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RELATIONSHIP BETWEEN PREPTIN SERUM LEVEL AND OTHER BIOCHEMICAL PARAMETERS IN IRAQI DIABETIC NEPHROPATHY PATIENTS

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By Arya Jabar Karam AL-DALAWI

June 2022

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ABSTRACT

RELATIONSHIP BETWEEN PREPTIN SERUM LEVEL AND OTHER BIOCHEMICAL PARAMETERS IN IRAQI DIABETIC NEPHROPATHY PATIENTS

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Master of Science in Chemistry

Advisor: Prof. Dr. Volkan EYÜPOĞLU

June 2022

As a chronic metabolic illness, diabetes mellitus impacts glucose, fat, and protein metabolic processes. Here, we looked at preptin levels as well as other biochemical variables. Iraqi diabetes mellitus was researched for the link between these values. As the condition progressed into middle age, it became more prevalent among those in their forties and fifties. Our research found no variations in weight or height that were statistically significant. Preptin levels were found to be higher in diabetics, indicating that it plays a crucial function. As a result, more research on this topic is warranted. Diabetic individuals have lower quantities of trace elements in their bloodstreams compared to the general population. Despite statistical disparities, the lipid profile's total cholesterol level had no diagnostic value despite its importance and the fact that it was altered by the other indicators. The levels of urea and creatinine not effect. As for GOT and GPT also not effect, but the HbA1c and FBG changed this is normal because they are considered diagnostic parameters for diabetes mellitus patients. This leads us to conduct future studies on larger groups of patients to ensure its role in diabetes mellitus patients.

2022, 36 pages

Keywords: Preptin, Diabetic nephropathy patients, Biochemical parameters

ÖZET

IRAK DİYABETİK NEFROPATI HASTALARINDA PREPTIN SERUM SEVİYESİ İLE DİĞER BİYOKİMYASAL PARAMETRELER ARASINDAKİ İLİŞKİ

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Diyabetes Mellitus, glukoz, lipid ve protein metabolizmasını etkileyen kronik bir metabolik bozukluktur. preptin ve biyokimyasal parametrelerin düzeylerini ölçmek için bu çalışmayı yaptık. Bu miktarlar arasındaki korelasyon Iraklı Diyabetes Mellitus'ta incelenmiştir. Yaşın, özellikle kırklı ve ellili yaşlarda olmak üzere ileri yaşlarda hastalıkta artışa yol açan istatistiksel bir önemi vardı. Çalışmamızda ağırlık ve boy açısından istatistiksel olarak anlamlı bir farklılık yoktu. Preptin ise diyabet hastalarında düzeylerinin artmasının önemli bir role sahip olduğunu göstermektedir. Bu nedenle, bu konuda daha fazla çalışma yapılmalıdır. İz elementler ile ilgili olarak, diyabet hastalarında seviyeleri etkilenmiştir. Lipid profiline gelince, toplam kolesterol önemliydi ve seviyeleri etkilendi. diğer belirteçlere gelince ve istatistiksel farklılıkların varlığına rağmen, tanısal önemleri yoktu. Üre ve kreatinin seviyeleri etki etmez. GOT ve GPT'nin de etkisi yoktur, ancak HbA1c ve FBG bunu değiştirdi, çünkü bunlar diabetes mellitus hastaları için tanı parametreleri olarak kabul edildi. Bu da bizi diyabetes mellitus hastalarındaki rolünü sağlamak için gelecekte daha büyük hasta grupları üzerinde çalışmalar yapmaya yönlendirmektedir.

2022, 36 sayfa

Anahtar Kelimeler: Preptin, Diyabetik nefropati hastaları, Biyokimyasal parametreler

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Arya Jabar Karam AL-DALAWI

Çankırı-2022

CONTENTS

LIST OF SYMBOLS

%	Percent
/	Divide
+	Plus
=	Equal
µL	Microliter
Cm	Centimeter
dL	Deciliter
Fe^{2+}	Ferrous
Fe^{3+}	Ferric
Kg	Kilogram
m2	Square Meter
mg	Milligram
mL	Milliliter
ng	Nanogram
nm	Nanometer
O_2	Oxygen
ºC	Degrees Celsius
Pg	Picogram
α	Alpha
β	Beta
γ	Gama

LIST OF ABBREVIATIONS

ALT	Alanine Transaminase
AST	Aspartate Aminotransferase
DM	Diabetes Mellitus
DKA	Diabetic Ketoacidosis
DN	Diabetic nephropathy
DR	Diabetic Retinopathy
ELISA	Enzyme-Linked Immune Sorbent Assay
ELISA	Enzyme-Linked Immune Sorbent Assay
FBG	Fasting Blood Glucose
FPG	Fasting Plasma Glucose
FFAs	Free Fatty Acids
GBM	Glomerular Basement Membrane
SGOT	Glutamic Oxaloacetic Transaminase
GK	Glycerol Kinase
GPO	Glycerol Phosphate Oxidase
G1	Group 1
G2	Group 2
G3	Group 3
HDL	High-Density Lipoprotein
HOMA IR	Homeostatic Model Assessment Insulin Resistance
IFG	Impaired Fasting Glucose
IR	Insulin Resistance
IDF	International Diabetes Federation's
LPL	Lipoprotein Lipase
LDL	Low-Density Lipoprotein
MDH	Malate Dehydrogenase
NKHS	Non-Ketotic Hyperosmolar State
POD	Peroxidase
GPT	Serum Glutamic Pyruvic Transaminase
SD	Standard Deviation
TC	Total Cholesterol
TG	Total Triglyceride
T1DM	Type 1 Diabetes Mellitus
T2DM	Type 2 Diabetes Mellitus
VLDL	Very-Low-Density Lipoprotein
WHO	World Health Organization

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1. INTRODUCTION

1.1 Diabetes Mellitus

Sugar, fat, and protein metabolism are all affected by diabetes mellitus (DM). Persistent hyperglycemia (high blood glucose) is characterized as hyperglycemia that persists despite adequate insulin production, aberrant insulin action, or both inadequate insulin secretion and wrong insulin action (a hormone produced by the pancreas). One of the most frequent chronic, non-contagious illnesses that put human health at risk, it is rapidly spreading around the globe. Diabetes mellitus impairs the function of almost every organ in the body, from the heart to the eyes to the kidneys to the nervous system. As a result, early detection of the condition is essential.

In the globe, DM is the leading cause of morbidity and suffering. DM continues to be one of the world's most pressing public health concerns, with patients, their families, and the broader society bearing huge emotional and financial burdens as a result of the disease. Diabetes affects an estimated 450 million individuals (8.8 percent of adults aged 20 to 79) across the globe. The World Health Organization predicts that diabetes will be the sixth leading cause of death in 2030. (WHO).

1.1.2 Clinical presentation

Polydipsia, polyuria, polyphagia, blurred vision, and slow weight loss are all common signs of diabetes. Even in the most severe instances of diabetes mellitus, coma and, in the event of inconvenient medication, mortality might result from ketoacidosis or non-ketotic hyperosmolar conditions. This may influence both type 1 and type 2 diabetes (Asmat *et al.* 2016).

1.1.3 Pathogenesis

Insulin insufficiency is caused by autoimmune destruction of pancreatic cells, whereas insulin resistance is caused by problems other than autoimmune destruction of pancreatic cells (Hadjadj *et al.* 2016). Diabetes mellitus is characterized by abnormalities in glucose, lipid, and protein metabolism because of insulin's inability to reach its target organs.

Diabetes can be a side impact of other clutters, such as pancreatitis or haemochromatosis, which cause a decrease in affront emission. A few endocrine maladies, such as affront hostility exists in acromegaly and cushing's disorder activity caused by unusual hormone generation with restricting movement A few. Drugs have an adversarial impact on glucose resilience (Boutayeb *et al*. 2013).

Aim of Study

1- This case control study included investigating from some of the causes of diabetic nephropathy through study preptin serum level and biochemical parameter.

2- Evaluate serum levels of the lipids among patients of diabetic nephropathy in Iraq governorate.

3- Investigate correlation of preptin serum level with lipids levels in diabetic nephropathy.

2. LITERATURE REVIEW

2.1 Prevalence of DM

The prevalence of diabetes mellitus is rising steadily over the globe. Diabetes affected 8.5% of the worldwide adult population in 2014, up from 4.7% in 1980, according to a WHO global study issued on April 6, 2016. IDF estimates that over 415 million people globally have diabetes (IDF). By 2040, the IDF predicts that this figure will climb to 642 million (Atlas 2015). Diabetes and its consequences accounted for (5-20 percent) of adult health care spending in 2015, with the death toll from diabetes at over five million, according to the same estimate (Riddy et al. 2018). Besides, diabetes predominance has recently expanded essentially in countries that were not already known to have tall rates of the illness. Agreeing to the Worldwide Diabetes League, 10% of individuals within the Center East and Africa have diabetes, which is expected to climb to 20% by 2035 (Benjamin *et al*. 2017).

2.2 Diagnosis of Diabetes Mellitus

The individual's life is impacted by the determination of DM. As a result, both the specialist and the individual being assessed must have total confidence within the determination. Typically simple in symptomatic people, but in asymptomatic individuals, in the event that an unusual test is found, it must be affirmed by another (Suk *et al*. 2016).

Diagnosis of diabetes is still based on blood glucose levels since there is no more specific biological marker. Diabetes is diagnosed using a certain glucose level at which micro-vascular problems are much more likely to occur. This is the cutoff glycemic level used to diagnosis diabetes. Impeded glucose resistance (IGT) is characterized as PG levels of 140–199 mg/dL after a 75-gram verbal glucose resilience test by the Master Committee on Determination and Classification of Diabetes Mellitus in 1997 and 2003. (OGTT). There's a 110 mg/dL (6.1 mmol/L) IFG cut-off esteem characterized

by the World Wellbeing Organization and other diabetes organizations (Mohan and Anbalagan 2013).

2.3 Classification of Diabetes

2.3.1 Type 1 diabetes mellitus

Insulin-dependent diabetes mellitus (IDDM) was once known as Sort 1 diabetes (IDDM). This is often the express utilized to portray a condition in which affront treatment is required since the understanding is at chance of creating ketoacidosis. It is more common all through childhood or puberty. TIDM is assessed to account for 5% to 10% of all analyzed cases of diabetes (Balakumar *et al*. 2010).

T1DM happens when the pancreas' beta cells are annihilated, coming about in a need of affront. The body's safe framework assaults and slaughters the beta cells that create affront in TIDM. In spite of the fact that beta cell passing can take a long time, the disease's signs and side effects ordinarily show up inside a couple of months (Skillet *et al*. 2015).

2.3.2 Type 2 diabetes mellitus

Deficiency in insulin and poor glucose homeostasis are the hallmarks of type 2 diabetes mellitus (T2DM), a multifactorial, non-autoimmune, and multi-gene inheritance metabolic illness. Its pathophysiology appears to be a complex interplay of genetic predisposition and environmental variables. When poor insulin resistance (IR) is combined with a failure to create enough -cell insulin, T2DM occurs (Pham and Eggleston 2015). T2DM is significantly more common in adults, accounting for 90 percent to 95 percent of all diagnosed cases of diabetes. It includes people with IR who have relative insulin insufficiency at least initially and frequently throughout their lives. To stay alive, some people do not require insulin medication. In patients with T2DM, at

slightest two major obsessive absconds have been detailed: one may be a decrease in insulin's capacity to act on fringe tissue, known as IR (Balaji *et al*. 2019).

2.4 Etiology of Type 2 Diabetes Mellitus

In addition to gender, age, diet, obesity, and sleep deprivation as well as nutritional supplements provided to the mother while she is pregnant, many other variables influence the development of this medical condition, but the most significant ones are lifestyle, genetics, and other health issues (Chen *et al*. 2015).

2.4.1 Lifestyle factors

T2DM is influenced by a number of lifestyle variables. Obesity, weight gain, and physical inactivity all increase the risk of obesity. Cigarette smoking is a risk factor for IR, which is a precursor to diabetes, since it alters intracellular glucose transport, changes body fat distribution, and is potentially harmful to pancreatic tissue (Chawla *et al.* 2016).

2.4.2 Genetic factors

Most occurrences of type 2 diabetes are caused by a combination of multiple genes, each of which has a modest role in increasing the risk of developing diabetes. One identical twin has a 90 percent risk of acquiring diabetes, whereas a non-identical twin has a 25–50 percent chance of developing the disease over his or her lifetime. As of 2011, more than 36 genes have been linked to an increased risk of developing type 2 diabetes. Only 10% of the disease's heritability can be traced back to any one of these genes alone. Diabetes mellitus has a large number of genes that are involved in beta cell functions (Chaudhury *et al.* 2017).

2.4.3 Medical conditions

This section is divided into two sections, each containing two elements that may predispose to T2DM. The first entails the administration of drugs such as diuretics (thiazides), antihypertensive therapy, and antipsychotic therapy. Gestational diabetes, hyperthyroidism, Cushing's disease and certain malignancies such as glucagonoma comprise the second health problem (Tankova *et al.* 2001).

2.5 Complications of Diabetes Mellitus

Individuals with diabetes are at chance for a run of wellbeing issues as time goes on. These issues are classed as either intense (with a speedy onset and reversible) or persistent (with a longer onset and reversible) (taking a long time or decades to create). Intense results of diabetes incorporate hypoglycemia, hyperglycemia, and diabetic ketoacidosis (DKA) (Schwartz *et al*. 2016).

Atherosclerosis (hardening of the arteries) and nephropathy (skin damage) are the most prevalent long-term complications of diabetes. With increased expression of inflammatory genes, vascular inflammation is a common occurrence in the progression of diabetes problems. Increased oxidative stress, hyperglycemia, and dyslipidemia have been linked to the progression of diabetes complications (Dhatariya and Umpierrez 2017).

2.5.1 Acute complications

DKA and NKHS are more common in persons with type 1 diabetes than in those without the condition. Individuals with T2DM are more likely to have the NKHS (Fuchsberger *et al*. 2016).

Over time, this may lead to hyperglycemia, ketoacidosis, and acidosis, all of which are symptoms of diabetic ketoacidosis. Insulin deficiency, as well as an increase in counter-

regulatory hormones like glucagon and cortisol, is the primary cause of DKA. Hyperglycemia is caused by hormonal instability, which speeds up the breakdown of stored glucose and the production of new glucose. Free fatty acids (FFAs) are processed during the ketogenesis process when lipolysis is increased. Extreme rises in serum glucose and hyperosmolality characterize NKHS, yet there is no evidence of ketosis. In children, it has been uncommon. In the absence of ketoacidosis, the NKHS is characterized by extreme hyperglycemia, hyperosmolarity, and dehydration (Imam 2013).

2.5.2 Chronic complications of diabetes mellitus

DM is continuously connected to an hoisted hazard of microvascular illness (infection of the little blood vessels) and macrovascular illness (malady of the supply routes).

Harm to blood vessels is the fundamental cause of microangiopathy, which shows as one or more of the taking after side effects: Diabetic neuropathy, nephropathy, and retinopathy (Ting *et al*. 2016).

1. Diabetic retinopathy

Patients with diabetic retinopathy (DR) suffer from vision loss and even blindness as a result of microvascular damage to the retina and/or macula. With a long time of DM, the risk of developing DR rises.

2. Diabetic neuropathy

About half of all people with DM have some kind of neuropathy, whether it is mononeuropathy, polyneuropathy, or autonomic neuropathy. It is difficult for patients with DM to heal ulcers, the most common reason for non-traumatic amputation, because of their impaired peripheral sensory function and a damaged microvascular/macrovascular junction (Ozougwu *et al.* 2013). One of the most

prevalent and dangerous complications of diabetes is diabetic autonomic neuropathy (Gomes *et al.* 2018).

3. Diabetic nephropathy

Diabetes-related kidney disease (DN) accounts for the majority of cases of kidney failure in diabetics. Glial mesangium production of extracellular matrix (ECM) and development of kidney interstitial tissue define DN, the most common end-stage renal disease cause that ultimately leads to renal failure (Masharani *et al.* 2011).

There are three key histological alterations that diabetic nephropathy patients' glomeruli endure. Mesangial growth is directly impacted by hyperglycemia, either by increased production of matrix proteins or their glycation. Increasing the glomerular basement membrane (GBM) thickness is seen in Figure 2.1 in the second stage. Interglomerular hypertension, brought on by dilatation of the afferent renal artery, or damage brought on by hypoxia in the glomeruli itself may also produce glomerular sclerosis (Genuth *et al.* 2003).

These diverse histological patterns seem to have comparable predictive relevance. The glomeruli of the kidney are damaged as a result of these alterations, resulting in albuminuria (albumin in the urine) (Wu *et al.* 2014). DN is a condition that normally has no symptoms and causes people to generate normal volumes of pee. Renal failure symptoms can take 5–10 years to develop after kidney damage has begun. Itchy skin, headaches, severe weariness, a general feeling of illness, nausea, vomiting, a lack of appetite, and leg edema are some of the symptoms.

DN does not affect everyone who has diabetes. DN is more likely to progress in patients with T1DM 5 to 10 years or more after the onset of diabetes. Because people with T2DM may have had diabetes for several years, they may find that they have a tiny amount of protein in their urine when they are diagnosed with diabetes.

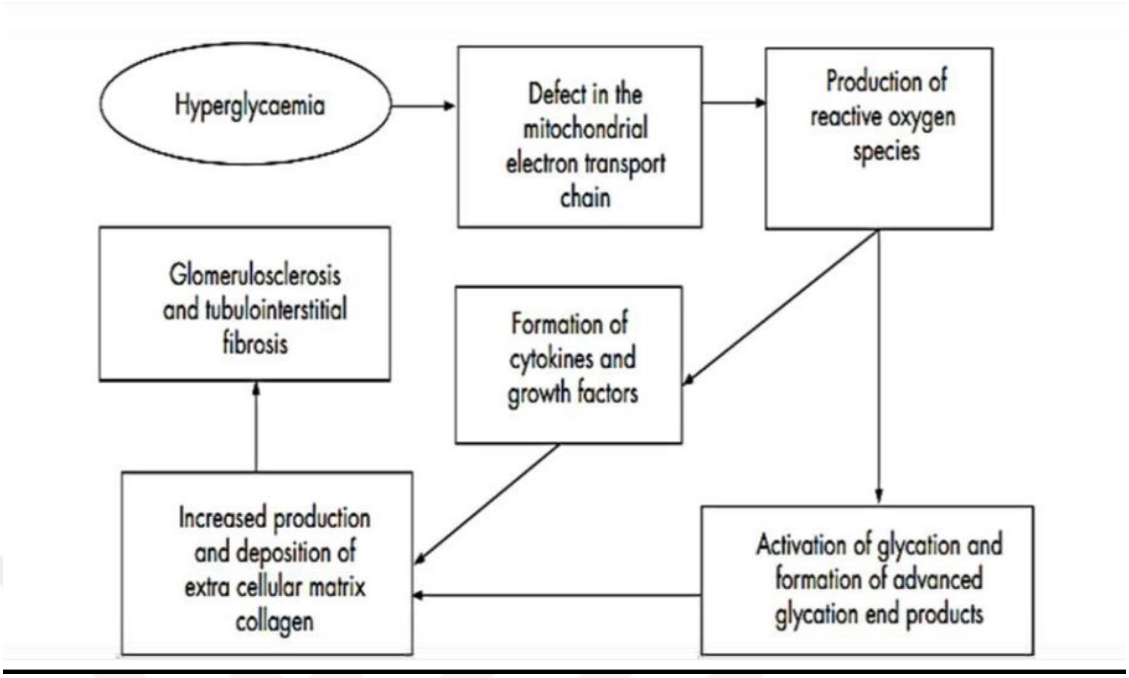

Figure 2.1 A simplified diagram of diabetic nephropathy etiology (Joseph *et al.* 2016).

High molecular weight proteins like albumin cannot penetrate the glomeruli's basement membrane, endothelial cell layer, or podocytes, which constitute a strong filtration barrier. Hemodynamic and metabolic variables associated with diabetes mellitus increase podocyte effacement when blood pressure and glucose levels rise and AGEs accumulate in the glomeruli (Herder and Roden 2011). There is a chain reaction that leads to pathogenic alterations in the course of the disease. These mediators' involvement in the DN's beginning and development may be seen in urine, and they can be used as biomarkers to evaluate the DN's current condition.

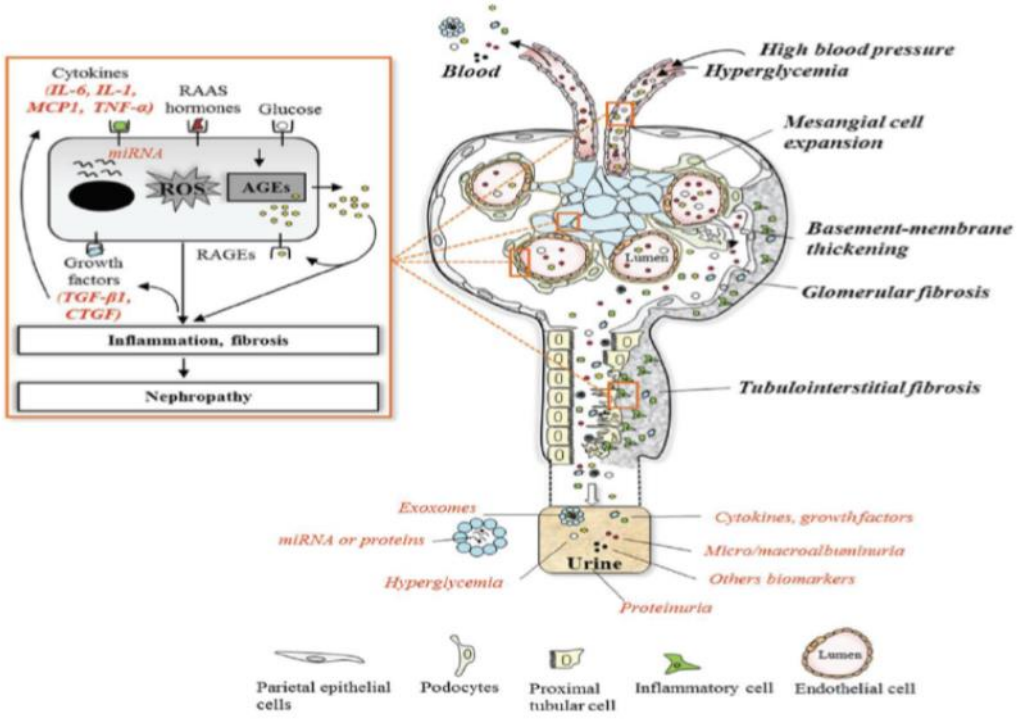

Figure 2.2 Relationship between nephropathy pathogenesis and relevant variables and biomarkers

Interleukin-6 (IL-6), interleukin-1 (IL-1), MCP-1 (MCP-1), and tumor necrosis factor-alpha (TNF-alpha) are only a few of the variables that contribute to the development of kidney impairment in people with diabetes (TNF-alpha) and connective tissue growth factor (CTGF) (TGF-1). Figure 2.2, Pathogenesis of Diabetic Nephropathy and Biomarkers (Karalliedde and Gnudi 2014).

2.6 Diagnosis of Nephropathy

According to the diagnosis of Marjan diabetic health center, which is based on ADA criteria, diagnosis is based on abnormal levels of urine albumin (protein) and PCR in a diabetic, as well as the elimination of alternative causes of albuminuria (proteinuria). The following are the definitions for albumin (protein) measurements:

-Urinary albumin excretion is less than 30 mg/24 hours in normal albuminuria (proteinuria).

- A urinary albumin excretion of 30–299 mg/24 hours is considered to be microalbuminuria.
- A urine albumin excretion rate under 300 mg per 24 hours is known as macroalbuminuria.

For the first five years after a type 1 or type 2 diabetes diagnosis, diabetics should have their albumin levels checked yearly (Kautzky-Willer *et al.* 2016)

3. MATERIALS AND METHODS

3.1 Selection of Patients

In this study involved 90 (47 male and 43 female). Their ages varied from thirty to sixty-two. Oral permission was acquired from the patients or their parents for both patients and control individuals. The chemistry center in Baquba Teaching Hospital, Diyala-Iraq, has these patients listed as T2DM with nephropathy and as T2DM without nephropathy. The patients were 30 T2DM with nephropathy, 30 T2DM without nephropathy and 30 controls also involved in this study.

3.2 Chemicals and Test Kits Used in Study

Table 3.1 lists all of the biochemical reagents and equipment.

Table 3.1 In the study of chemical compounds and test kits

Fasting blood glucose	Roche	Germany
HbA1c	Roche	Germany
İnsulin	Roche	Germany
HOMA IR	Roche	Germany
Cholesterol, triglyceride, HDL, LDL, and VLDL	Roche	Germany
Triglyceride	Roche	Germany
GOT and GPT	Roche	Germany
Urea and creatinine	Roche	Germany
Preptin	Human	Germany

3.3 Laboratory Equipment and Tools

As shown in Table 3.2, the study's tools and gadgets were utilized to help the researcher achieve correct data while also speeding up the process if everything was automated.

Table 3.2 Tools and equipment for laboratories

Equipments and tools	Made	Note
C311 Cobas	Germany	It was put to use in the lab for medical diagnosis. It was used to assess blood sugar levels, insulin, glucose, cholesterol, triglyceride levels, HDL cholesterol, LDL cholesterol, and VLDL cholesterol.
ELISA	Germany	In the laboratory, it was tested. The following measurements were made with this instrument: preptin.
Centrifuge	Germany	Serum and plasma, which are less dense than red blood cells and eggs, were placed on top, while the latter were placed below because they had a larger density
Freezer	Germany	Biochemicals, blood, serum, and plasma were saved and stored in it.
Incubator	Germany	Since the tests require serum at 37 °C, it was utilized to store blood, serum, and plasma.
Syringe	China	In arrange to conduct tests on the blood collected from the hands of patients, this gadget was utilized.
Pipettes	Germany	It was a logical gear for measuring or transporting microliters (L) or milliliters (ml) of fluid, such as serum or blood. It was utilized in a wide run of restorative and biochemical tests.
Test Tubes	China	Blood, serum, and plasma were all saved and stored in this device.

3.4 Method

3.4.1 Working of FBG, HbA1c, insulin, and HOMA IR

Principle of FBG: Tests for ultraviolet (UV) radiation are carried out. Hexokinase enzymatic activity standard method. In order to convert glucose to glucose-6-phosphate, hexokinase is necessary. Within the nearness of NADP, glucose-6-phosphate dehydrogenase oxidizes glucose-6-phosphate to gluconate-6-phosphate. Oxidized carbohydrates are the only ones that can be found in the body. Since NADPH generation is directly proportional to glucose concentration, it may be monitored using a photometer throughout the course of the reaction.

Principle of HbA1c: Glycated or glycosylated hemoglobins are subfractions of hemoglobin A1 (HbA) generated by the glycation of the alpha or beta chains. When glucose and amino groups at the N-terminus of hemoglobin A's beta chain are

combined, they generate hemoglobin A1c, which is the most well-defined of the three hemoglobins. Some of the aldimine is converted to a stable ketoamine form (Amadori rearrangement) while the red cell circulates (HbA1c). Glycated hemoglobin levels grow in tandem with blood glucose levels and red cell longevity, thus an increase in either is expected. In the clinical treatment of diabetes, A1c levels are used to evaluate the long-term effectiveness of diabetic control. An individual's glycated hemoglobin level reflects their two- to three-month average blood glucose level and the degree of carbohydrate imbalance.

Principle of insulin: insulin dosage, insulin injection timing, and the most suitable self-monitoring blood glucose (SMBG) test for evaluating the prescription's effectiveness. Since preprandial tests are simpler to recall than postprandial tests, preprandial testing is the recommended method of testing for people with type 2 diabetes. Postprandial SMBG is recommended if preprandial glucose results are within the goal range but A1C levels are still above the target range.

Principle of HOMA IR: HOMA is based on the idea that glucose feedback on -cells increases insulin production, which is central to the theory. Insulin resistance (R) of 1 and 100% -cell function are assumed in the model for normal-weight participants younger than 35 years of age.

3.4.2 Working of cholesterol, triglyceride, HDL, LDL, and VLDL

Principle of cholesterol: Using an enzymatic, colorimetric test on COBAS c systems, cholesterol can be determined quantitatively. Cholesterol esterase breaks down cholesterol esters into free cholesterol and fatty acids in order to break them down. Chemoatidyl oxidase is responsible for the conversion of cholesterol to hydrogen peroxide. 4-aminophenazone and phenol are oxidized to produce a red quinone-imine dye in the presence of hydrogen peroxide and the peroxidase enzyme. Depending on the amount of cholesterol present, the color intensity of the dye generated changes. The rise in absorbance is measured to ascertain this.

Principle of triglyceride: Triglycerides are hydrolyzed by lipoprotein lipase (LPL) into glycerol and fatty acids. ATP serves as the catalyst in the conversion of Glycerol to Glycerol-3-Phosphate by an enzyme known as Glycerol Kinase (GK). Glycerol-3-phosphate oxidation produces H2O2 and dihydroxyacetone as byproducts (a enzyme that catalyzes the oxidation). 4-Chlorophenol and 4-Aminophenazone must be oxidized in the presence of peroxidase with hydrogen peroxide to produce a red dye (POD). When a sample has more triglycerides, its absorbance rises accordingly.

Principle of HDL: An enzymatic colorimetric test in a homogeneous solution was performed. When dextran sulfate and magnesium ions are present, LDL, VLDL, and chylomicrons become resistant to PEG-enhanced enzymes. Enzymatically, HDL-cholesterol levels may be measured using cholesterol esterase and cholesterol oxidase (approx. 40 percent). Free cholesterol and fatty acids are formed as a result of the esterase known as "cholesterol esterase" breaking down cholesterol esters. When cholesterol is oxidized by cholesterol oxidase in the presence of oxygen, hydrogen peroxide and delta 4-cholestenone are produced. In the presence of peroxidase, hydrogen peroxide may be used to generate HSDA and 4-Amino-Antipyrine. An increase in HDL cholesterol is associated with a rise in the color intensity of the blue dye. The increase in absorbance may be assessed photometrically. An analogue of HSDA, or Sodium N-(2-hydroxysulfopropyl)-3,5-dimethoxyaniline.

Principle of LDL: A colorimetric assay that is perfectly homogeneous. It is possible to measure the levels of free and esterified cholesterol in LDL using a cholesterol esterase and an oxidase in the presence of surfactants that selectively solubilize LDL. Other lipoproteins than LDL are inhibited by enzyme reactions by surfactants and a sugar molecular. The amounts of HDL, VLDL, and chylomicron cholesterol have not been determined.

3.4.3 Working of GOT and GPT

Principle of GOT: The International Federation of Clinical Chemistry has developed a more accurate version of this test (IFCC). An amino group is transferred from L

aspartate to 2 oxoglutarate by AST in the sample, leading to the production of L glutamate and oxaloacetate. The oxaloacetate combines with NADH and malate dehydrogenase to create NAD+ (MDH). To put it another way, the rate at which NADH is oxidized correlates with the activity of AST. In order to ascertain this, the decrease in absorbance is analyzed.

Principle of GPT: The IFCC's criteria were adhered to, however the test was altered to increase its performance and stability, as required by the rules. 3,4 ALT reacts with L alanine and 2 oxoglutarate. In a process facilitated by NADH, lactate dehydrogenase (LDH) converts pyruvate to L lactate and NAD+. The rate of NADH oxidation directly affects the activity of the catalytic ALT enzyme. In order to ascertain this, the decrease in absorbance is analyzed.

3.4.4 Working of urea and creatinine

Principle of urea: Glutamate dehydrogenase and urease-based kinetic assay. Ammonium and carbonate are formed when urease hydrolyzes urea. As in the first process, the coenzyme NADH combines with ammonium to create L-glutamate in the presence of the glutamate dehydrogenase enzyme (GLDH). Two moles of NADH are oxidized to NAD+ for every mole of hydrolyzed urea that is used up in the reaction. As the amount of urea in a sample is decreased, the NADH concentration drops photometrically.

Principle of creatinine: As an in vitro test for creatinine concentration in human serum, plasma, and urine, creatinine is used by Roche and Hitachi cobas c systems. This enzymatic method, which makes use of creatininase, creatinase, and sarcosine oxidase, yields glycine, formaldehyde, and hydrogen peroxide. Hydrogen peroxide reacts with 4-aminophenazone and HTIBa to generate a quinone imine chromogen under the supervision of peroxidase. In high creatinine environments, the quinone imine chromogen's color intensity rises.

3.4.5 Working of preptin

Using sandwich enzyme-linked immunosorbent assay technique, we developed this kit. 96-well plates were coated with an anti-Preptin antibody. As for the detecting antibodies, the biotin conjugated anti-Preptin antibody was utilized. Following the addition of the standards, test samples, and biotin-conjugated detection antibodies, the wells were washed and dried. Wash buffer was used to remove unbound HRP-Streptavidin conjugates. The HRP enzymatic process was seen using TMB substrates. An acidic stop solution turned the blue TMB product into yellow when it was catalyzed by HRP. Depending on the quantity of Preptin in the sample, the density of yellow will vary. In a microplate reader, measure the O.D. absorbance at 450nm, and then determine the concentration of Preptin.

3.6 Statistics Analysis

A study based on numbers Version 21 of the statistical program for social science was used to examine the data (SPSS). All quantifiable data was analyzed using descriptive statistics. ANOVA was used to compare four groups on a variety of metrics. Pearson's correlation analysis was used to examine the link between two variables in a one-way comparison, as well as the risk of variables on four groups of patients (second) and to examine the association between the variables.

4. RESULTS AND DISCUSSION

DM. is a chronic metabolic disorder that affects glucose, lipid, and protein metabolism. I started working on my thesis from from October 2021 to march 2022 on 90 patients and healthy peoples suffer from diabetes type 2. We divided to three groups: the first group 30 people (17 male and 13 female) T2DM with nephropathy, the second group 30 people (14 male and 16 female) T2DM without nephropathy, and the third group 30 people (16 male and 14 female) control group.

Explored Hoisted serum preptin concentrations in patients with diabetic nephropathy. Preptin could be a peptide that's synthesized and discharged beside affront from pancreatic β cells. Here, we tried the theory that serum preptin concentrations are related with diabetic nephropathy (DN). Our ponder was conducted in a populace of 234 patients with sort 2 diabetes (T2DM) and 78 sound subjects. Patients with T2DM were partitioned into three bunches concurring to pee albumin/creatinine proportion (ACR), as normoalbuminuria gather (DN0, n=106), microalbuminuria gather (DN1, n=90) and macroalbuminuria gather (DN2, n=38). Serum preptin concentrations were altogether expanded within the three T2DM subgroups compared to controls. The DN2 gather appeared essentially higher serum preptin concentrations compared to the DN0 and DN1 bunches. Moreover, the DN1 gather had higher serum preptin concentrations than the DN0 bunch. Serum preptin was related with a better hazard of T2DM and DN after calculated relapse examination. Basically direct relapse investigation appeared a positive relationship between serum preptin and sex, body mass record (BMI), blood urea nitrogen, creatinine, ACR, and a negative relationship between serum preptin and glomerular filtration rate, metformin, acarbose treatment. Sex, BMI, and ACR were still emphatically connected with serum preptin after numerous straight relapse investigation. Our discoveries appear that serum preptin concentrations are related with kidney work and DN (Wang *et al.* 2019).

4.1 Discussion of Age, Weight, Height, and BMI

Investigated the relationship between obesity and diabetic nephropathy in China. BMI was calculated as weight (kg) partitioned by height2 (m2). Based on the cut-off focuses in Chinese grown-ups, BMI was classified as: hefty, ≥28.0 kg/m2; overweight, 25.0-27.9 kg/m2; powerless <25.0 kg/m2. Proteinuria and creatinine clearance rate (Ccr) per 24 hours were surveyed by 24-hour pee collection at the time of biopsy, and Ccr was calculated by the standard equation: pee creatinine (mg/dL) × urine volume (mL/min) /serum creatinine (mg/dL) and balanced for body surface. Other definitions were utilized: 1) proteinuria within the nephrotic run, 24-hour urinary protein excretion ≥3.5 g; 2) hypoalbuminemia, serum egg whites levels ≤3.5 g/L; 3) K/DOQ1 organize I, Ccr >90 mL/min/1.73 m2; and 3) renal disappointment, Ccr <60 mL/min/1.73 m2. Affront resistance was surveyed utilizing the Homeostasis Show Evaluation of Affront Resistance equation: fasting serum affront (μU/mL) × fasting plasma glucose (mmol/L)/22.5. Frailty was characterized as hemoglobin < 12 and < 11 g/dL in guys and females, separately (Chen *et al.* 2013).

In this study for the age we compared between three groups, for the T2DM with nephropathy the mean of age was (53.83 ± 6.54) and the mean of weight was (75.1 ± 8.1) and the mean of height was (165.9 ± 9.54) and BMI was (27.48 ± 3.85).while the mean for the T2DM without nephropathy was (55 ± 5.93) and the mean of weight was (76.9 ± 10.7) and the mean of height was (165.3 ±7 .95) and the BMI was (28.16 ± 3.62). for as the mean in control group age was (39.46 ± 6.82) and the weight was (73.3 ± 11.3) and the height was (169.6 ± 5.45) and the mean of BMI was (25.46 ± 3.49). As show Age, weight, height, and BMI for the T2DM with nephropathy, T2DM without nephropathy, and control in the Table 4.1 and Figure 4.1.

Table 4.1 The data of age, weight, height, and BMI

Parameter	Mean ± SD			P-value		
	G1 (n=30)	G2 (n=30)	G3 (n=30)	G1 & G2	G1 & G3	G2 & G3
Age (year)	39.46±6.82	53.83±6.54	55±5.93	0.06	0.08	0.47
Weight(kg)	73.3±11.3	75.1±8.1	76.9±10.7	0.48	0.21	0.47
Height(cm)	169.6±5.45	165.9±9.54	165.3±7.95	0.07	0.02	0.8
BMI (kg/m2)	25.46±3.49	27.48±3.85	28.16±3.62	0.03	0.005	0.48

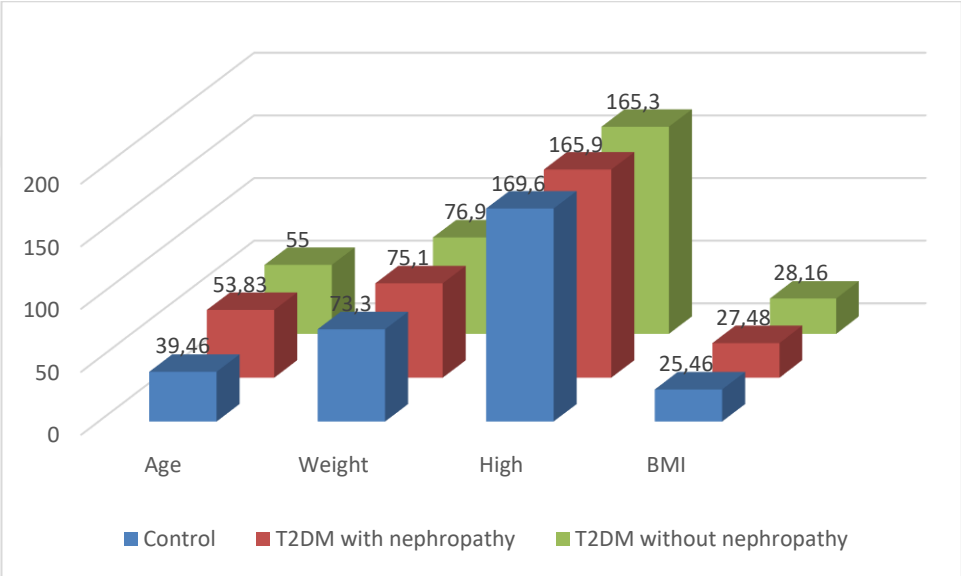

Figure 4.1 The diagram of age, weight, height, and BMI

4.2 Discussion of FBG, HbA1c, HOMA IR, and Insulin

Investigated the circulating levels of asprosin and its association with insulin resistance and renal function in patients with type 2 diabetes mellitus and diabetic nephropathy. Asprosin was found to be higher within the T2DM (6.73 ± 1.67) and T2DM + NP (7.11 ± 1.54) patients compared to the controls (4.81 ± 1.09) ($p < 0.001$), whereas adiponectin shown a lower concentration in both quiet bunches compared to the control bunch. In addition, IL-6 and TNF-α indicated higher levels within the two patients gather compared to the control bunch. Asprosin was watched to have a positive relationship with HbA1c, FBG, TC, LDL-C, IL-6, and TNF-α within the T2DM bunch. Within the patients with T2DM + NP, asprosin was found to be emphatically connected with BMI,

HbA1c, insulin, HOMA-IR, Cr, UAE, IL-6, and TNF-α, and it was conversely related with eGFR (Goodarzi *et al.* 2021).

The FBG for the T2DM with nephropathy the mean was (210.1 ± 64.4) compared with the control group, was a high statistically significant were statistically significant when compared to controls group, but the FBG for the T2DM without nephropathy was (159.4 ± 25.6), we found the significant compared with the normal group. While the control group was (86.3 ± 9.53).

For the patients suffer from T2DM with nephropathy we tooked also HbA1c the mean was (8.87 ± 2.23) we compared with the normal group we found high sensitive, also for the patients from T2DM without nephropathy the mean of HbAIc was (7.04 ± 0.95) compared with the normal group we see sensitive. While the mean of HbA1c in control group was (4.72 ± 0.29).

For the insulin the study substantially different between three group T2DM with nephropathy, T2DM without nephropathy, and control group, the serum level in T2DM with nephropathy the P. value was (0.139) compared with control group. While in the group of T2DM without nephropathy the P. value was (0.32) compared with the control group.

For the HOMA IR the patients in T2DM with nephropathy group the standard deviation was (6.60 ± 2.28) compared with the control group, but the patients in the T2DM without nephropathy group the mean was (4.92 ± 1.27) compared with the healthy group. While the healthy group the mean was (2.46 ± 1.02). Statistically, we found a high differences between the three groups. FBG, HbA1c, Insulin, and HOMA as showing in Table 4.2 and Figure 4.2.

Table 4.2 The data of FBG, HbA1c, HOMA IR, and insulin

Parameter	Mean ± SD			P-value		
	G1 (n=30)	G2 (n=30)	G3 (n=30)	G1 & G2	G1 & G3	G2 & G3
FBG(mg/dl)	86.3±9.53	210.1±64.4	159.4±25.6	0.0001	0.0001	0.0001
HbA1c (%)	4.72±0.29	8.87±2.23	7.04±0.95	0.0001	0.0001	0.0001
Insulin	11.56±4.45	13.1±3.58	12.5±2.51	0.139	0.32	0.43
HOMA IR	2.46±1.02	6.60±2.28	4.92±1.27	0.0001	0.0001	0.001

G1: Control, G2: T2DM with nephropathy, G3: T2DM without nephropathy

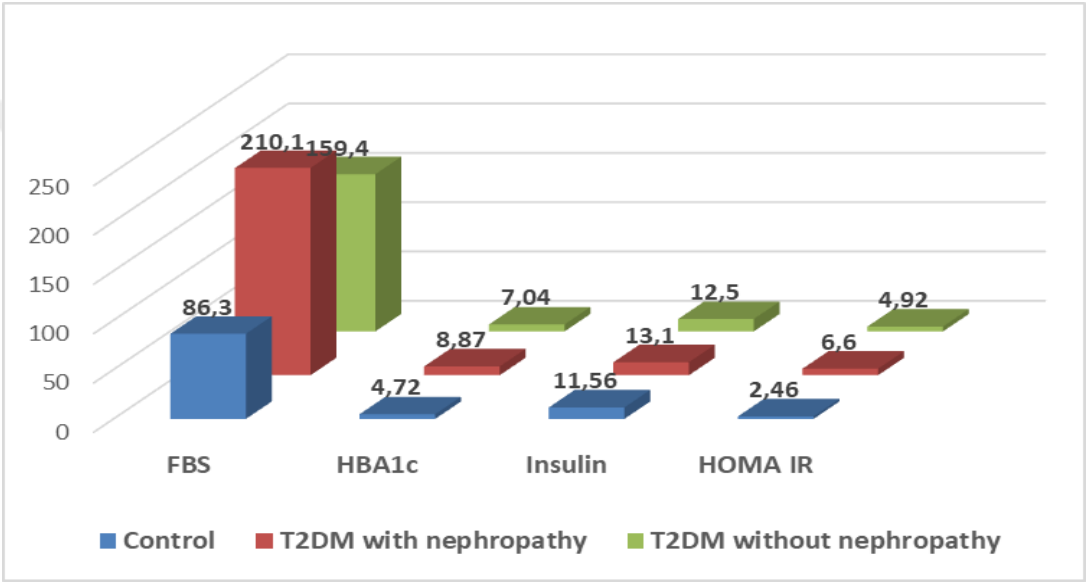

Figure 4.2 The diagram of FBG, HbA1c, HOMA IR, and insulin

4.3 Discussion of Cholesterol, Triglyceride, HDL, LDL, and VLDL

Investigated The Study of Lipid Abnormalities in Type 2 Diabetes Mellitus Patients with Nephropathy in Eastern India. Mean Total cholesterol, TG, LDL, sdLDL are essentially tall in nephropathy patients with p values 0.026, 0.012, 0.014, 0.04 individually. Assessed GFR features a noteworthy positive relationship with TCHOL ($r = -0.850$, $p = 0.01$), TG ($r = -0.14$, $p = 0.008$), LDL ($r = -0.62$ $p = 0.037$). Evaluated GFR encompasses a critical negative relationship with S. urea ($r = -0.587$, $p \leq 0.01$), S. creatinine ($r = -0.59$, $p \leq 0.01$), UACR ($r = -0.47$, $p \leq 0.01$). Dyslipidema sdLDL is essentially more in nephropathy gather in comparison to diabetic gather with p values

0.033, 0.045 separately. Our think about appears that dyslipidemia was exceedingly predominant among subjects with nephropathy. So cardiovascular dangers can be deflected by normal screening for dyslipidemia in diabetic nephropathy patients (Behera *et al.* 2020).

For the effect of T2DM with nephropathy, T2DM without nephropathy on lipid profile compared with control group we found difference. The P. value for the cholesterol was (0.0001) in the T2DM with nephropathy compared with normal group, but the T2DM without nephropathy compared with control group was (0.0001). While the compared between T2DM with nephropathy and T2DM without nephropathy the p. value was (0.05).

While the mean of triglyceride in T2DM with nephropathy was (207.7 ± 71.31) compared with the normal group, while in the T2DM without nephropathy the mean was (193.9 ± 32.9), but the mean in control group was (99.13 ± 14.0), that referred to statistic significant difference between three groups.

For the HDL the mean and standard deviation T2DM with nephropathy were (46.2 ± 5.34) compared with normal group (53.16 ± 4.94), while for the HDL the mean of T2DM without nephropathy were (44.96 ± 6.06) compared with normal group. That referred to statistic significant difference between three groups.

The effect of T2DM with nephropathy and T2DM without nephropathy on LDL level noted difference compared with the control group, the mean for the T2DM with nephropathy was (238.1 ± 51.7) and in the T2DM without nephropathy were (231.1 ± 40.6), while in the healthy group was (85.44 ± 29.4).

The VLDL means and standard deviation of studied three groups were (41.5 ± 14.2 as a group G2), (38.79 ± 6.58 as a group G3) comparing with control (19.82 ± 2.81). That referred to statistic significant difference. Cholesterol, triglyceride, HDL, LDL, and VLDL as showing in the Table 4.3 and Figure 4.3.

Table 4.3 The data of cholesterol, triglyceride, HDL, LDL, and VLDL

Parameter	Mean ± SD			P-value		
	G1 (n=30)	G2 (n=30)	G3 (n=30)	G1 & G2	G1 & G3	G2 & G3
TC	158.4±29.1	242.8±45.6	237.3±39.4	0.0001	0.0001	0.05
TG	99.13±14.0	207.7±71.31	193.9±32.9	0.0001	0.0001	0.05
HDL	53.16±4.94	46.2±5.34	44.96±6.06	0.0001	0.0001	0.05
LDL	85.44±29.4	238.1±51.7	231.1±40.6	0.0001	0.0001	0.05
VLDL	19.82±2.81	41.5±14.2	38.79±6.58	0.0001	0.0001	0.05

G1: Control, G2: T2DM with nephropathy, G3: T2DM without nephropathy

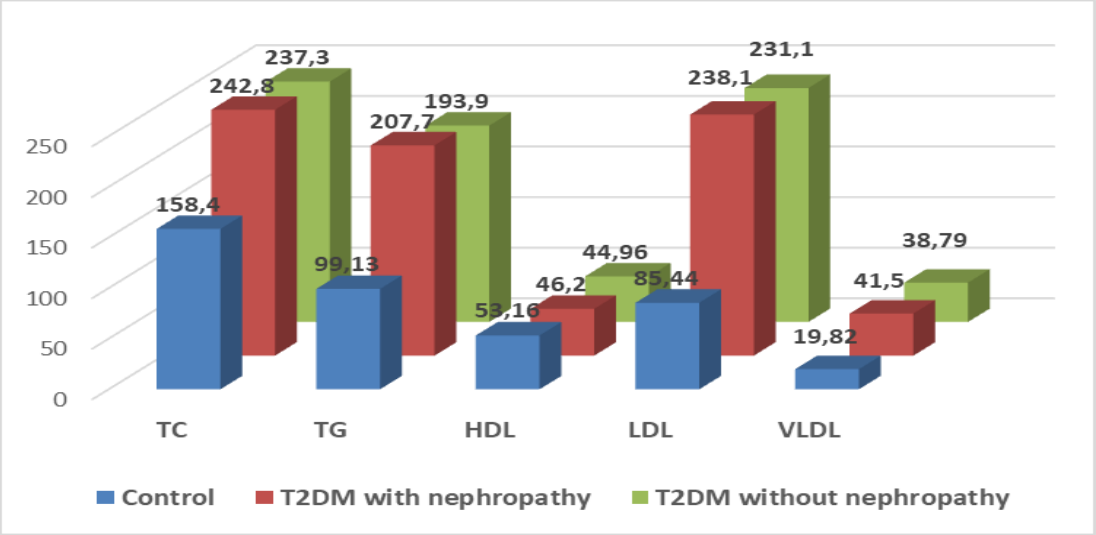

Figure 4.3 The diagram of cholesterol, triglyceride, HDL, LDL, and VLDL

4.4 Discussion of GOT and GPT

Investigated the serum glutamic oxaloacetic transaminase (GOT) and glutamic pyruvic transaminase (GPT) levels in diabetes mellitus. The exercises of serum glutamic oxaloacetic transaminase (GOT) and glutamic pyruvic transaminase (GPT) in 72 Libyan diabetic patients were decided. The particular cruel values were 24 +/- 1.1 U/L and 23.03 +/- 0.87 U/L. Noteworthy relationships were found between the exercises of GOT or GPT and fasting blood glucose, age, body mass record, heart rate and blood weight levels. The cruel values of GOT and GPT in diabetic patients with auxiliary complications were altogether higher than those without complications. On the other

hand, patients with family history of diabetes have higher cruel values of GOT and GPT when compared with those without diabetes within the family (Rao *et al*. 1989).

Investigated a comparative study of glutamate oxaloacetate transaminase (GOT) and glutamate pyruvate transaminase (GPT) levels in the saliva of diabetic and normal patients. Diabetes has been detailed to influence salivary organs antagonistically in people and test models. Glutamate oxaloacetate transaminase (GOT), glutamate pyruvate transaminase (GPT) and lactate dehydrogenase (LDH) are salivary chemicals that moreover are broadly conveyed in creature tissues. We decided GOT and GPT levels in spit tests of 100 sort 1 and 30 sort 2 diabetic patients utilizing reflectance spectrophotometry and compared them to 30 age and sex coordinated sound controls. Factually critical contrasts were watched within the cruel values of GOT and GPT in sort 1 diabetics compared to type 2 and control bunches. Altogether higher GOT levels were found within the 1-20 year age bunch of type 1 diabetics. Our discoveries propose that salivary organ harm is due to the same immunological assault that influences pancreatic β cells and comes about in type 1 diabetes (Verma *et al.* 2014).

The effect of T2DM with nephropathy and T2DM without nephropathy compared with the normal group, we found difference between them. The mean for the GOT in the T2DM with nephropathy group were (24.6 ± 5.06), while the mean for the T2DM with nephropathy group was (25.3 ± 5.11). While the control group were (23.53 ± 1.36).

Fot the effect of T2DM without nephropathy on GPT also showed difference between two group compared with normal. The mean in T2DM with nephropathy group were (38.1 ± 4.63) and in the T2DM without nephropathy group was (38.06 ± 4.43). While the healthy group were (28.7 ± 8.27). The GOT and GPT showing in Table 4.4 and Figure 4.4.

Table 4.4 The data of GOT and GPT

Parameter	Mean ± SD			P-value		
	G1 (n=30)	G2 (n=30)	G3 (n=30)	G1 & G2	G1 & G3	G2 & G3
GOT mg/dl	23.53±1.36	24.6±5.06	25.3±5.11	0.05	0.05	0.59
GPT mg/dl	28.7±8.27	38.1±4.63	38.06±4.43	0.05	0.05	0.97

G1: Control, G2: T2DM with nephropathy, G3: T2DM without nephropathy

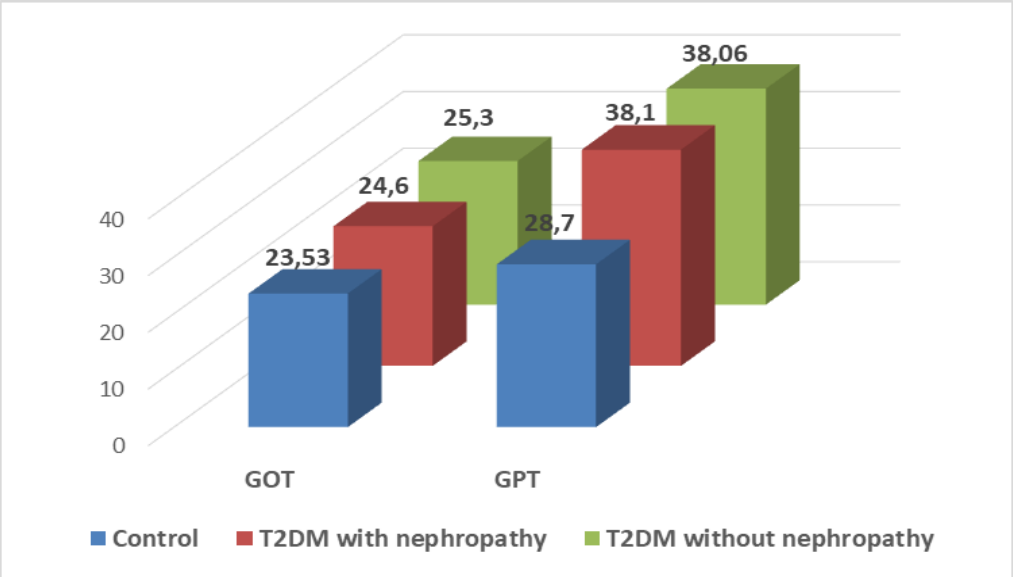

Figure 4.4 The diagram of GOT and GPT

4.5 Discussion of Urea and Creatinine

Investigated prevalence and determinants of Impaired Serum Creatinine and Urea among type 2 diabetic patients of jimma medical center, Jimma, Southwestern Ethiopia, 2019. The predominance of creatinine and urea impedance was 43.2% and 18.8% respectively. Duration of DM ≥ 10 a long time (AOR = 3.215, C.I = (1.542-6.702)) and expanded WHR (AOR = 2.238, C.I = (1.279-3.918)) were determinant factors for serum creatinine impedance. Term of DM (AOR = 3.721, C.I = (1.763-7.853)) and liquor drinking (AOR = 3.163, C. I = (1.079-9.273)) were altogether related with serum urea impedance. Creatinine and Urea disability was moderately common amongTtype-2

diabetic patients. Length of DM, liquor drinking and family history of CVD were autonomous indicators for urea and creatinine impairment (Kene *et al.* 2021).

The urea mean and standard deviation of study in two group were (50.86 ± 13.2 as a group T2DM with nephropathy) and in (42.9 ± 4.44 as a group T2DM without nephropathy) compared with the control group the mean was (28.8 ± 7.91), we found difference between them.

The creatinine mean and standard deviation of study in two group were (1.2 ± 0.45 as a group T2DM with nephropathy) and in (0.93 ± 0.44 as a group T2DM without nephropathy) compared with the control group the mean was (0.73 ± 0.24), we found difference between them. The urea and creatinine as showing in Table 4.5 and Figure 4.5.

Table 4.5 The data of urea and creatinine

Parameter	**Mean ± SD**			**P-value**		
	G1 (n=30)	**G2 (n=30)**	**G3 (n=30)**	**G1 & G2**	**G1 & G3**	**G2 & G3**
Urea	28.8 ± 7.91	50.86 ± 13.2	42.9 ± 4.44	0.01	0.01	0.004
Creatinine	0.73 ± 0.24	1.2 ± 0.45	0.93 ± 0.44	0.01	0.03	0.026

G1: Control, G2: T2DM with nephropathy, G3: T2DM without nephropathy

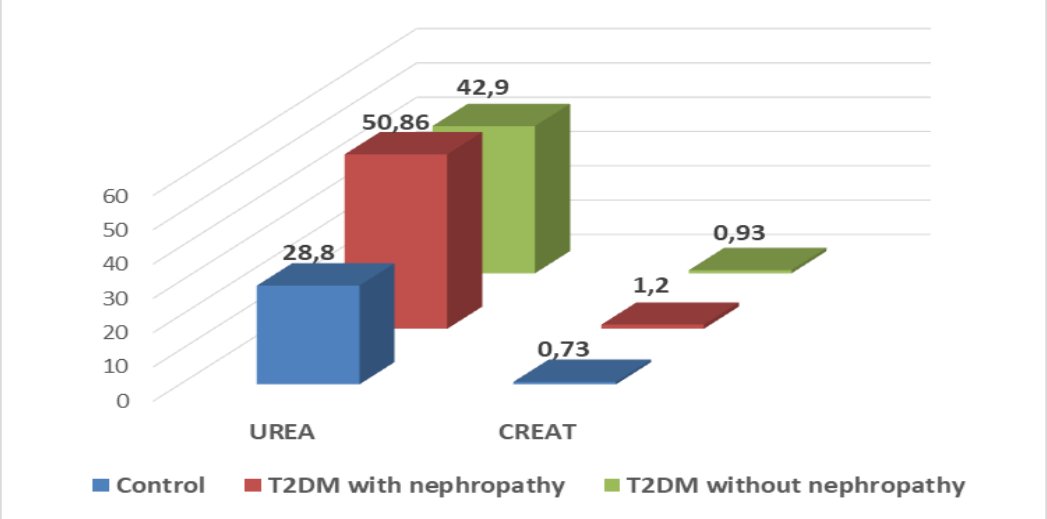

Figure 4.5 The diagram of urea and creatinine

4.6 Discussion of Preptin

Investigated plasma preptin levels in patients with type 2 diabetes mellitus. had altogether higher glucose, HbA1c, affront, homeostatic show evaluation of affront resistance (HOMA-IR), add up to cholesterol, triglyceride, low-density lipoprotein cholesterol, and preptin values ($p<0.001$ for each), and altogether lower high-density lipoprotein cholesterol levels compared with the control bunch ($p<0.001$). The preptin level illustrated a positive relationship with affront, HOMA-IR, HbA1c, and glucose levels (for control bunch: r=0.452-p=0.000, r=0.472-p=0.000, r=0.389-p=0.001, r=0.286-p=0.021, respectively; for T2DM bunch: r=0.484-p=0.000, r=0.544-p=0.000, r=0.288-p=0.009, r=0.236-p=0.033, respectively). The watched relationship between preptin level and DM a proposes that this peptide may have a conceivable part within the pathogenesis of DM, which may be especially valuable, given that DM could be a major open wellbeing problem (Kalayci *et al.* 2019).

The preptin mean in T2DM with nephropathy group was (567.6 = 121) and in the T2DM without nephropathy group the mean were (422.4 = 63.7) compared with the control group the mean was (229.7 = 23.8),That referred to statistic significant difference between three groups, as shown in the Table 4.6 and Figure 4.6.

Table 4.6 The data of preptin

Parameter	**Mean ± SD**			**P-value**		
	G1 (n=30)	**G2 (n=30)**	**G3 (n=30)**	**G1 & G2**	**G1 & G3**	**G2 & G3**
Preptin(Pg/ml)	229.7 = 23.8	567.6 = 121	422.4 = 63.7	0.0001	0.0001	0.0001

G1: Control, G2: T2DM with nephropathy, G3: T2DM without nephropathy

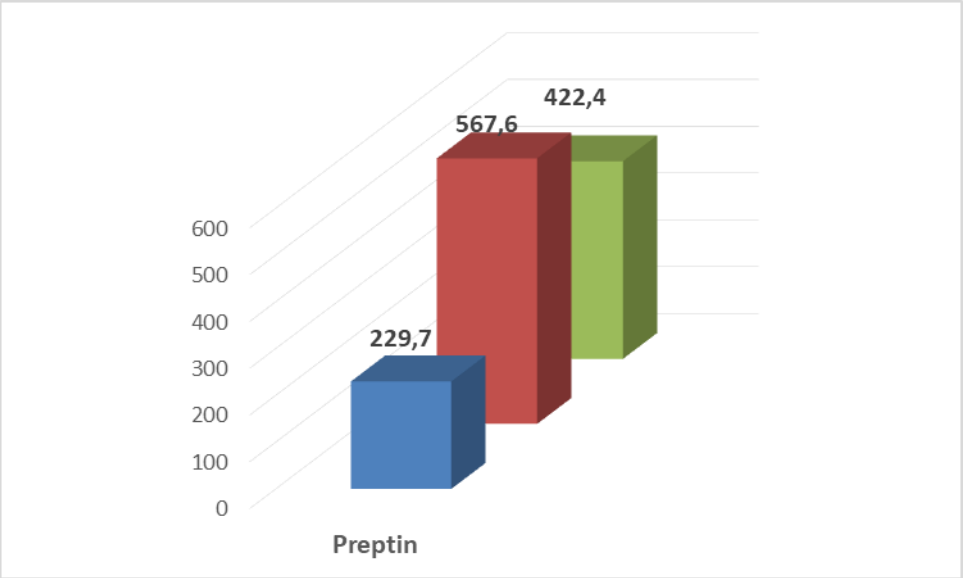

Figure 4.6 The diagram of preptin

4.7 Parameter Correlations

Correlation between Preptin and different parameters in T2DM with nephropathy and T2DM with nephropathy. As showing in Table 4.7.

Table 4.7 The data between preptin and different parameter

parameters	Preptin	
	r	p-value
Age	0.163	0.068
Weigh	-0.219	0.244
Height	-0.097	0.609
BMI	-0.074	0.697
FBG	0.323	0.01
HBA1c	0.030	0.875
TC	0.073	0.703
TG	0.252	0.180
HDL	0.057	0.765
LDL	0.128	0.501
VLDL	0.252	0.180
Urea	0.134	0.481
Creatinine	-0.144	0.449
Insulin	-0.002	0.991
HOMA IR	-0.057	0.767
GOT	0.192	0.310
GPT	0.117	0.537

5. CONCLUSION AND RECOMMENDATIONS

DM is a chronic metabolic disorder that affects glucose, lipid, and protein metabolism. Persistent hyperglycemia (high blood glucose) is characterized as hyperglycemia that persists despite adequate insulin production, aberrant insulin action, or both inadequate insulin secretion and wrong insulin action (a hormone produced by the pancreas). One of the most frequent chronic, non-contagious illnesses that put human health at risk, it is rapidly spreading around the globe. Diabetes mellitus impairs the function of almost every organ in the body, from the heart to the eyes to the kidneys to the nervous system. As a result, early detection of the condition is essential.

Some of the causes of diabetes include pancreatic autoimmune destruction, which results in insulin insufficiency, as well as anomalies that contribute to insulin resistance (Hadjadj *et al*. 2016). Diabetes can be a side effect of other disorders, such as pancreatitis or haemochromatosis, which cause a decline in insulin secretion. Some endocrine diseases, such as insulin antagonism exists in acromegaly and cushing's syndrome action caused by aberrant hormone production with opposing activity Several. Drugs have an antagonistic effect on glucose tolerance (Boutayeb *et al.* 2013).

Diabetic nephropathy (DN) is the most common cause of chronic kidney failure in people with diabetes. DN, the most common cause of end-stage renal disease and renal failure, is characterized by glomerulosclerosis, extracellular matrix production in the glomerular mesangium, and kidney interstitial tissue (Masharani *et al.* 2011). There are three key histological alterations that diabetic nephropathy patients' glomeruli endure. In the first place, increased matrix synthesis or glycation of matrix proteins in hyperglycemia influences mesangial expansion directly.

In our study, we found the effect of diabetes nephropathy on preptin, which led to an increase in its level, as well as an increase in the lipid profile, which may be the reason for weight gain. It also affected the levels of Hba1c, glucose and insulin, as diabetes is directly related to these parameters.

RECOMMENDATIONS

1. Increasing samples and working on other categories and comparing them with other parameters.
2. Working compared between preptin and other parameters.

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