through the activity of free radicals or Leukocytes, or become foam cells after being devoured by macrophages\monocyte [*Ferrari, et al., 2016*].

ApoA-I main apolipoprotein of (HDL), It usually carries 20% of all the cholesterol in the blood, moving extra cholesterol to the liver from the foam macrophages of arterial walls is largely facilitated through adenosine triphosphate-binding cassette transporters (ABC) and by lecithin-cholesterol acyltransferase (LCAT) assistances transform the cholesterol esterification into complete HDL2 and HDL3 units after the pass of cholesterol from peripheral cells through adenosine triphosphate-binding cassette transporters (ABC). Transfer protein of cholesterol ester (CETP) transmissions to HDL cholesterol esters of additional apolipoprotein B- inclosing units, then, HDL units are stuck via hepatocytes through scavenger receptor B1 (SR-B1); so, the study association between the hazard for atherosclerosis and low levels of HDL [*Maha, et al., 2017*]. The results of the current study are in agreement with previous studies [*Hassan, et al., 2013; Maha, et al., 2017; Laltesh, & Ajay, 2018*].

The high level of glucose in the blood is one of the main causes of atherosclerosis, which results in cardiovascular disease; it plays an essential role in the dysfunction of endothelial pathogenesis. Additionally, high glucose levels produce damage to the metabolic environment via inflammation of chronic inflammation, pro-coagulability, oxidative stress, and other abnormalities which alter vascular structure [*Shuai, et al.,2021; Paolisso, et al.,2021*]. The results of the current study match a number of previous studies by researchers including [*Metwally, et al., 2020; Çetin , et al., 2021; Suzanne, et al., 2021*]. So the results of the current study are similar to previous studies that dealt with measuring the levels of HbA1C% % in patients with heart disease [*Ul-Haque, et al., 2019; Yildiz & Aclan, 2021; Abu Tailakh, 2021*].

Hyperinsulinemia is the elevation in the insulin level than normal, It's the state that categorized by injured myocardial insulin signaling, dysfunction of mitochondrial function, different homeostasis of calcium, uneven coronary microcirculation, endoplasmic stress of reticulum, dysfunction sympathetic of nervous system, and other. These pathophysiological modifications product in enlarged fibrosis, oxidative stress, hypertrophy, dysfunction of diastolic cardiac, and ultimate systolic heart failure [Dominic, et al., 2019]. The functions of the endothelium (is contain the inner lining of the vessels) are affected by increased insulin and its result among which endothelium dysfunction such as cheap nitric oxide (NO) bioavailability, enlarged oxidative stress via raised production of reactive oxygen species (ROS), abnormal vasoreactivity and the create of pro-thrombotic and pro-inflammatory factors. This state may influence proliferation liability to hypertension, atherosclerosis, and coronary heart disease [Rahman, et al., 2021]. This study gives results that are close to previous studies [Metwally, et al., 2020; Suzanne, et. al., 2021]. Cardiovascular disease is affected by high insulin resistance, as it works on high pressure, and these diseases work on a similar pathomechanical. In specific, they make an imperfection of renin-angiotensin and sympathetic nervous systems that effects in the higher stimulation receptors of both angiotensin II and adrenergic, a decrease of  $NO_2$  with dysfunction of endothelial, the improvement of oxidative stress, and the creation of inflammatory cytokines [*Fiordelisi, et. al.,2019; Teresa, et al., 2021*].

	The Stud			
	Patients (70)	Controls (30)	p-value	
Test	Mean ± S.D.	Mean ± S.D.		
	Min Max.	Min Max.		
	Range	Range		
	241.556 ± 44.727	98.327 ± 10.436		
TG (mg/dL)	128-345	75-116.8		
-	217	41.8		
	$268.560 \pm 43.871$	$115.695 \pm 11.845$		
Cholesterol (mg/dL)	151.7-361.6	96.13-138.53	0.000	
-	209.9	42.4		
	$29.501 \pm 9.582$	$50.773 \pm 2.636$		
HDL (mg/dL)	7.4-52	44.5-56.5	0.000	
	44.6	12		
	48.439± 8.975	$19.664 \pm 2.088$		
vLDL (mg/dL)	25.6-69 15-23.36		0.000	
	43.4	8.36		
	190.602 ± 38.545	44.957 ± 11.244		
LDL (mg/dL)	92-273.5 28.1-63.15		0.000	
	181.5	35.05		
	154.096 ±73.065	$87.600 \pm 15.732$		
Glucose (mg/dL)	69-381	60.6-120	0.000	
	312	59.4		
	$7.919 \pm 2.252$	$5.297 \pm 0.781$		
HbA1c%	4.15-13.88	4.15-13.88 3.79-6.99		
	9.72	3.21		
	$21.681 \pm 6.427$	$12.560 \pm 2.416$		
Insulin (mIU/L)	13.89-68.82	8.98-17.93	0.000	
	54.93	8.95		
	8.275 ± 4.585	$2.747 \pm 0.726$		
HOMA-IR	2.9-27.6	1.5-4.3	0.000	
	247	2.8		

 Table 1: Levels (Mean±S.D.) of Lipid Profiles, Glucose, Insulin and HbA1c% levels

 in The Sera of The Study Individuals

The Mean Difference is Significant at 0.05 Level

# Table 2: Levels (Mean ± S.D.) of Lipid Profiles, Glucose, HbA1c% and Insulin inSera of Patient with One PCI, Patient with More PCI and Healthy Groups

Parameters	Patients with One PCI 30	Patients with More PCI 40	Controls 30	p-value	
	Mean±S.D.	Mean±S.D.	Mean±S.D.		_
					6

	Min-Max	Min-Max	Min-Max	
	Range	Range	Range	
$TC(m_{\sigma}/H)$	239.631±44.335	$243.000 \pm 45.528$	98.327±10.436	0.715 for 1 vs 2
IG (mg/aL)	128-322.8	177.40-345.00	75.00-116.80	0.000 for 1 vs 3
	194.8	167.60	41.80	0.000 for 2 vs 3
	262.991±46.882	272.736±41.582	115.695±11.845	0.283 for 1 vs 2
Cholesterol (mg/dL)	151.7-361.6	201.60-354.70	96.13-138.53	0.000 for 1 vs 3
	209.9	153.10	42.40	0.000 for 2 vs 3
	28.763±11.092	30.055±8.379	50.773±2.636	0.515 for 1 vs 2
HDL (mg/dL)	8.8-52	7.4-43.3	44.5-56.5	0.000 for 1 vs 3
	43.2	35.9	12.00	0.000 for 2 vs 3
	47.925±8.867	48.824±9.148	19.664±2.088	0.628 for 1 vs 2
VLDL (mg/dL)	25.60-64.56	35.48-69	15-23.36	0.000 for 1 vs 3
	38.96	33.52	8.36	0.000 for 2 vs 3
IDI (mg/dI)	186.252±42.259	193.865±35.710	44.957±11.244	0.341 for 1 vs 2
LDL (llig/aL)	92-264.1	131.87-273.50	28.1-63.15	0.000 for 1 vs 3
	172.1	141.63	35.05	0.000 for 2 vs 3
	138.280±57.858	165.957±81.357	87.600 ±15.732	0.064 for 1 vs 2
Glucose (mg/dL)	69-280.6	70.70-381.00	60.6-120	0.002 for 1 vs 3
-	211.6	310.30	59.4	0.000 for 2 vs 3
	7.259±1.675	8.414±2.510	5.297±0.781	0.013 for 1 vs 2
HbA1c%	5.1-11.46	4.15-13.88	3.79-6.99	0.000 for 1 vs 3
	6.36	9.72	3.21	0.000 for 2 vs 3
	19.669±3.209	23.190±7.744	12.560±2.416	0.008 for 1 vs 2
Insulin (mU/L)	13.89-25.57	17.19-68.82	8.98-17.93	0.000 for 1 vs 3
	11.68	51.63	8.95	0.000 for 2 vs 3
	6.684±2.849	9.467±5.267	2.746±0.726	0.002 for 1 vs 2
HOMA-IR	2.9-12.11	3.9-27.6	1.5-4.3	0.000 for 1 vs 3
	9.21	23.70	2.8	0.000 for 2 vs 3

1: One Elective PCI Patients, 2: More Elective PCI Patients, 3: Healthy. The Mean

Difference is Significant at 0.05 Level

Table 3: Levels (Mean ± S.D.) of Lipid Profiles, Glucose, HbA1c% and Insulin in Sera of Patient with One PCI, Patient with at least two PCI and Healthy Groups

	Subjects (N)						
	Patients with One PCI 30		Patients with More than One PCI 40		Controls 30		
Parameter s	Females-1 (13)	Males-2 (17)	Females- 3 (13)	Males-4 (27)	Females- 5 (13)	Males-6 (17)	p-value
	Mean ± S.D. Min-Max Range	Mean ± S.D. Min-Max Range	Mean ± S.D. Min-Max Range	Mean ± S.D. Min-Max Range	Mean ± S.D. Min-Max Range	Mean ± S.D. Min-Max Range	
TG (mg/dL)	219.36±40.93 128-289.4 161.4	255.12±41.43 188.8-322.8 134	267.64±43 212.7-345 132.3	231.13±42.49 177.4-320.4 143	95.85±10. 09 81.96- 16.8 34.84	100.21±10.59 75-112.9 37.9	<0.05 FOR: 1vs2, 3vs4, 1vs3, 1vs5, 2vs4, 2vs6, 3vs5, and 4vs6
Cholesterol (mg/dL)	247.51±40.87 151.7-300.2 148.5	274.82±48.85 9 200.45-361.6 161.15	292.95±37 .89 231.6- 354.7 123.1	263±40.35 201.6-341.5 139.9	115.97± 10.85 101.06- 138.53 37.470	115.485±12.8 96.13-135.32 39.19	<0.05 FOR: 1vs2, 3vs4, 1vs3, 1vs5, 2vs6, 3vs5, and 4vs6
HDL (mg/dL)	32.7±12.04 9.7-52 42.3	25.747±9.591 8.8-40 31.2	32.96±8.0 8 19.9- 43.3 23.4	28.65±8.3 7.4-42 34.6	51.63±3.1 4 45.1- 56.5 11.4	50.11±2.02 44.5-54.3 9.8	<0.05 FOR: 1vs2, 1vs5, 2vs6, 3vs5, and 4vs6

vLDL (mg/dL)	43.87±8.18 25.6-57.88 32.28	51.02±8.28 37.76-64.56 26.8	54.22±8.2 9 42.54- 69 26.46	46.22±8.49 35.48-64.08 28.6	19.17±2.0 1 16.39- 23.36 6.97	20.04±2.12 15-22.58 7.58	<0.05 FOR: 1vs2, 3vs4, 1vs3, 1vs5, 2vs4, 2vs6, 3vs5, and 4vs6
LDL (mg/dL)	170.88±39.25 9 92-235.15 143.15	198±41.74 118.77-264.1 145.33	205.79±34 .07 149.76- 67.6 117.84	188.12±35.65 131.87-273.5 141.63	44.47±10. 67 28.1- 62.93 34.83	45.32±11.97 28.28-63.15 34.87	<0.05 FOR: 1vs2, 1vs3, 1vs5, 2vs6, 3vs5, and 4vs6
Glucose (mg/dL)	152.91±70.13 69-280.6 211.6	127.08±45.50 5 75.6-232.3 156.7	178.29±78 .8 82- 336.4 254.4	160.01±83.36 70.7-381 310.3	82.67±17. 06 60.6- 109.0 48.4	91.36±13.98 72.2-120 47.8	<0.05 FOR: 1vs5, 3vs5, and 4vs6
HbA1c%	7.465±1.886 5.14-11.46 6.31	7.102±1.535 5.10-9.95 4.85	8.68±2.33 5.36- 13.57 8.21	8.28±2.62 4.15-13.88 9.72	5.624±0.7 29 4.50-6.99 4.49	5.046±0.744 3.79-6.83 3.04	<0.05 FOR: 1vs5, 2vs4, 2vs6, 3vs5, and 4vs6
Insulin (mU/L)	18.10±3.04 13.89-23.60 9.71	20.86±2.86 16.80-5.57 8.77	22.8±2.41 19.56- 26.8 7.24	23.37±9.33 17.19-8.82 51.63	13.29±2.5 1 8.98- 16.95 7.97	12.00±2.24 9.06-17.93 8.87	<0.05 FOR: 1vs3, 1vs5, 2vs6, 3vs5, and 4vs6
HOMA-IR	6.68 ± 2.90 2.93-12.12 9.19	6.69±2.89 3.27-11.95 8.68	9.96±4.18 4.00- 17.88 13.88	9.21±5.78 3.92-27.61 23.69	3.56±0.68 2.48-4.83 2.35	4.08±0.86 2.74-5.85 3.11	<0.05 FOR: 1vs3, 1vs5, 2vs4, 2vs6, 3vs5, and 4vs6

1: One Elective PCI Female Patients, 2: One Elective PCI Male Patients, 3: More Elective PCI Female Patients, 4: More than one Elective PCI Male Patients 5: Healthy Female Control, and 6: Healthy Male Control. The Mean Difference is Significant at 0.05 Level

#### **References**

- (1) Abu Tailakh M., Ishay S., Awesat J., Poupko L., Sahar G., ovack, V., (2021)," Hemoglobin A1c in Patients with Diabetes Predict Long-Term Mortality Following Coronary Artery Surgery", J. Clin. Med., 10 (2739). <u>https://doi.org/10.3390/jcm10122739</u>
- (2) American Diabetes Association; Standards of Medical Care in Diabetes—2022 Abridged for Primary Care Providers. Clin Diabetes 1 January 2022; 40 (1), p: 10– 38. <u>https://doi.org/10.2337/cd22-as01</u>.
- (3) Bo Z., Bingjie Z., Zhulin Z., Yutong G., &Dan W., (2021), "The value of glycosylated hemoglobin in the diagnosis of diabetic retinopathy: a systematic review and Meta-analysis", BMC Endocrine Disorders, 21(82), p:1-11. https://doi.org/10.1186/s12902-021-00737-2
- (4) Çetin Ş. S., Nar G., Şen G., Günver M.G., Şanlıalp M., (2021), "High Triglyceride Glucose Index Does Not Show the Presence and the Severity of Coronary Artery Disease: A Single-Center Study", EJCM, 9(2), p:76-82. DOI: 10.32596/ejcm.galenos.2021-01-03.
- (5) Charlotte E., (2020) "the Cell Membrane", Teach Me Physiolog.

- (6) Dayana E., (2021), "Coronary Artery Disease", MountSinai, Icahn School of Medicine Mount Sinai.
- (7) Debra S., & Brian K., (2018), "Stent: Why and How They Are Used" Healthline Medai.
- (8) Denise R. F.,(2017), "Lippincott Illustrated Reviews: Biochemistry" 7th Edition, Printed in China
- (9) Dominic S., & Paul M. T.,(2019), "Resolving the Paradox of Hepatic Insulin Resistance", Cellular and Molecular Gastroenterology and Hepatology, 7(2), p:447-456. <u>https://doi.org/10.1016/j.jcmgh.2018.10.016</u>.
- (10) Emanuel F., Josefin O., Karin M., Lena S., Martin S., Helen B., Li-Ming G., Silvano P., Alessandro P., Barbara B., Lars L., Giovanni S., & Per-Anders J., (2021), "Hyperinsulinemia and insulin resistance in the obese may develop as part of a homeostatic response to elevated free fatty acids: A mechanistic case-control and a population-based cohort study", E Bio Medicine, 65 (103264), p:1-15. DOI:https://doi.org/10.1016/j.ebiom. 2021.103264.
- (11) Emine E. A., Susan D. C., David S. F., & Susan M. V., (2021), "Lippincott's Illustrated Reviews: Biochemistry", Eighth Edition, printed in China.
- (12) Fernandes R., (2021), "The controversial role of glucose in the diabetic kidney", Porto Biomedical Journal, 6 (1), p: e113. doi: 10.1097/j.pbj.00000000000113.
- (13) Fiordelisi A., Iaccarino G., Morisco C., Coscioni E., Sorriento D., (2019), "NFkappaB Is a Key Player in the Crosstalk Between Inflammation and Cardiovascular Diseases". Int. J. Mol. Sci., 20(1599). [CrossRef] [PubMed]
- (14) Huang J. H., Lin Y. K., Lee T.W., Liu HW., Chien YM., Hsueh YC., Lee T., & Chen YJ., (2021), "Correlation between short- and mid-term hemoglobin A1c and glycemic control determined by continuous glucose monitoring", Diabetol Metab Syndr 13(94). https://doi.org/10.1186/ s13098 -021-00714-8.
- (15) Laltesh K., &Ajay L. D., (2018), "Assessment of Serum Lipid Profile in Patients of Coronary Artery Disease: A Case-Control Study", International Journal of Contemporary Medical Research, 5(5), p:59-62. <u>http://dx.doi.org/10.21276/ijcmr.2018.5.5.42</u>.
- (16) Małgorzata Z., Kinga P., Leokadia B. R., and Dorota K., (2020), "Correlates of Blood Pressure and Cholesterol Level Testing Among a Socially-Disadvantaged Population in Poland", Int. J. Environ. Res. Public Health, 17(2123), p:1-15. doi:10.3390/ijerph17062123.
- (17) Manda Y.R., & Baradhi K. M., (2021), "Cardiac Catheterization Risks and Complications", In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-.

- (18) Mansoor A., Parth M., Anil K. R., & Sudhir M., (2021)," Percutaneous Coronary Intervention", National Center for Biotechnology Information, U.S. National Library of Medicine.
- (19) Metwally Y.G., Sedrak H.K. & Shaltout I.F., (2020), "The relationship between coronary artery severity and insulin resistance in patients with impaired glucose tolerance and metabolic syndrome", Egypt J Intern Med., 32(21). https://doi.org/10.1186/s43162-020-00022-z.
- (20) Ming L., Michael A. W., Anoop A., Jing Y., Nischay R., Jinhong S., Leena H., Randal J. K., and Peter A., (2018)," Biosynthesis, structure, and folding of the insulin precursor protein), Diabetes Obes Metab., 20(2), p:28–50. doi:10.1111/dom.13378.
- (21) Mouri M. I., Badireddy M., (2021), "Hyperglycemia", [Updated 2021 May 10]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2022 Jan-.
- (22) Nader R,. (2018), "Tietz Fundamentals of Clinical Chemistry and Molecular Diagnostics", 8th Edition, Printed in the USA.
- (23) Paolisso P., Foà A., Bergamaschi L., Donati F., Fabrizio M., Chiti C., Angeli F., Toniolo S., Stefanizzi A., Armillotta M., Rucci P., Iannopollo G., Casella G., Marrozzini C., Galiè N., & Pizzi C., (2021), (Hyperglycemia, inflammatory response and infarct size in obstructive acute myocardial infarction and MINOCA", Cardiovascular Diabetology, 20(1), p=1–11. <u>https://doi.org/10.1186/s12933-021-01222-9</u>.
- (24) Rahman M. S., Hossain K. S., Das S., Kundu S., Adegoke E.O., Rahman M. A., Hannan M. A., Uddin M. J., Pang M. G., (2021), "Role of Insulin in Health and Disease: An Update", Int. J. Mol. Sci., 22(6403), p:1-19. https://doi.org/10.3390/ijms22126403.
- (25) Virani S. S., Alonso A., Aparicio H. J., Benjamin E. J., Bittencourt M. S., Callaway C. W., Carson A. P., (2021), "Heart Disease and Stroke Statistics—2021 Update", American Heart Association, Inc., Circulation,143(8), p: 254-743 . https://doi.org/10.1161/CIR. 00000000000950.
- (26) Shuai C., Ying S., Yong- Hua L., Yang D., Zhi- Ming W., Xiao- Qun W., Chen- Die Y., Le- Ying L., Jing- Meng L., Li- Ping Z., Wei- Feng S., Ri J., Lin L., & Feng- Hua D., (2021), "Impact of glycemic control on the association of endothelial dysfunction and coronary artery disease in patients with type 2 diabetes mellitus", Chen et al. Cardiovasc Diabetol, 20(64), p:1-9. <u>https://doi.org/10.1186/s12933-021-01257-y</u>.
- (27) Suzanne V. A., Deepak L. B., Gregory W. B., Alexis L. B., P. C., Silvio E. I., Mikhail K., Kasia J. L., Jonathan D. N., & Francine K. W., (2021), "Clinical Management of Stable Coronary Artery Disease in Patients With Type 2 Diabetes Mellitus", Circulation., 141, p:779–806. DOI: 10.1161/CIR.0000000000000766.

- (28) Tareq H. A., Hardi R. B., Salar H. K., & Dashty A. G., (2020), "Association of Lipid Profile with Body Mass Index in Public Employees in Halabja City, Kurdistan Region of Iraq", Polytechnic Journal, 10(2), p: 71-80. http://journals.epu.edu.iq/index.php/polytechnic.
- (29) Teresa S., Raffaele I., Emanuele B., Giuseppe D. G., Iginio C., Antonella F., Carmine M., Jozef B., Danilo F., Giuseppe A., Valerio P., Livio I., Bruno T., Giovanni E. and Antonio R., (2021), "Insulin Resistance Predicts Severity of Coronary Atherosclerotic Disease in Non-Diabetic Patients", J. Clin. Med., 9(2144), p:1-10. doi:10.3390/jcm9072144.
- (30) Ul-Haque I., Ud Deen Z., Shiza S., Syed I. u., Maryam Z., Syeda T. B., Misbah M., & Yusra W., (2019)," The Role of Glycated Hemoglobin A1c in Determining the Severity of Coronary Artery Disease in Diabetic and Non-Diabetic Subjects in Karachi", Cureus, 11(6): e4982. DOI 10.7759/cureus.4982.
- (31) Vargas E., Joy N. V., Carrillo S. M., (2022)," Biochemistry, Insulin Metabolic Effects", [Updated 2021 Oct 2]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing.
- (32) Yildiz K., Aclan O., (2021),"Glycosylated hemoglobin A1c predicts coronary artery disease in non-diabetic patients", J Clin Lab Anal., 35, e 23612. DOI: 10.1002/jcla.23612.
- (33) Zaman, G., (Ed.). (2019) "Ultimate Guide to Insulin", IntechOpen. https://doi.org/10.5772/intechopen.71990.