"STUDY OF RENAL RESISTIVE INDEX IN DIABETIC KIDNEY DISEASE"

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Under the Guidance of

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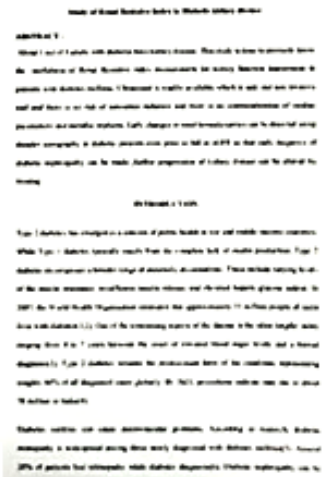

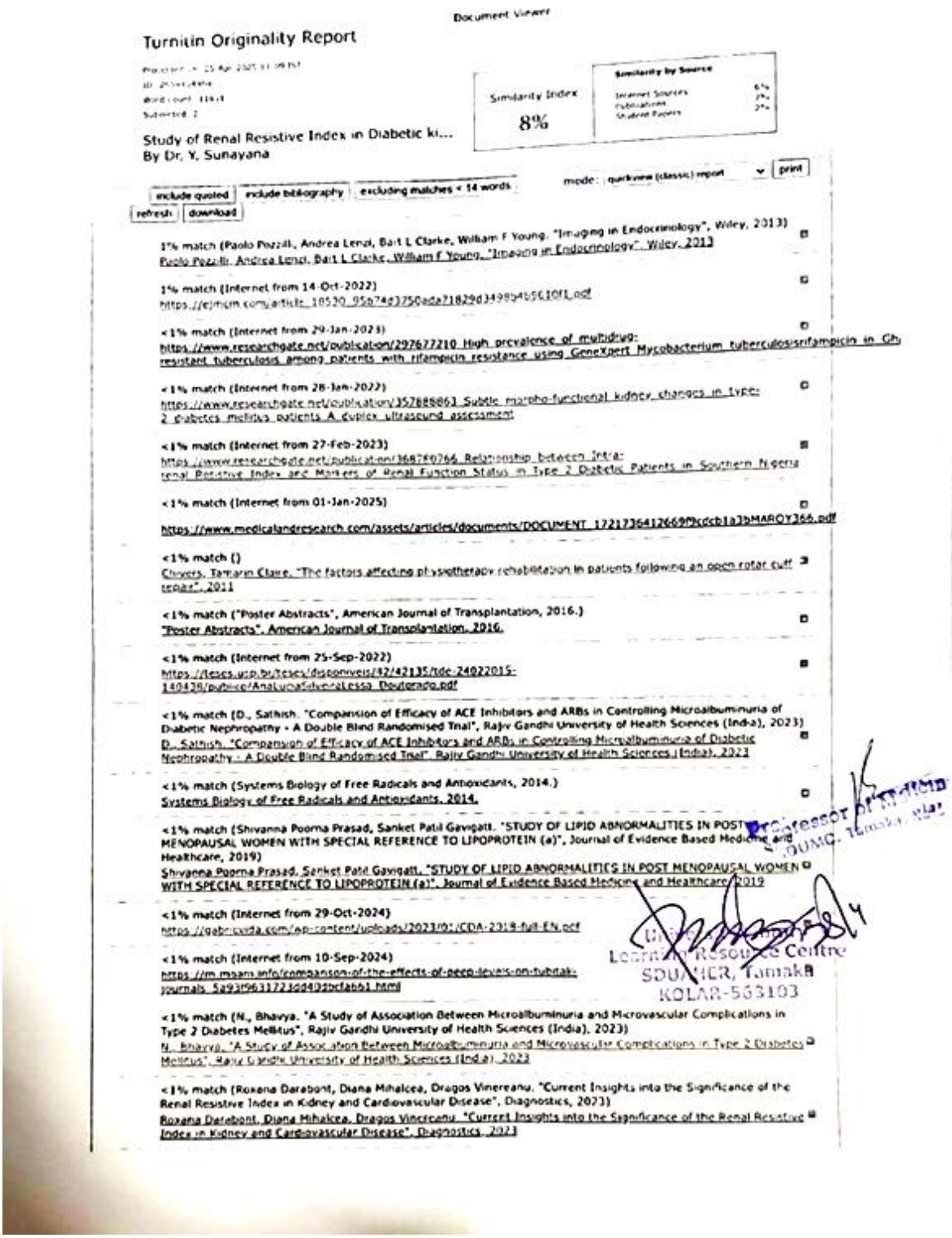

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Study of Renal Resistive Index in Diabetic kidney disease ABSTRACT - About 1 out of 3 adult with diabetes have kidney disease. This study is done to precisely know the usefulness of Renal Resistive index measurement for kidney function impairment in patients with diabetes mellitus. Ultrasound is readily available which is safe and non invasive tool and there is no risk of ionisation radiation and there is no contraindication of cardiac pacemakers and metallic implants. Early changes in renal hemodynamics can be detected using doppler sonography in diabetic patients even prior to fall in eGFR so that early diagnosis of diabetic nephropathy can be made ,further progression of kidney disease can be altered by treating. INTRODUCTION Type 2 diabetes has emerged as a concern of public health in low and middle-income countries. While Type 1 diabetes typically results from the complete lack of insulin production, Type 2 diabetes encompasses a broader range of metabolic abnormalities. These include varying levels of the insulin resistance, insufficient insulin release, and elevated hepatic glucose output. In 2007, the World Health Organization estimated that approximately 35 million people of India lives with diabetes(1,2). One of the concerning aspects of the disease is the often lengthy delay ranging from 4 to 7 years between the onset of elevated blood sugar levels and a formal diagnosis(3). Type 2 diabetes remains the predominant form of the condition, representing roughly 90% of all diagnosed cases globally. By 2025, projections indicate may rise to about 70 million in India(4). Diabetes mellitus can cause microvascular problems. According

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PRIOR PERMISSION TO START OF STUDY

The Institutional Ethics Committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has examined and unanimously approved the synopsis entitled **"Study Of Renal Resistive Index In Diabetic Kidney Disease"** being investigated by **Dr.Y Sunayana & Dr Prabhakar K** in the Department of General Medicine at Sri Devaraj Urs Medical College, Tamaka, Kolar. **Permission is granted by the Ethics Committee to start the study.**

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Place: Kolar **DR. Y SUNAYANA**

STUDY OF RENAL RESISTIVE INDEX IN DIABETIC KIDNEY DISEASE

ABSTRACT

BACKGROUND: About 1 out of 3 adults with diabetes have kidney disease. This study is done to precisely know the usefulness of Renal Resistive index measurement for kidney function impairment in patients with diabetes mellitus. Ultrasound is readily available which is safe and non invasive tool and there is no risk of ionisation radiation and there is no contraindication of cardiac pacemakers and metallic implants. Early changes in renal hemodynamics can be detected using doppler sonography in diabetic patients even prior to fall in eGFR so that early diagnosis of diabetic nephropathy can be made ,further progression of kidney disease can be altered by treating.

MATERIAL AND METHOD: MATERIAL AND METHODS: This cross-sectional study carried out in Department of General Medicine at R.L. Jalappa Hospital, Kolar, over a period spanning from May 2023 to December 2024. The study began only after receiving ethics committee approval, and every participant provided signed consent before inclusion. Both cases and controls before their enrollment in the research. Total 102 patients included in this study and divided into 3 groups i.e diabetes mellitus with diabetic kidney disease , diabetes without complications, age and sex matched healthy individuals

RESULTS: Most of the cases in this study were aged 50 - 60 years. Females were more in number than males. Mean HbA1c levels were 7.58%, 7.87%, and 5.6% in respective groups, showing a statistically significant difference across all groups . The average sr creat level was 2.3 mg/dL, 1.45 mg/dL, and 0.72 mg/dL in respective groups . BUN levels averaged 46.2 mg/dL, 33.3 mg/dL, and 31.0 mg/dL in respective groups, with statistically significant differences among the groups . Nearly 47.1% of group 1 had stage 3 chronic kidney disease, while group 2 primarily included patients with stage 1 disease . UACR levels averaged 123.32 mg/g, 10.9 mg/g, and 9.12 mg/g in respective groups with significant variation across all groups. The mean RRI was 0.9, 0.74, and 0.56. These findings were statistically significant.

CONCLUSION: This study states that both groups of diabetic individuals exhibited higher RRI values compared to the control group, with the highest indices observed in those with established diabetic kidney disease.

KEY WORDS: Renal resistive index , diabetic kidney disease .

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LIST OF ABBREVIATIONS USED

(in alphabetical order)

ACEIs	**Angiotensin converting enzyme inhibitors**
ARBs	**Angiotensin Receptor Blockers**
AGEs	**Advanced Glycation endproducts**
CI	**Confidence Interval**
CKD	**Chronic Kidney Disease**
DCCT	**Diabetes Control and Complications Trial**
DM	**Diabetes Mellitus**
DN	**Diabetic Nephropathy**
DNA	**Deoxy Ribonucleic acid**
ESRD	**End stage Renal Disease**
FDA	**Food and Drug Administration**
GFAT	**Glutamine Fructose – 6 – Phosphate amidotransferase**
GFR	**Glomerular Filtration Rate**
HbA_{1c}	**Glycosylated Hemogloobin**
HIFs	**Hypoxia Inducible Factors**
HNF- 1α	**Hepatocyte nuclear Factor – 1 alpha**
HOPE	**Heart Outcomes Prevention Evaluation**
HOT	**Hypertension Optimal Treatment**
IPF	**Insulin Promoting Factor**
MODY	**Maturity Onset Diabetes in Young**
NAD	**Nicotinamide adenine Dinucleotide**
NADPH	**Nicotinamide adenine dinucleotide phosphate**
PKC	**Protein Kinase – C**
RAAS	**Renin Angiotensin Aldosterone system**
RENAAL	**Reduction of End points in NIDDM with the Angitensin II anatagonist Losartan study**
RI	**Resistive Index**
RRI	**Renal Resistive Index**
T1D	**Type 1 Diabetes**
T2D	**Type 2 Diabetes**
UAE	**Urine albumin excretion**
WHO	**World Health organization**

INTRODUCTION

INTRODUCTION

Type 2 diabetes has emerged as a concern of public health in low and middle-income countries. While Type 1 diabetes typically results from the complete lack of insulin production, Type 2 diabetes encompasses a broader range of metabolic abnormalities. These include varying levels of the insulin resistance, insufficient insulin release, and elevated hepatic glucose output. In 2007, the World Health Organization estimated that approximately 35 million people of India lives with diabetes(1,2). One of the concerning aspects of the disease is the often lengthy delay ranging from 4 to 7 years between the onset of elevated blood sugar levels and a formal diagnosis(3). Type 2 diabetes remains the predominant form of the condition, representing roughly 90% of all diagnosed cases globally. By 2025, projections indicate may rise to about 70 million in India(4).

Diabetes mellitus can cause microvascular problems. According to research, diabetic retinopathy is widespread among those newly diagnosed with diabetes mellitus(5). Around 20% of patients had retinopathy while diabetes diagnosis(6). Diabetic nephropathy can be easily identified by assessing urine microalbumin levels. Diabetic neuropathy refers to peripheral neuropathy that manifests either clinically or subclinically in the presence of diabetes without any other underlying cause.

Extensive research has shown that maintaining strict blood sugar control help prevent or significantly prolong the development of side effects associated with diabetes. However, one of the greatest challenges lies in objectively determining whether high blood sugar levels are directly correlated with long-term diabetic complications, as there is currently no universally accepted method for evaluating diabetes management(7).

Diabetic nephropathy stands as the leading global cause of kidney failure, with its occurrence reported in approximately 15% to 40% of patients suffering from ESRD(8,9). Within India, the prevalence varies significantly, ranging from 0.9% to as high as 62.3%, and the burden is expected

to grow by 170% in developing nations. As of 2019, Type 2 DM was recognized as second most frequent cause of CKD and third largest factor contributing to CKD-related DALYs. Approximately 2.62 million cases of CKD associated to diabetes(10).

Kidney damage associated to DM is a serious health concern, affecting between 20% and 40% of diabetic individuals. Although both DM 1, DM 2 can lead to nephropathy, only a small proportion one with Type 2 DM leads to ESRD(11). Among the microvascular complications of diabetes, diabetic nephropathy is the most prevalent and is the primary driver of ESRF worldwide. It is typically identified by presence of albumin in urine levels exceeding 300 mg/day or also 200 µg/min, confirmed two times over a 3 to 6 month period(12).

Renal Doppler ultrasound is a widely accessible, non-invasive diagnostic modality that does not expose patients to ionizing radiation and is safe for those with pacemakers or metal implants. It can detect early disturbances in renal blood flow before noticeable declines in estimated glomerular filtration rate (eGFR), making it especially useful in the early identification of diabetic kidney involvement. Moreover, early detection facilitates timely intervention, which may slow or halt disease progression(12).

The resistive index (RI), measured via Doppler ultrasound, evaluates blood flow resistance in the intrarenal arteries. Normal RI values typically fall between 0.50 and 0.70. An elevated RI often signals a poor renal prognosis, particularly in CKD and kidney transplant settings. Measurements are taken at the interlobar or arcuate arteries and serve as a non-invasive tool to guide early therapeutic strategies and reduce complications(13).

Importantly, renal artery constriction can persist long before there are detectable changes in blood urea nitrogen or serum creatinine levels. Doppler ultrasonography helps monitor glomerular hemodynamics by measuring RI in arcuate arteries, supporting early detection and clinical management of diabetic nephropathy(14).

Despite its potential, research on the application of renal resistivity index the region remains scarce. so current study aims to examine the relationship between RI and renal function, and to identify potential predictors of elevated RI among diabetic individuals, both with and without clinical evidence of nephropathy.

AIMS & OBJECTIVES

AIM AND OBJECTIVES

OBJECTIVES OF THE STUDY

1. To assess renal resistive index in diabetic mellitus patients, including those with and without nephropathy.
2. 2)To examine renal resistive index, eGFR, and urine microalbuminuria in diabetic mellitus patients.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

"HISTORICAL PERSPECTIVE

Historical review

Diabetes mellitus (DM) is distinguished by hyperglycemia, which is accompanied by various associated conditions. These conditions have been recognized for centuries, with references found in ancient Egyptian papyrus discussing a polyuric condition resembling diabetes around 150 BC. Celsius (30 BC-50 AD) also acknowledged this illness. However, term diabetes, translates to 'run through' or 'a siphon' in Greek, originally used by Aretaeus in the second century AD, to describe individuals with increased urine output. Diabetes was referred to as "a remarkable condition, not very common among men, causing the melting of flesh and limbs into urine" by physicians Celsus and Aretaeus during the first century (30-90 AD). The patients produced copious amounts of water continuously, akin to water flowing from aqueducts, as described by Aretaeus of Cappadocia (15). The existence of sweet-taste compound in the urine was mentioned in Sanskrit manuscripts during the 5th and 6th AD, during the era of prominent Indian doctors Susrutha and Charaka (16). During this same timeframe, similar observations were made by Chinese and Japanese medical practitioners.

In 17th century, a person named Thomas Willis famously noticed taste of urine is sweet in individuals with diabetes. It was later clarified by Mathew Dobson that this sweetness was due to an abnormally high concentration of sugar. To differentiate this condition from other forms of excessive urination that lacked such characteristics, John Rollo introduced the term "mellitus," meaning "honey-sweet." Interestingly, as early as the 11th century, the Arab physician Aricanne documented cases of gangrene with diabetes.

The relation between prolonged diabetes and kidney injury has ancient roots, with Aretaeus in the second century AD providing early descriptions of such outcomes. During the 1700s and 1800s, medical thinkers such as Contunniues, Rollo, Darwin, Rayer, and Van Noorden identified and wrote about a link between diabetes and the accumulation of bodily fluids, a condition historically referred to as dropsy. In the late 1800s, Armani (1875) and Ebstein (1881) reported structural changes in kidney tubules, specifically vacuolization of the epithelial cells a change that Ehlrich later attributed to glycogen accumulation in 1888(17).

Rollo and Darwin also noted existence of protein in urine of diabetic individuals, an early sign of kidney involvement (18). The first histological descriptions of glomerular changes came from Clawson, and in 1936, Kimmelstiel and Wilson formally characterized a condition called intercapillary glomerulosclerosis (19). Subsequent findings by Bell revealed that both afferent and efferent arterioles in the kidneys were affected. Indian studies from the 1970s and 1980s highlighted alarmingly excessive mortality rate due to ESRD(20).

Pioneering work by Mongensen further clarified the progressive nature of diabetic nephropathy. The 1960s marked a turning point in management, with the initiation of kidney transplantation. In 1963, Keen and Chloverakis developed a radioimmunoassay to detect microalbuminuria an early marker of diabetic kidney damage(21).

DEFINITION(22)

Diabetes mellitus refers to group of metabolic disorders where blood sugar remains elevated because of defects in how insulin is made, how it works, or both. It can cause metabolic disruptions, resulting in subsequent pathophysiological alterations in a variety of organ systems. Diabetes' global prevalence may become a substantial factor to future death and morbidity.

Etiological Classification of Diabetes Mellitus

Diabetes mellitus encompasses broad range of metabolic disorders with various underlying causes. The classification is primarily based on the etiology and mechanisms of development of hyperglycemia.

Type 1 diabetes this form can be further divided into immune-mediated, where autoantibodies target the insulin-producing cells, and idiopathic, where no known immune markers are present, yet insulin deficiency still occurs.

The most widespread form of diabetes is type 2, which involves the body's reduced sensitivity to insulin along with an insufficient insulin response. It tends to have a gradual onset and is often linked to lifestyle and genetic predispositions.

Other diabetes types arise from a range of rare genetic and secondary causes. Some individuals develop diabetes due to single-gene mutations that impair β-cell function. For example, mutations in genes such as glucokinase, HNF-1α, HNF-1β, IPF-1, and NeuroD1 lead to forms of MODY. Mitochondrial mutations and defects in proinsulin or insulin conversion also fall under this category.

Some uncommon syndromes that lead to insulin resistance include conditions like Type A insulin resistance, leprechaunism, Rabson-Mendenhall syndrome, and various forms of lipodystrophy.

Secondary diabetes can result from pancreatic disorders such as chronic pancreatitis, surgical removal of the pancreas, tumors, cystic fibrosis, iron overload disorders like hemochromatosis, and fibrocalculous pancreatic disease.

Additionally, several endocrine abnormalities like Cushing's syndrome, acromegaly, hyperthyroidism, pheochromocytoma, and glucagonoma can disrupt glucose metabolism, leading to diabetes. Drug-induced diabetes has been observed in patients taking medications like corticosteroids, nicotinic acid, certain antihypertensives, protease inhibitors, antipsychotics, and immunomodulatory agents.

Infections have also been implicated in triggering diabetes in predisposed individuals. Notable pathogens include congenital rubella, cytomegalovirus, and coxsackievirus. Certain uncommon autoimmune disorders like stiff-person syndrome or those with antibodies targeting insulin receptors can also play a role.

Furthermore, diabetes can be part of larger genetic syndromes. Impaired blood sugar regulation has been linked to a range of genetic disorders, such as Prader-Willi, myotonic dystrophy, Huntington's disease, Wolfram syndrome, Turner and Klinefelter conditions, as well as Down syndrome, porphyria, and Friedreich's ataxia.

Gestational diabetes is diagnosed when abnormal glucose tolerance is detected for the first time during pregnancy. Although it often resolves after delivery, it can signal a higher likelihood of developing diabetes later in life.

Complications of Diabetes Mellitus

Diabetes can give rise to both acute and chronic health complications, based on how long the condition has been present and how well it is managed.

Acute complications include serious metabolic emergencies like diabetic ketoacidosis, hyperosmolar hyperglycemic state, and dangerously low blood sugar levels each requiring immediate medical attention.

Prolonged high blood sugar levels often lead to chronic issues, primarily involving damage to both small and large blood vessels. Small vessel involvement can result in complications like damage to the eyes, kidneys, and nerves, while large vessel disease raises the risk of heart attacks, strokes, and poor circulation in the limbs.

Other health issues frequently observed in diabetic patients include a heightened susceptibility to infections such as urinary tract infections, tuberculosis, oral and genital candidiasis, and severe fungal infections like mucormycosis. Diabetes also predisposes individuals to necrotizing fasciitis, chronic periodontal disease, and poor wound healing.

Microvascular complications in Diabetes Mellitus

Diabetes mellitus, whether it arises due to genetic predisposition or develops later in life, is characterized by sustained elevations of blood glucose. This persistent hyperglycemia due to deficiency in insulin production. Over time, the condition gives rise to various complications, particularly those involving small blood vessels.

Pathogenesis (23)

The long-term elevation of blood glucose in diabetes leads to damage across multiple organ systems. Microvascular issues arise due to injury to the body's smallest blood vessels. Simultaneously, diabetes also contributes to macrovascular complications through its impact on larger blood vessels and systemic circulation.

According to research, microvascular difficulties are mostly caused by high blood sugar levels, whereas insulin resistance and atherosclerosis are the major causes of macrovascular illness.

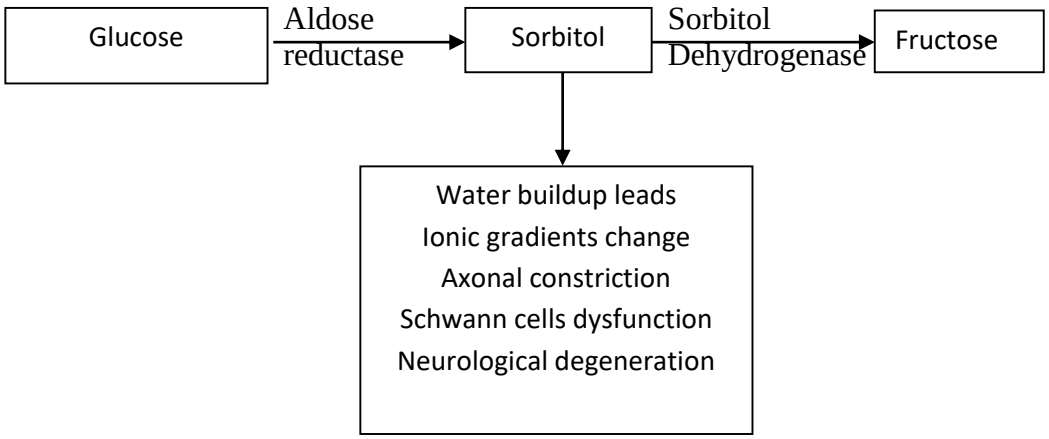
Microvascular complications arise from the following mechanisms:

In conditions of hyperglycemia, most cells can successfully regulate glucose intake, resulting in stable internal glucose levels. However, hyperglycemia can harm cells that are unable to execute this role properly, such as endothelial and mesangial cells.

1. Increase activity along the polyol pathway

This polyol route is driven by aldose reductase. This enzyme normally turns toxic aldehydes into innocuous alcohols; but, when glucose levels rise within the cell, it converts glucose into sorbitol, is subsequently oxidised into fructose and aldose reductase requires NADPH as a cofactor for the conversion step. NADPH also required for production of reduced glutathione, a crucial antioxidant found in cells. This mechanism makes cells more vulnerable to oxidative stress by lowering reduced glutathione levels.

NADPH NADP NAD^+ NADP

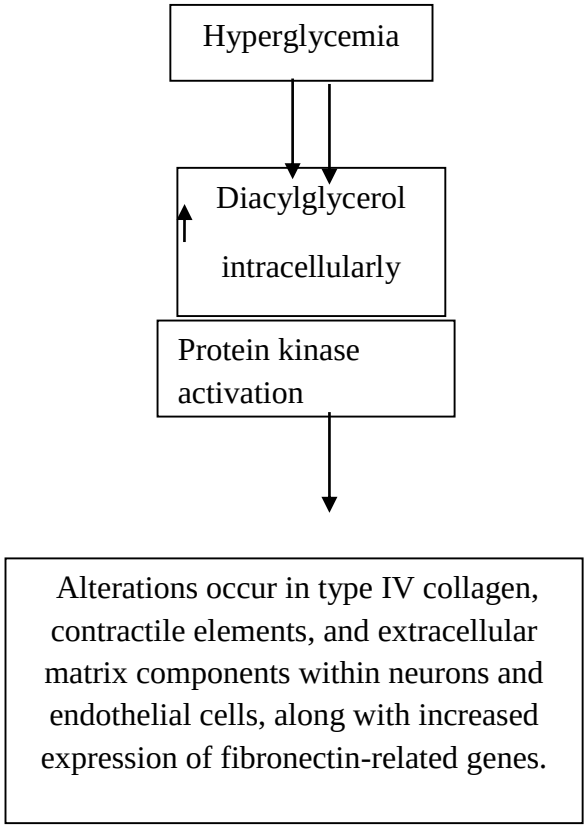


2] The formation of advanced glycation endproducts (AGEs)

AGEs formed , sugars bind to proteins or fats without enzyme involvement and undergo oxidative changes. AGEs injure cells by changing intracellular proteins, affecting extracellular matrix signalling, and modifying blood proteins, all of which contribute to inflammation and vascular disease.

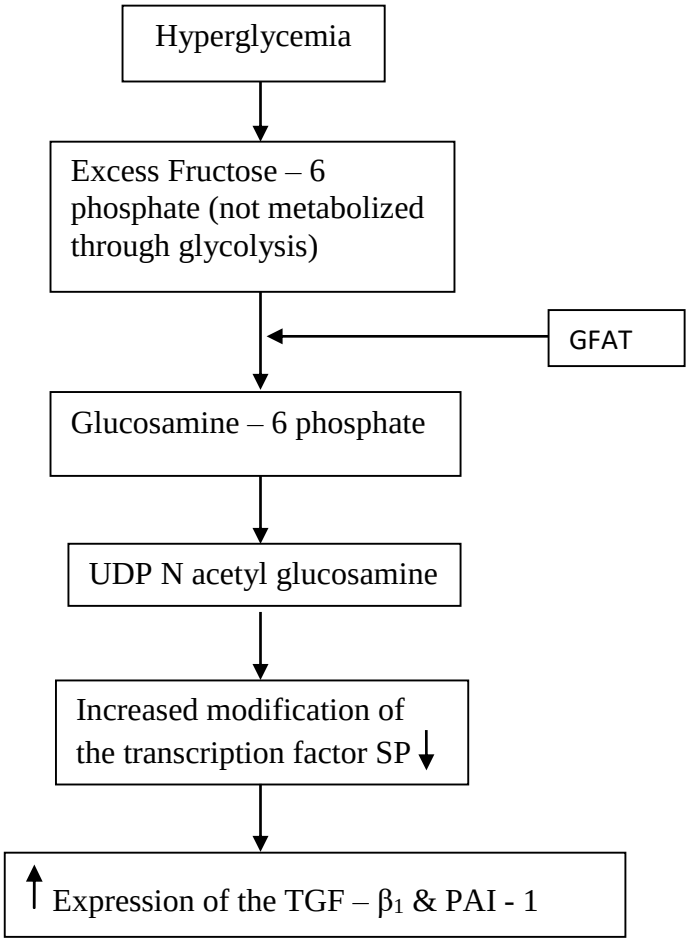
3] Protein kinase activation (PKC activation)

Hyperglycemia triggers the production of a substance called diacylglycerol within the cell, It serves as a crucial cofactor for the conventional isoforms of protein kinases. Elevated intracellular glucose levels activate PKC, leading to various consequences in gene expression. In all cases, beneficial products are diminished while harmful products are increased.



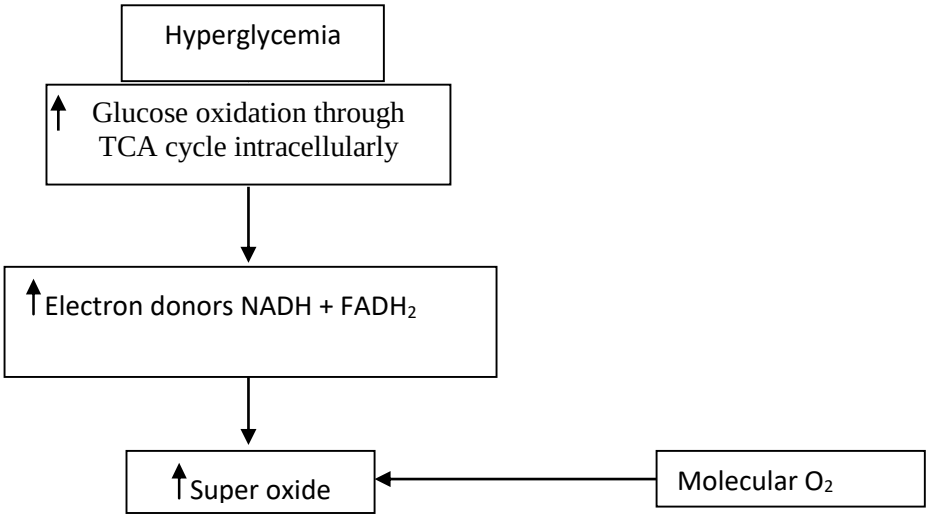
4] Elevated hexosamine pathway activity

One of the contributing mechanisms behind microvascular complications in diabetes involves increased activity of the hexosamine biosynthetic pathway. As intracellular glucose levels rise, a greater proportion of glucose is metabolized through glycolysis, starting with its conversion G-6-P, which is then transformed into F-6-P. While most of this enters the standard glycolytic cascade, a portion is rerouted by GFAT. This compound modifies transcription factors by attaching N-AG to serine and threonine residues, in a manner comparable to phosphorylation. When these modifications occur excessively, they can disrupt normal gene regulation, ultimately contributing to pathogenesis of diabetes- linked vascular side effects.



Elevated blood sugar levels enhance superoxide generation by mitochondria and induce oxidative stress within cells

In individuals with diabetes, rises blood glucose levels causes enhanced glucose metabolism through TCA cycle. Leads to increase electron carriers such as NADH and $FADH_2$, which in turn feed into the mitochondrial ETC. Once this gradient reaches a certain threshold, the electron flow through complex III becomes inefficient, leading to a backlog of electrons at the level of coenzyme Q. These excess electrons shift to molecular oxygen, ultimately generating superoxide a reactive oxygen species in cellular oxidative stress and tissue damage.



Excessive production of superoxide within the mitochondria plays a key role in initiating hyperglycemia- induced cellular damage. It does so primarily by inhibiting the enzyme GAPDH, which serves as a pivotal control point. This inhibition triggers the cascade of four key pathways involved in glucose toxicity, making mitochondrial oxidative stress the upstream event that amplifies downstream complications.

Macrovascular Pathogenesis

When glycated hemoglobin (HbA1c) levels rise from 5.5% to 9.5%, microvascular complications increases tenfold, while chance of macrovascular events nearly doubles. However chronic hyperglycemia alone is not the primary driver of macrovascular disease in diabetes. Rather, insulin resistance emerges as the dominant pathological mechanism.

In the context of insulin resistance, elevated levels and oxidation of free fatty acids (FFAs) in macrovascular endothelial cells lead to excessive generation of ROS. This oxidative stress mirrors the damaging effects seen in microvascular complications by activating several harmful pathways, This includes triggering hexosamine pathway, activating protein kinase C, generating advanced glycation end-products (AGEs).

DIABETIC NEPHROPATHY

Diabetic nephropathy remains important reason for ESRD, affecting greater than 40% with type 1 or type 2 diabetes. This renal complication increase risk of mortality, largely because of its association with CVD, particularly CAD. Clinically, it is marked by elevated urinary albumin excretion (UAE) absence of rest of identifiable kidney pathologies. The condition typically evolves from microalbuminuria, classified as urinary albumin excretion ranging from 20 to 199 μg/min for microalbuminuria, and above 200 μg/min for macroalbuminuria.

Several risk factors leads progression of diabetic nephropathy. These include persistent hyperglycemia, elevated blood pressure, a genetic predisposition, dyslipidemia, tobacco use, and excessive protein intake.

Screening for nephropathy begin five years post-diagnosis, or earlier in cases of early puberty or inadequate glycemic control for type 1 DM. In contrast, while type 2 diabetes undergo annual screening starting from diagnosis time . Individuals presenting with albuminuria should also be assessed for associated complications, such as diabetic retinopathy and cardiovascular disease.

Effective management strategies aim to delay disease progression and reduce cardiovascular risk. These include maintaining glycated hemoglobin (HbA1c) levels below 7%, controlling blood pressure to under 130/80 mmHg (and under 125/75 mmHg in patients with heavy proteinuria or elevated serum creatinine), initiating treatment with RAAS inhibitors, and keeping LDL cholesterol below 100 mg/dL.

Epidemiological Insights

Data from EURODIAB indicated that 12.6% of individuals with type 1 DM developed microalbuminuria over a 7.3-year observation period (24). A separate longitudinal study conducted in Denmark found the condition affected nearly one-third of patients over an 18-year timeframe. Among individuals of type 2 DM, the U.K. Diabetes Study also reported an annual incidence of microalbuminuria of around 2.0% , with cumulative ubiquity 25% after a decade(25).

Proteinuria, a hallmark of advancing nephropathy, occurs in 15–40% of those with type 1 diabetes, with peak onset typically occurring during adolescence or early adulthood. In type 2 diabetes, prevalence estimates vary significantly by population, ranging between 5% and 20%(26).

Ethnic differences also influence disease risk. Higher rates of diabetic nephropathy have been documented among African Americans, Asians, and Native American populations contrast to Caucasians. Between 1991 and 2001, proportion of patients with diabetic nephropathy requiring

kidney transplantation doubled. Although tools such as the renal resistive index have been introduced for earlier detection and slowing disease progression, their widespread implementation has yet to produce the anticipated improvements in outcomes(27,28)

Stages and Clinical Features

The ADA classifies diabetic nephropathy based on albumin in urine levels. The condition is categorized into two stages: microalbuminuria and macroalbuminuria. These stages guide diagnosis, monitoring, and the intensity of therapeutic interventions(29).

Stages	**Albuminuria cutoff values**	**Clinical characteristics**
Microalbuminuria	20–199 μg/min	Abnormal nighttime reduction in blood pressure and heightened blood pressure levels
	30–299 mg/24 h	Elevated triglycerides, total and LDL cholesterol, and saturated fatty acids
	30–299 mg/g	Increased occurrence of metabolic syndrome components
		Endothelial dysfunction
		Association of diabetic retinopathy, limb amputation, and cardiovascular diseases
		Higher cardiovascular mortality risk
		Stable GFR
Macroalbuminuria	≥200 μg/min	Hypertension
	≥300 mg/24 h	Elevated triglycerides and total and LDL cholesterol levels
	>300 mg/g	Asymptomatic myocardial ischemia
		Progressive decline in GFR

The onset of diabetic nephropathy and elevated cardiovascular risk can occur even when urinary albumin excretion (UAE) appears to be within limits. In type 2 diabetics , even a slight rise in UAE exceeding 2.5 mg over 24 hours has been associated with a heightened likelihood of developing either microalbuminuria or macroalbuminuria. Long-term data from a decade-long study revealed that when UAE surpasses 10 μg/min, the risk of progressing to nephropathy increases by nearly 29 times, a risk level comparable when seen in type 1 diabetes. These findings underscore importance of closely monitoring UAE values, even those below the conventional thresholds for microalbuminuria, as the associated risk may already be clinically significant(30).

Screening and Diagnosis of Diabetic Nephropathy

Catching diabetic kidney damage early is critical, since around 7% of patients already have microalbuminuria when they're first diagnosed. In type 1 diabetes, routine screening usually begins five years after diagnosis(31). However, studies suggest that nearly 18% of such patients may develop microalbuminuria even before this timeframe, particularly those with poor blood glucose and lipid regulation or coexisting hypertension. For younger patients with type 1 diabetes especially less sugar control or post-puberty testing should start within a year of diagnosis. If initial results are normal, yearly screenings are still essential for both types of cases(32,33).

The most practical and widely accepted method for screening diabetic nephropathy measuring albumin concentration in the spot urine sample, which may be collect in the morning or during any random visit. This test, by American Diabetes Association, is both accurate and convenient. Although 24-hour urine collection offers a more comprehensive profile, it is often impractical due to the complexities of accurate sample handling. Spot urine tests can report albumin levels directly in mg/L or as a ratio to creatinine (UACR in mg/g or mg/mmol)(34,35). Despite possible dilution effects, it's still a dependable and low-cost method. A threshold of 17 mg/L is generally used as a

diagnostic benchmark, aligning closely with the European Diabetes Policy Group's cut-off value of 20 mg/L. To confirm the presence of microalbuminuria, minimum of two out of three samples measured in three to six months should yield abnormal results(36).

While UAE is a critical marker for early renal involvement in diabetes, it may not capture all cases. Some patients show decreased glomerular filtration rate (GFR) despite having normal UAE levels. This pattern appears more frequently in women with diabetes, concurrent hypertension, and diabetic retinopathy. Findings from the NHANES III survey (n = 1,197) indicated that 30% of type 2 diabetics had GFR values under 60 ml/min/1.73 m^2 even , absence of microvascular complications(37,38). These observations suggest that normal albumin levels do not necessarily exclude progressive kidney function loss in diabetic patients. Therefore, regular assessment of both estimated GFR (eGFR) and urine albumin levels is essential for comprehensive evaluation.

Assessment of GFR

Several methods exist for measuring GFR. Techniques such as inulin clearance, and radioisotope-based methods using ^{51}Cr-EDTA, ^{125}I-iothalamate, or iohexol provide precise measurements but are complex and not routinely used. Creatinine clearance, based on creatinine, is common employed even its limitations. In practice, eGFR is typically calculated using formulas that integrate serum creatinine along with patient-specific variables like age, sex, ethnicity, body size. The MDRD (Modified Diet in Renal Disease) equation is widely recommended by the NKF and is expressed as: The MDRD equation estimates GFR using serum creatinine, age, sex, and race, while the Cockcroft-Gault formula includes age, weight, and creatinine but is generally less precise. Typically, healthy GFR values fall between 80–130 ml/min/1.73 m^2, dropping by around 10 units each decade past 50(39,40).

Monitoring Renal Function

eGFR is a key role in renal function and routinely monitored in microalbuminuria or macroalbuminuria patients . While some individuals with microalbuminuria maintain relatively stable GFR levels, others may experience a rapid decline. In type 1 diabetes, GFR may decrease at a rate of approximately 1.2 ml/min per month without appropriate therapy. In type 2 diabetes, the detoriate is more different , averaging about 0.5 ml/min/month, with some patients maintaining stable function over time. Those with a consistently declining GFR often show more advanced glomerular damage and poorer glycemic control(41–43).

Once GFR fall under 30 ml/min/1.73 m^2, patients need immediate evaluation and intervention. Renal transplantation at this stage can significantly reduce both morbidity and mortality, highlighting the importance of early detection and timely management.

Prevention of Diabetic Nephropathy

In Patients Without Albuminuria

Preventive efforts in normoalbuminuric individuals with diabetes focus on addressing modifiable risk factors like hypertension, persistent hyperglycemia, tobacco use, also lipid imbalances.

Glycemic Control

Keeping blood sugar levels in check specifically aiming for HbA1c under 7% has been shown to significantly reduce the chance of kidney complications. The DCCT found nearly a 40% drop in microalbuminuria rates, while the UKPDS saw a reduction of about 30% with strict glucose control.Similarly, the Kumamoto Study found that early and sustained glucose management significantly delayed both microalbuminuria and macroalbuminuria(44,45).

Blood Pressure Management

Managing blood pressure is a cornerstone of preventing both cardiovascular and microvascular complications in diabetes. Even when albumin levels are normal, high blood pressure is seen 40%, 70% in type 1, 2 Respectively. The UKPDS showed that dropping systolic pressure by 10 mmHg cut the chance of microalbuminuria by 29%. Aiming for 130/80 mmHg is ideal, and findings from the HOT study revealed that reducing diastolic pressure slightly—from 85 to 81 mmHg—could slash heart risk by half(46,47).

RAAS Inhibition

The benefit of using ACE inhibitors in preventing nephropathy among normotensive type 1 diabetes patients remains debated. However, a study on perindopril showed delayed onset of albuminuria over a 3-year period in this population. In type 2 DM, both ACE inhibitors along with ARBs have demonstrated a protective effect against kidney disease and cardiovascular events. The MICRO-HOPE trial found that 10 mg of Ramipril daily in people over 55 with type 2 diabetes, the chance of kidney disease dropped by 24% and heart-related death by 37% when treated appropriately (48).

Management of Diabetic Nephropathy

Treatment mainly aims to stop microalbuminuria from advancing, slow down kidney damage, and cut the risk of heart complications. While these strategies mirror those used in prevention, they require a more comprehensive and patient-specific approach.

Glycemic Management

Early and consistent glucose regulation play a vital part of decreasing incidence of microvascular complications and improving overall survival(49,50). The long-term advantages of early glycemic control often referred to as the “legacy effect” underscore the importance of maintaining HbA1c below 7%. However, stringent control must be individualized, particularly in elderly patients or those with comorbidities, as it can increase the risk of hypoglycemia. Current guidelines from KDOQI and KDIGO recommend personalized targets based on risk factors and comorbidities(51). While alternative markers such as glycated albumin or fructosamine may be considered in cases of advanced kidney disease, HbA1c remains the gold standard (52).

RAAS Blockade

KDIGO guidelines recommend maintaining blood pressure at or below 120/80 mmHg in diabetic patients, depending on individual risk. ACE inhibitors or ARBs are first-line agents for managing hypertension in diabetic patients, and their dosages can be adjusted to optimize outcomes(52). Their benefits extend to non-hypertensive patients with albuminuria, although use should be tailored. These agents have also shown efficacy in diabetic patients undergoing dialysis or post-transplantation, where they help reduce cardiovascular mortality. Clinical trials such as RENAAL and IDNT supported the role of ARBs in slowing renal decline, while UKPDS confirmed the benefits of blood pressure control. Although studies like HOPE, LIFE, and ALLHAT showed the efficacy of ACEIs in early-stage CKD (eGFR >60), intensive SBP reduction (<120 mmHg) did not show significant additional benefit. Combining ACEIs and ARBs is discouraged due to a heightened risk of acute kidney injury(53–55).

Metformin and GLP-1 Receptor Agonists

According to KDIGO and ADA recommendations, Metformin is considered safe when eGFR is over 30 ml/min/1.73m², but it shouldn't be started if below 45, and must be stopped if it falls under 30. For eGFR between 45 and 60, a dose reduction is advised(56). Hospitalized patients should not receive metformin. Monitoring for vitamin B12 and folate levels is essential with prolonged use. For patients with increased cardiovascular risk, GLP-1RAs or SGLT2 inhibitors are preferred. GLP-1RAs are effective for patients needing improved glucose control and should not be combined with DPP-4 inhibitors (57).

Mineralocorticoid Receptor Antagonists

Activation of mineralocorticoid receptors has been implicated in renal fibrosis and inflammation. Steroidal antagonists like spironolactone and eplerenone have shown benefit in patients of IHD and reduced EF. These drugs also help lower proteinuria, particularly in patients with CKD linked to diabetes(58). Finerenone, a non-steroidal antagonist, has been approved for managing CKD in patients of type 2 DM and provides therapeutic benefit by blocking mineralocorticoid receptor overactivity.

SGLT2 Inhibitors

SGLT2 inhibitors acts by stopping kidneys from reabsorbing glucose in proximal tubules, thereby enhancing urinary glucose excretion, reducing blood pressure, and lowering albuminuria. This is particularly helpful as nephron function declines. These agents also modulate renal hemodynamics, including reduction of glomerular hypertension and activation of hypoxia-inducible factors (HIFs), which promote erythropoietin synthesis. Their benefits extend beyond glucose control—they facilitate weight loss by promoting lipid metabolism and reducing visceral

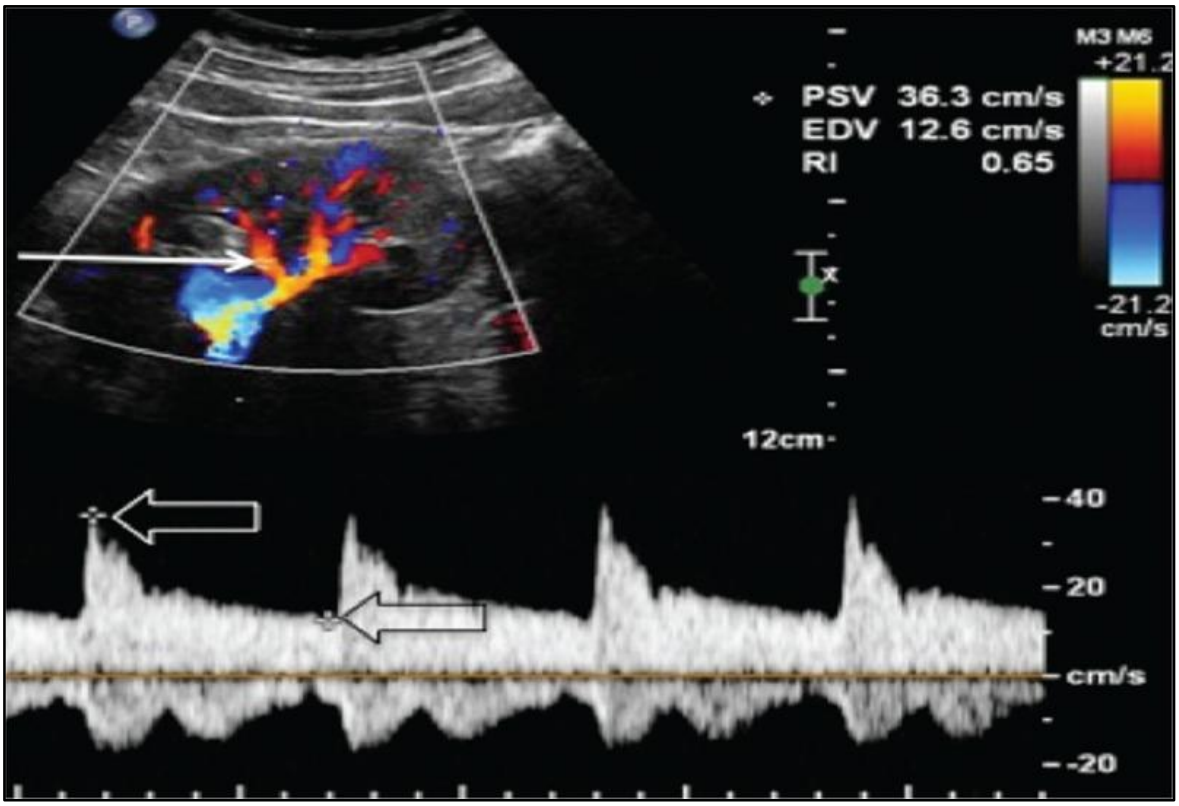
fat. Numerous cardiovascular outcome trials have shown that SGLT2 inhibitors improve renal endpoints, including reductions in microalbuminuria and serious renal complications. Consequently, kidney-specific outcomes are now a central focus in evaluating these drugs(59).

Renal Resistive Index and Monitoring

CKD has traditionally been evaluated using ultrasound combined with Doppler techniques which is capable of detecting both macroscopic vascular abnormalities and subtle disturbances in intrarenal blood flow. Measurements taken from various parts of the renal parenchyma help in identifying both functional and structural renal changes, providing valuable diagnostic and prognostic information.

Technique and Application

Accurate measurement of the renal resistive index (RI) requires standardized imaging protocols. Typically, a high-frequency transducer is used in conjunction with color or power Doppler to localize renal vessels. Resistance to blood flow tends to rise as it moves from the central hilar arteries toward the outer renal branches, which makes it essential to obtain measurements from the interlobar or arcuate arteries for reliable assessment.



Following the acquisition of at least three uniform Doppler waveforms, measurements were performed at different segments of each kidney specifically the upper, middle, and lower poles. Renal resistive index (RRI) was derived using the usual equation: **the difference of peak systolic and end diastolic velocity then divided by peak systolic velocity.**

For each kidney, an average of three readings was used to determine the final value. According to published literature, typical RRI values fall around **0.60 ± 0.01** (mean ± standard deviation), while an RRI of **0.70** is commonly recognized as the higher threshold (60).

While 0.70 is considered normal for adults, children's RI values during their first year of life are often higher. It has been observed that RI rises in healthy older people due to age-related changes in vascular compliance(61).

Initially, a high RI was taken simply as a sign of renal vascular resistance; however, subsequent investigations have shown that it is also It is affected by factors such as vessel elasticity, overall pulse pressure, and cardiac rate and rhythm. Various pathological changes, including tissue interstitial fibrosis and vascular stiffness, can significantly influence RI. The complex and often unusual nature of underlying systems can lead to suboptimal outcomes in distinguishing specific kidney diseases, which may elucidate recent advancements in research regarding its use in assessing cardiorenal risk(62).

Doppler-derived renal resistive index in the chronic renal disease

Elevated RRI is now widely accepted as a sign of systemic haemodynamic stress rather than isolated renal vascular resistance. RRI values greater than 0.70 have been associated to early cardiovascular remodelling, arterial stiffness, and poor renal outcomes. RRI elevation in CKD patients is associated with fast eGFR decrease, progression to ESRD, and greater hospitalisation rates for heart failure(63).

Higher RRI levels have been linked to poor cardiac prognosis like left ventricular hypertrophy and diastolic dysfunction. In type 2 DM, RRI is a non-invasive test that reflects both renal disease severity and cardiovascular risk, indicating its importance in complete risk stratification.

In CKD patients undergoing renal biopsy, higher RRI was associated with increased degrees of interstitial fibrosis and arterial sclerosis, regardless of glomerular abnormalities. This identifies RRI as a possible surrogate measure for histopathologic damage, allowing for early intervention methods even before considerable GFR decline occurs (64)

REVIEW OF RELATED LITERATURE

Jinadu et al. conducted that diabetics with presence or absence of nephropathy had significantly greater mean intra-renal artery resistance indices (RRIs) (0.60 ± 0.04) contrast to controls (0.56 ± 0.04), with p = 0.02. In the patients with diabetes but without nephropathy, high RRIs were associated with glycated hemoglobin (HbA1c) levels (OR 2.81; CI: 1.73-9.03), while in those with nephropathy, hypertension was a predictor of elevated RRIs (OR 3.60; CI: 1.06-12.22). Both groups of diabetes patients—those without and with diabetic nephropathy—often exhibited increased intra-renal artery RRIs, particularly in participants with high HbA1c and hypertension, indicating elevated RRIs (65).

In a study by Tahir et al., the average age of participants 54.06 years, with 58.0% men and 42.0% women among 150 cases. This group's typical diabetes duration was 5.53 years, with an average HbA1c level of 7.58. The renal resistive index for the right kidney was 0.72, and the left kidney had a RI of 0.72, resulting in a total average RI of 0.72 for both kidneys. Individuals with type 2 diabetes showed elevated renal resistance indices, implying that renal Doppler ultrasonography might serve as a simple marker for early detection of diabetics with increase risk of nephropathy, simplifying prompt care (66).

Sistani et al. investigated 100 patients with diabetic nephropathy who visited the nephrology clinic at Al-Ibn Abi-Talib Hospital. Researchers used the most recent lab data from their medical records to quantify USG Doppler RI in conjunction with Additional parameters examined included systolic and diastolic pressures, albumin levels in urine, filtration rate of the kidneys, and HbA1c. Pearson’s correlation analysis showed strong associations between the renal resistive index and systolic pressure (R = 0.75), urinary albumin (R = 0.67), and GFR (R = 0.76). However, meaningful relationship was not found with diastolic pressure (P = 0.45, R = 0.32) or

HbA1c levels (P = 0.56, R = 0.43). In diabetic nephropathy patients, higher SBP, albumin excretion (as evidenced by microalbuminuria), and disease severity were all related with raised RI values, as well as a reduction in GFR (67).

In a study by Delsart et al., involving 236 participants—half of whom were male—the average HbA1c was 8.1 ± 1.7% (equivalent to 65 ± 13.6 mmol/mol), and sr creat averaged 8 mg/L[68]. Blood pressure measurements over 24 hours showed mean systolic, diastolic, and pulse pressures of 133.4 ± 16.7 mmHg, 76.5 ± 9.4 mmHg, and 56.9 ± 12.4 mmHg, . The renal resistive index was typically around 0.7 [range: 0.6–0.7], a level consistent with expected values (68).

Maksoud's study included 63 girls and 37 men, reported as 13.6 years, with a standard deviation of ±2.5. (10-19) and illness duration of 8.86 ± 2.26 years (5-13). Significant relationships were discovered between successive increases in RI and early pubertal phases, increased blood HbA1c levels above 7.5%, and illness duration of more than ten years. However, RI had no significant association with weight, gender, height, lipid profiles, or blood pressure. Participants with RI greater than 0.58 had a higher incidence of microvascular sequelae, such as nephropathy and sensory neuropathy. As a result, RI may be a useful supplementary test for detecting functional changes in the renal vasculature during the early stages of diabetes with nephropathy (69).

Shirin et al. conducted a research in which the majority of patients were in their sixth decade, aged 38 to 65 years, with participants with mean age 52.66 years. Of the total, 54.7% were male diabetics, resulting in a male to female ratio of approximately 1.2 to 1.

A total of 73.6% of diabetes patients had resistive indices less than 0.7, with an average of 0.71. Positive associations were found between serum creatinine, albuminuria, and the resistive index. The resistive index value obtained from Duplex Doppler ultrasonography is an effective diagnostic tool for diagnosing kidney failure in diabetic nephropathy patients. Furthermore, it has a high connection with serum creatinine and albuminuria, two laboratory tests used to diagnose diabetic kidney disease (70).

Li et al. studied 137 individuals with NDKD, as well as an extra number of patients. The resistive indices of the DKD and NDKD groups differed significantly (0.70 vs. 0.63, $p = 0.001$). The resistive index threshold for predicting DKD was set at 0.66, with a sensitivity of 69.2% and specificity of 80.9%. Regression analysis showed strong links between high RI (0.66), long-standing diabetes (over 5 years), poor glycemic control (HbA1c $\geq$7%), increased BMI, and the presence of retinopathy. As a consequence, the scientists used these findings to create a novel diagnostic model(62) .

MATERIALS & METHODS

MATERIAL AND METHODS

This cross-sectional study carried out in Department of General Medicine at R.L. Jalappa Hospital, Kolar, over a period spanning from May 2023 to December 2024. The study began only after receiving ethics committee approval, and every participant provided signed consent before inclusion. Both cases and controls before their enrollment in the research.

Sample size

The sample size estimated using the observed differences in proportion to higher resistive indices among diabetes patients without nephropathy, those with nephropathy, and healthy controls. According to Jinadu et al., the proportion of diabetic patients without nephropathy with an increased resistive index was 30%, 43.4% with diabetic nephropathy, and 12.5% with healthy controls. Putting these values into the following formula:

$N = 3 (Z_{\alpha/2} + Z_{\beta})2 P (1-P) (p1 –p2)2$

- Where ,
- $Z_{\alpha/2}= Z_{0.05/2}=Z_{0.025}$ = 1.96 at type 1 error of 5%
- $Z_{\beta}=Z_{0.20}$= 0.84= At 80% power
- p1 –p2= Difference in proportion in the two different groups with Max and Min value = 30.9%
- P= Pooled prevalence = [Proportion in Healthy control (p1) + Proportion in DM without DN Group (p2) + Proportion in DM with DN Group (p3)]/3 = [30 + 43.4 + 12.5]/3 = 28.6
- $N = 3 \times 28.6 \times 71.4 (1.96 + 0.84)^2/30.9 \times 30.9$ = 34 in each group.

Inclusion Criteria

Participants were selected based on the American Diabetes Association's diagnostic framework for diabetic nephropathy, which includes:

- Constantly elevated urinary albumin levels—specifically above 300 mg per day or 200 µg per minute—verified at least twice over a 3 to 6-month period.
- A gradual drop in glomerular filtration rate, reflecting continued deterioration of kidney function.
- Elevated arterial blood pressure, commonly observed in conjunction with the above parameters.

This syndrome is primarily identified by sustained albuminuria, which must be validated through repeated testing to confirm its chronicity.

Exclusion criteria

1. Non-diabetic kidney disease
2. Renal artery stenosis
3. Active urinary tract infection
4. Urinary tract abnormalities
5. Hypotension
6. Bradycardia
7. Hemoglobinopathy.

Methodology

The study population comprising of patients are divided in to 3 groups. i.e.

Study population comprising of patients are divided into 3 groups i.e

Group 1: Diabetes Mellitus with Diabetic Kidney Disease

Group 2: Diabetes Mellitus without complications

Group 3: Age and sex matched healthy individuals

History and physical examination will be done in all the patients according to prefixed proforma. All the patients were subjected to various investigation like

- Renal function tests by semi-automated clinical chemistry analysis.
- Renal arterial doppler ultrasonography.

Data collected from participants using pre-test questionaire, and information taken was lifestyle, socio-demographic details ,history of DM , medical history, medication history and any features of kidney disease.

- anthropometry was measured using standard stadiometers and weighing scales.
- Urine (10 ml) collected for micro albuminuria.
- Blood collected to measure serum creatinine, glycated haemoglobin (HbA1c) & FLP analysis.
- The eGFR calculated using CKD-EPI equation.
- Participants underwent renal Doppler sonography ,to minimise bowel gas shadows ,patient was kept overninght fasting or eight hour fasting
- PSV and EDV was obtained from the interlobar arteries bilateral using M-mode analysis.
- 3 to 5 reproducible wave forms from either kidney was obtained, RI from this waveforms were Doppler examinations carried out.

Statistical analysis

Data entry was done using Microsoft Excel and analyzed with SPSS version 20. Qualitative variables were summarized as proportions and illustrated using bar charts. Quantitative variables described using means and standard deviations. To assess significance, ANOVA was used for numerical variables, while categorical data were analyzed using chi-square method. A p-value under 0.05 indicated statistical relevance.

RESULTS

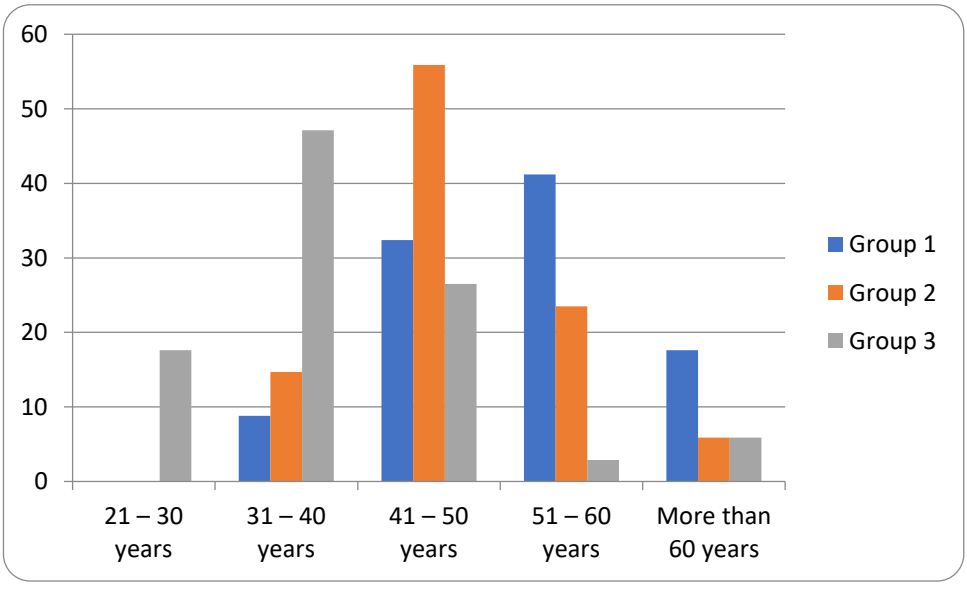
RESULTS

Table 1. study groups according to age group

Age group	Group 1 n (%)	Group 2 n (%)	Group 3 n (%)
21 – 30 years	0	0	6 (17.6)
31 – 40 years	3 (8.8)	5 (14.7)	16 (47.1)
41 – 50 years	11 (32.4)	19 (55.9)	9 (26.5)
51 – 60 years	14 (41.2)	8 (23.5)	1 (2.9)
More than 60 years	6 (17.6)	2 (5.9)	2 (5.9)
Total	34 (100)	34 (100)	34 (100)

χ^2 Value=42.801 df=8 p value, Sig=0.000, Sig

Chart 1. study groups according to age group



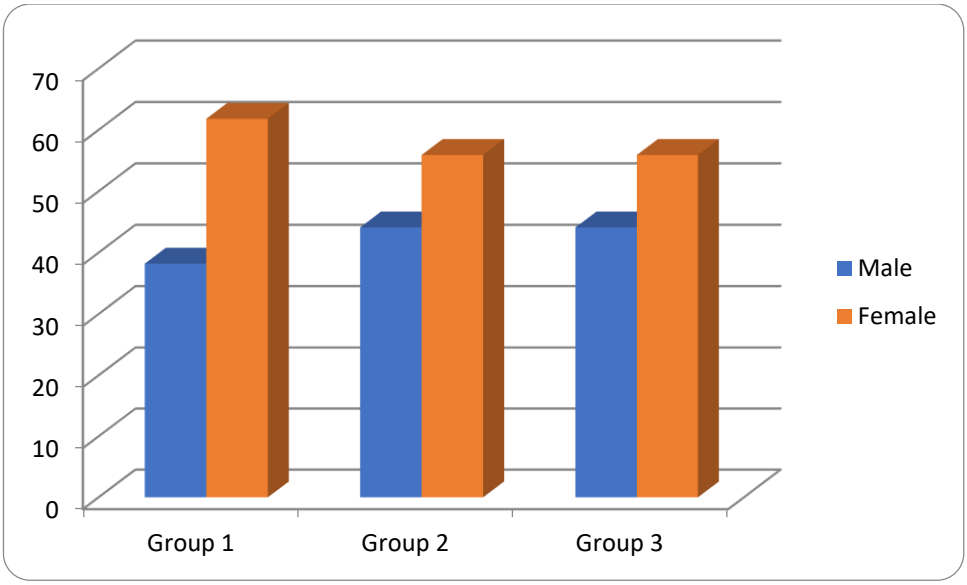
About 41.2% of cases were age in between 51 – 60 years, about 55.9% of the cases were 41 – 50 years and 47.1% of cases in between 31 – 40 years. This difference was statistically significant.

Table 2. study groups according to Sex

Sex	Group 1 n (%)	Group 2 n (%)	Group 3 n (%)
Male	13 (38.2)	15 (44.1)	15 (44.1)
Female	21 (61.8)	19 (55.9)	19 (55.9)
Total	34 (100)	34 (100)	34 (100)

χ^2 Value=0.322 **df=2** **p value, Sig=0.851, NS**

Chart 2. study groups according to Sex

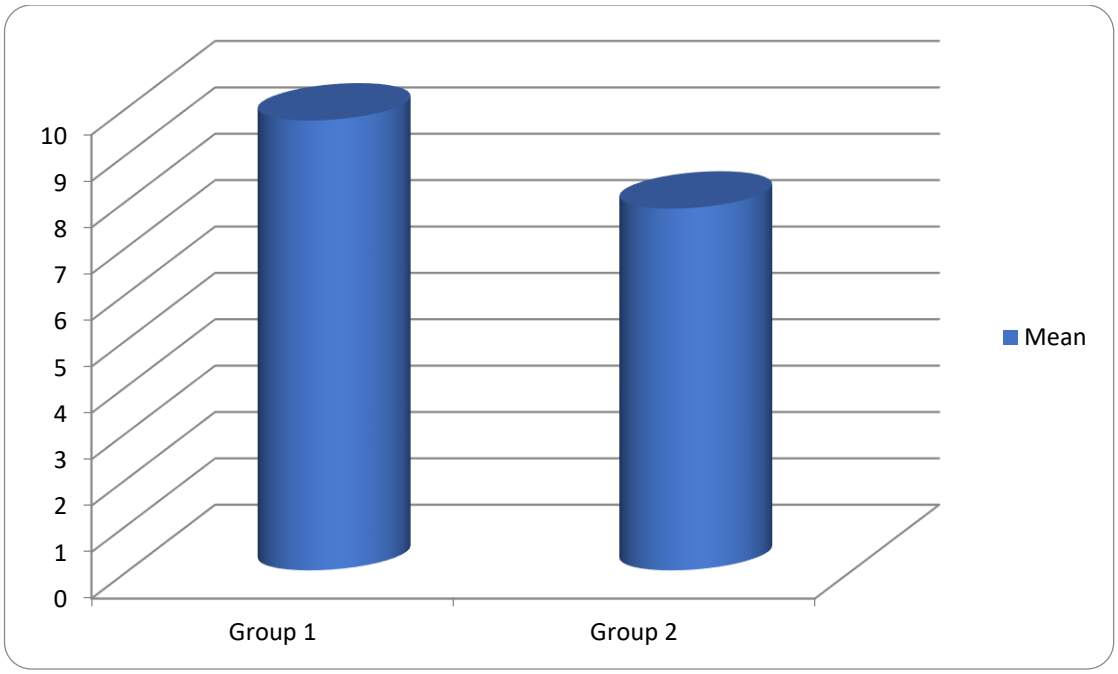


Majority of the cases were females in all three groups . This difference was not statistically significent.

Table 3. study groups according to duration of diabetes mellitus

Duration of diabetes mellitus	Group 1	Group 2	T value	P value,
Mean ± SD	9.7 ± 6.7	7.8 ± 5.4	1.249	0.216, NS

Chart 3. study groups according to duration of diabetes mellitus



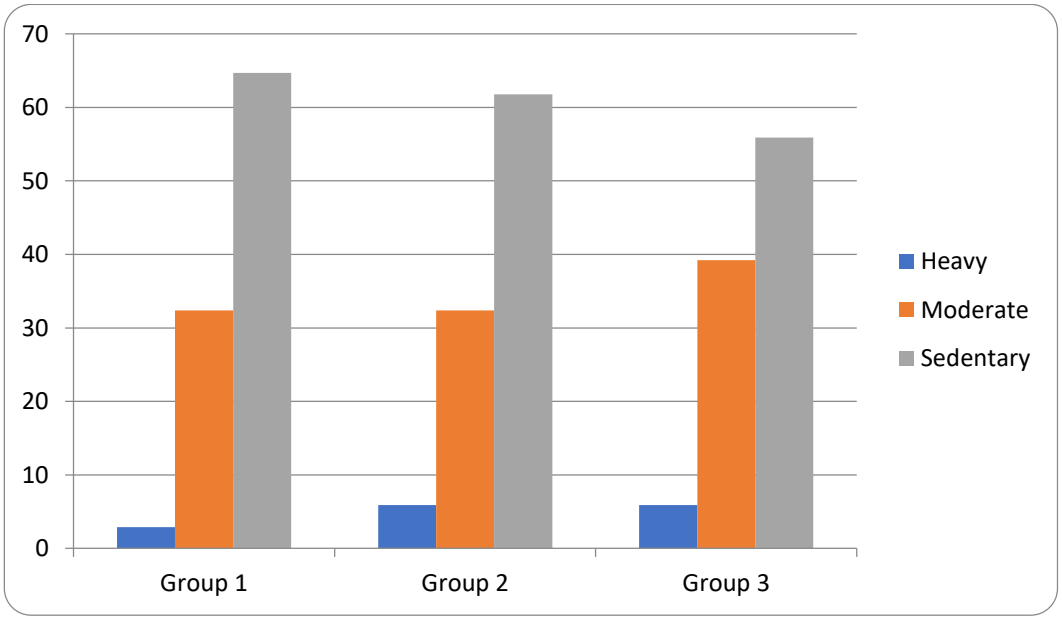
Average duration of diabetes in group 1 was 9.7 years and group 2 was 7.8 years. This difference was not statistically significant.

Table 4. study groups according to type of work

Type of work	Group 1 n (%)	Group 2 n (%)	Group 3 n (%)
Heavy	1 (2.9)	2 (5.9)	2 (5.9)
Moderate	11 (32.4)	11 (32.4)	13 (39.2)
Sedentary	22 (64.7)	21 (61.8)	19 (55.9)
Total	34 (100)	34 (100)	34 (100)

χ^2 Value=0.954 **df=4** **p value, Sig=0.931, NS**

Chart 4. study groups according to type of work



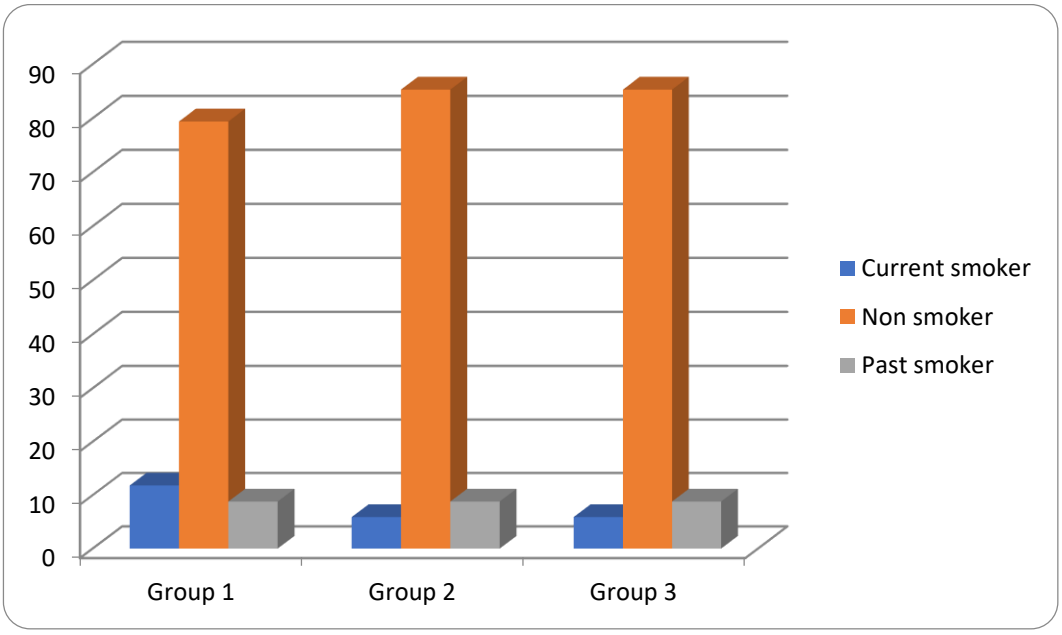
Sedentary workers accounted for approximately 64.7% of cases in group 1, 61.8% in group 2, and 55.9% in group 3. This difference was not statistically significant.

Table 5. Distribution of the study groups according to Smoking

Smoking	Group 1 n (%)	Group 2 n (%)	Group 3 n (%)
Current smoker	4 (11.8)	2 (5.9)	2 (5.9)
Non smoker	27 (79.4)	29 (85.3)	29 (85.3)
Past smoker	3 (8.8)	3 (8.8)	3 (8.8)
Total	34 (100)	34 (100)	34 (100)

χ^2 Value=1.094 **df=4** **p value, Sig=0.895, NS**

Chart 5. study groups according to Smoking



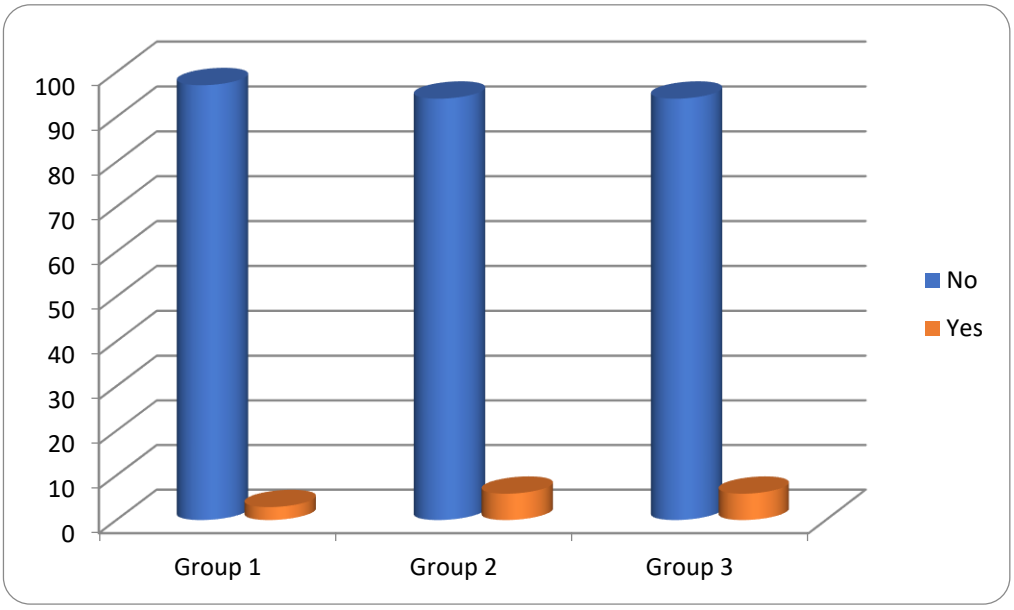
About 11.8% of the patients in Group 1, 5.9% in Group 2, and 5.9% in Group 3 were active smokers. This difference was not statistically significant.

Table 6. study groups according to alcohol consumption

Alcohol consumption	Group 1 n (%)	Group 2 n (%)	Group 3 n (%)
No	33 (97.1)	32 (94.1)	32 (94.1)
Yes	1 (2.9)	2 (5.9)	2 (5.9)
Total	34 (100)	34 (100)	34 (100)

χ^2 Value=0.421 df=2 p value, Sig=0.81, NS

Chart 6. study groups according to alcohol consumption



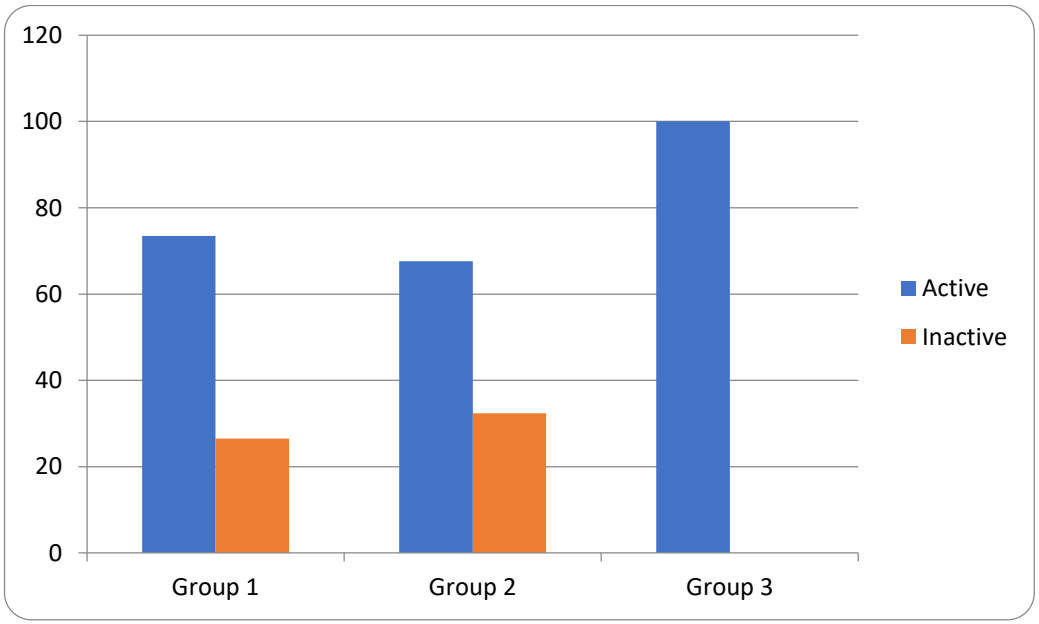
About 2.9% cases in group 1, 5.9% cases in group 2 , 5.9% cases in group 3 were alcoholics. This difference was not statistically significant.

Table 7. study groups according to physical activity

Physical activity	Group 1 n (%)	Group 2 n (%)	Group 3 n (%)
Active	25 (73.5)	23 (67.6)	34 (100.0)
Inactive	9 (26.5)	11 (32.4)	0
Total	34 (100)	34 (100)	34 (100)

χ^2 Value=12.812 df=2 p value, Sig=0.002, Sig

Chart 7. study groups according to physical activity



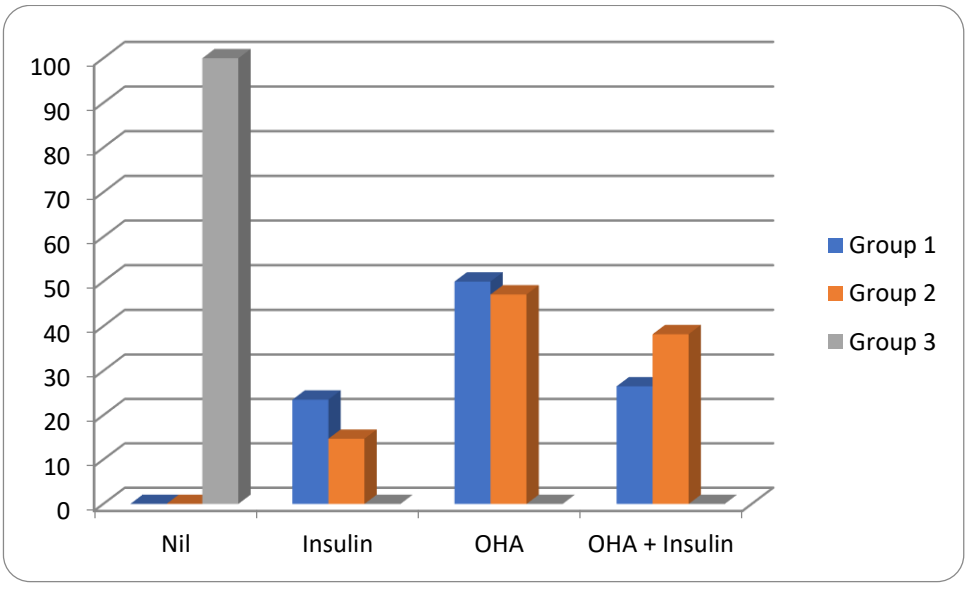
About 73.5% cases in group 1, 67.0% cases in group 2 , all the cases in group 3 were physically active. This difference was statistically significant.

Table 8. study groups according to Drug used

Drug	Group 1 n (%)	Group 2 n (%)	Group 3 n (%)
Nil	0	0	34 (100.0)
Insulin	8 (23.5)	5 (14.7)	0
OHA	17 (50.0)	16 (47.1)	0
OHA + Insulin	9 (26.5)	13 (38.2)	0
Total	34 (100)	34 (100)	34 (100)

χ^2 Value=104.175 df=6 p value, Sig=0.000, Sig

Chart 8. study groups according to Drug used

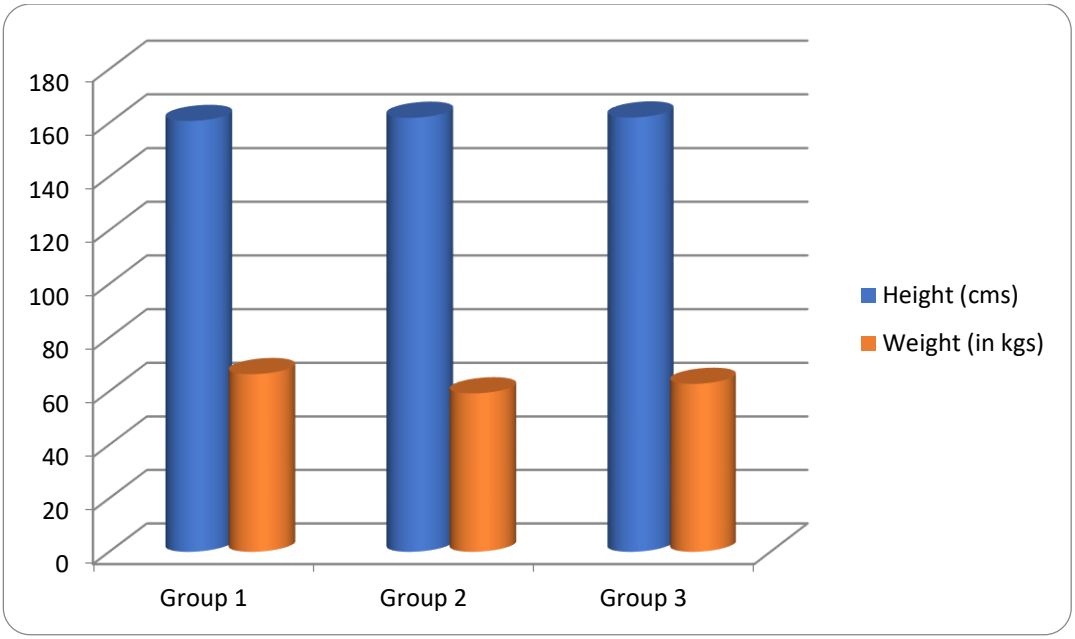


About 50.0% cases in group 1 and 47.1% cases in group 2 were using oral hypoglycemic agents. This difference was statistically significant.

Table 9. study groups according to Anthropometry

Anthropometry Mean ± SD	Group 1	Group 2	Group 3	F value	P value, sig
Height (cms)	160.7 ± 4.6	161.88 ± 5.52	162.0 ± 4.5	0.718	0.490, NS
Weight (in kgs)	66.44 ± 6.01	59.23 ± 6.95	62.76 ± 7.14	9.766	0.000, Sig

Chart 9. study groups according to Anthropometry

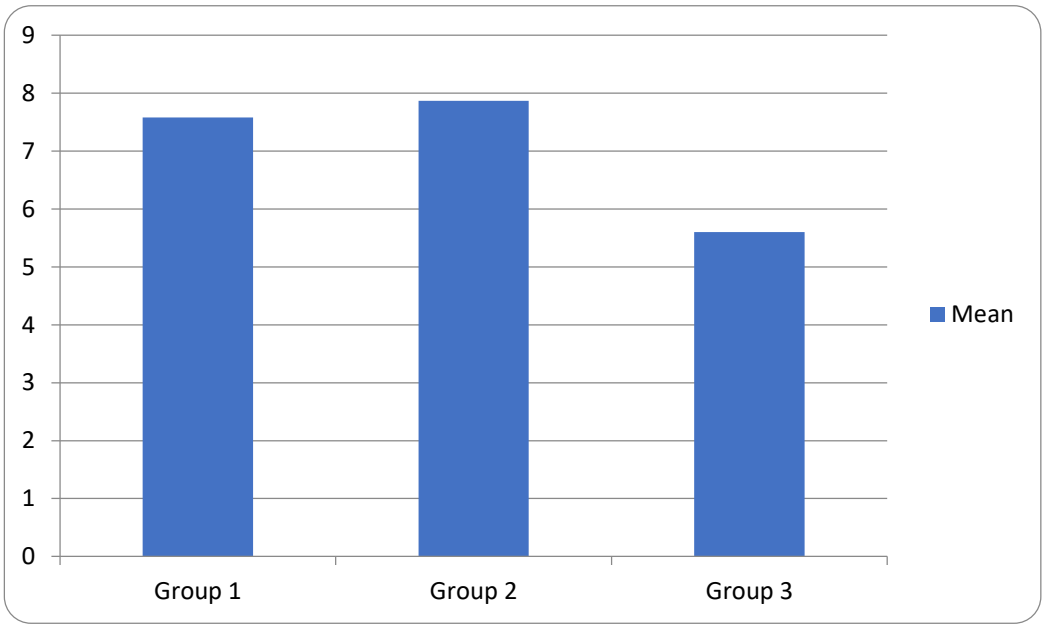


Group 1 had a mean height of 160.7 cm, compared to 161.88 cm in group 2 and 162.0 cm in group 3. These differences showed no statistical significance. The mean weight in group one was 66.44 kgs, in group two it was 59.23 kgs, and in group three it was 62.76 kgs. The difference was statistically significant.

Table 10. study groups according to HbA_{1c} levels

HbA_{1c}	Group 1	Group 2	Group 3	F value	P value, sig
Mean ± SD	7.58 ± 1.2	7.87 ± 1.31	5.6 ± 0.4	46.577	0.000, Sig

Chart 10. study groups according to HbA_{1c} levels

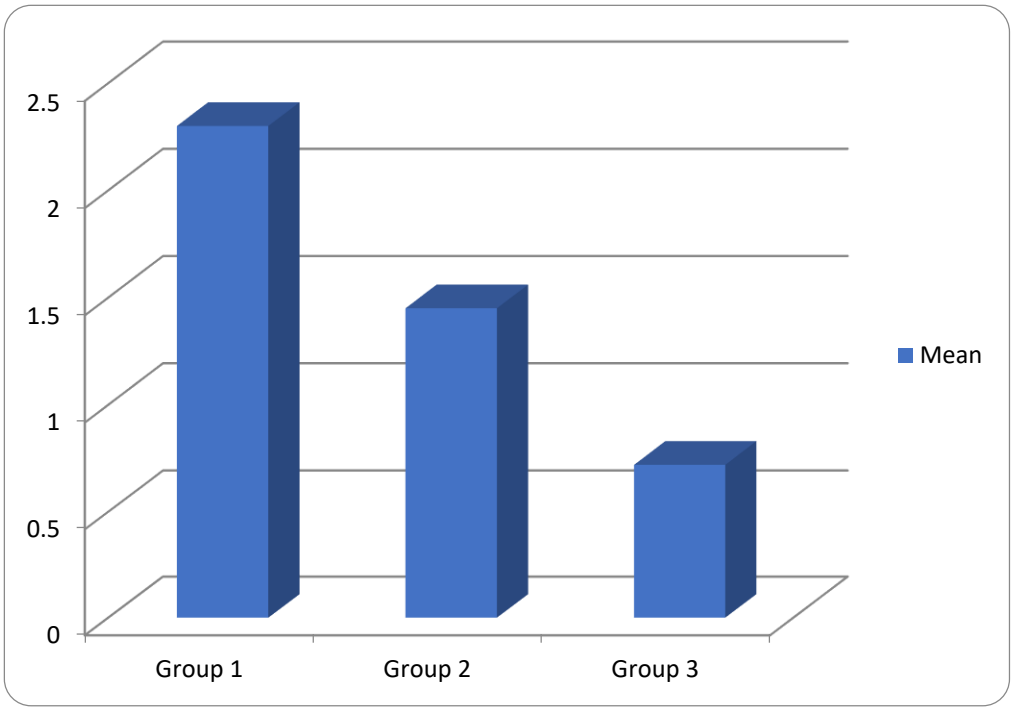


The mean HbA1c readings in group 1 were 7.58%, 7.87% in group 2, and 5.6% in group 3. Difference between the three groups is a statistically significant.

Table 11. study groups according to Serum creatinine

Serum creatinine	Group 1	Group 2	Group 3	F value	P value, sig
Mean ± SD	2.3 ± 0.66	1.25 ± 0.43	0.72 ± 0.6	65.195	0.000, Sig

Chart 11. study groups according to Serum creatinine

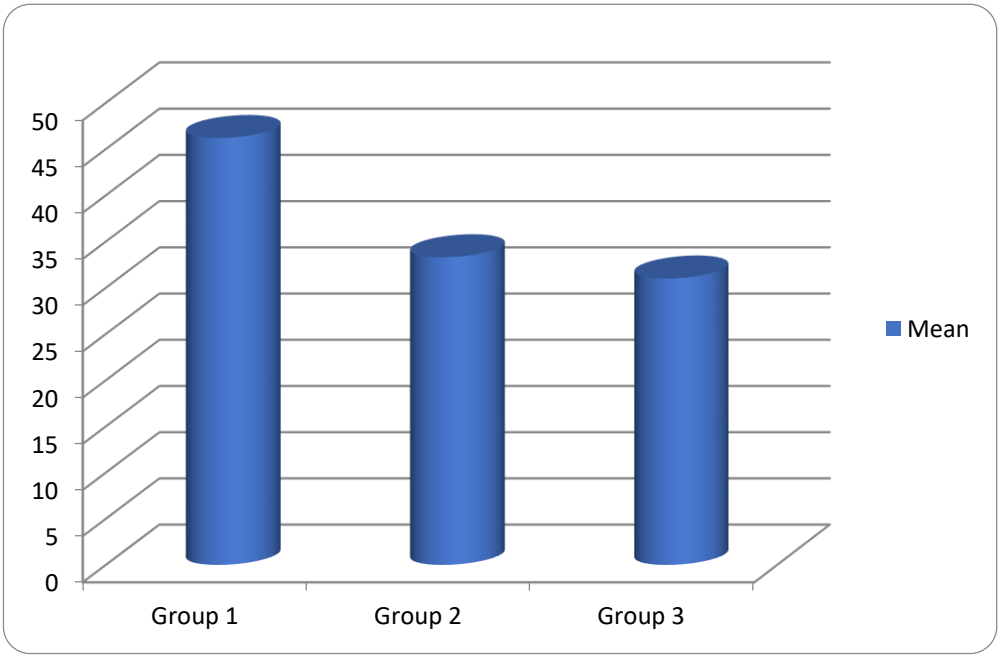


Mean serum creatinine level in group 1 was 2.3 mg/dl, 1.45 mg/dl in group 2 & 0.72 mg/dl in group 3. This difference was statistically significant.

Table 12. study groups according to BUN levels

BUN	Group 1	Group 2	Group 3	F value	P value, sig
Mean ± SD	46.2 ± 28.1	33.3 ± 20.4	31.0 ± 7.8	5.395	0.000, Sig

Chart 12. study groups according to BUN levels



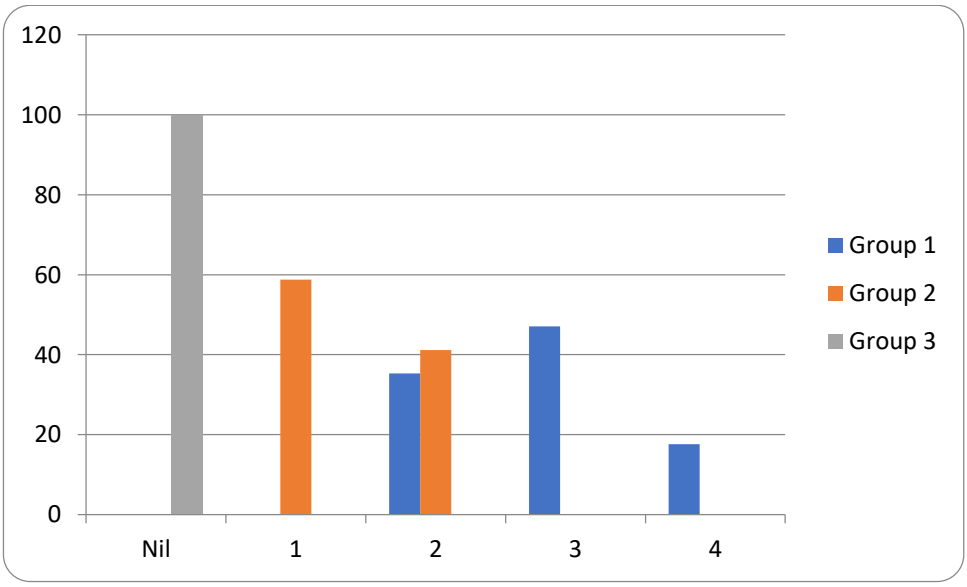
Mean BUN level in group 1 was 46.2 g/dl, 33.3 mg/dl in group 2 & 31.0 mg/dl in the group 3. This difference was statistically significant.

Table 13. study groups according to Stage of kidney disease

Stage of Kidney disease	Group 1 n (%)	Group 2 n (%)	Group 3 n (%)
Nil	0	0	34 (100.0)
1	0	20 (58.8)	0
2	12 (35.3)	14 (41.2)	0
3	16 (47.1)	0	0
4	6 (17.6)	0	0
Total	34 (100)	34 (100)	34 (100)

χ^2 Value=165.231 df=8 p value, Sig=0.000, Sig

Chart 13. study groups according to Stage of kidney disease

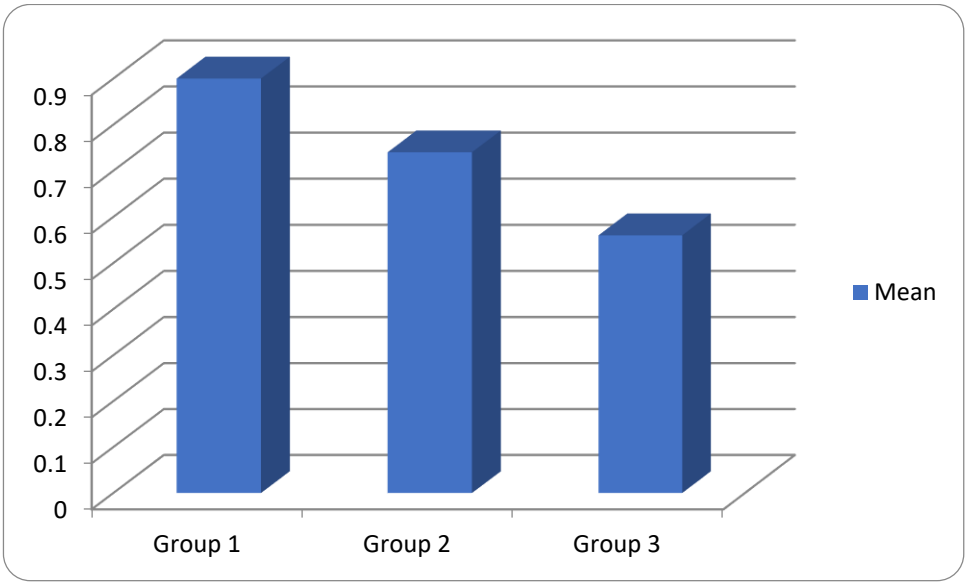


Stage 3 kidney disease was seen in 47.1% of the cases in group 1 and stage 1 kidney disease was present in group 2. This Difference between the three groups is statistically significant.

Table 14. study groups according to renal resistance index

RRI	Group 1	Group 2	Group 3	F value	P value, sig
Mean ± SD	0.9 ± 0.15	0.74 ± 0.11	0.56 ± 0.07	70.346	0.001, Sig

Chart 14. study groups according to renal resistance index

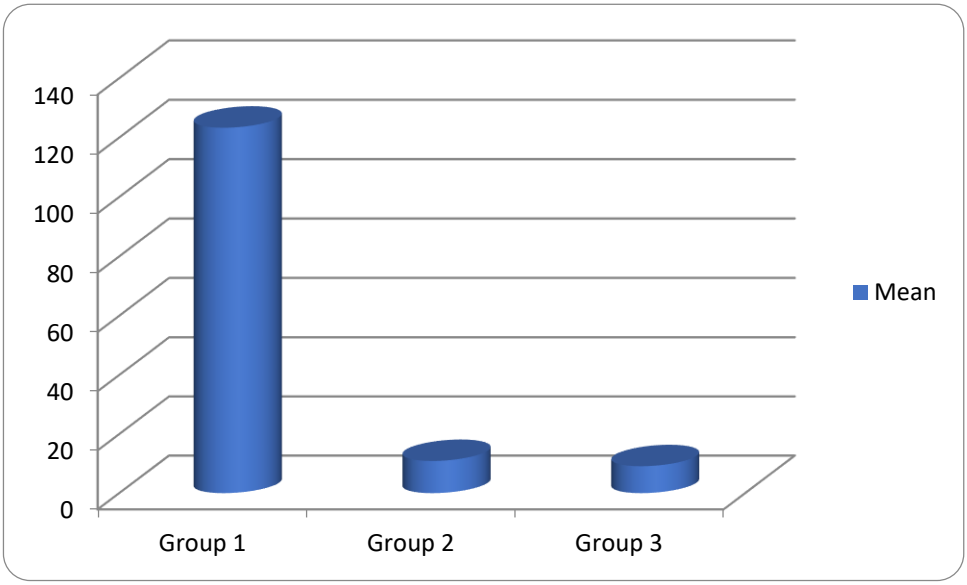


Mean renal resistive index in group 1 was 0.9, group 2 was 0.74 and 0.56 in group 3. Difference was statistically significant.

Table 15. study groups according to UACR levels

UACR	Group 1	Group 2	Group 3	F value	P value, sig
Mean ± SD	123.32 ± 87.9	10.9 ± 7.54	9.12 ± 4.13	55.95	0.000, Sig

Chart 15. study groups according to UACR level

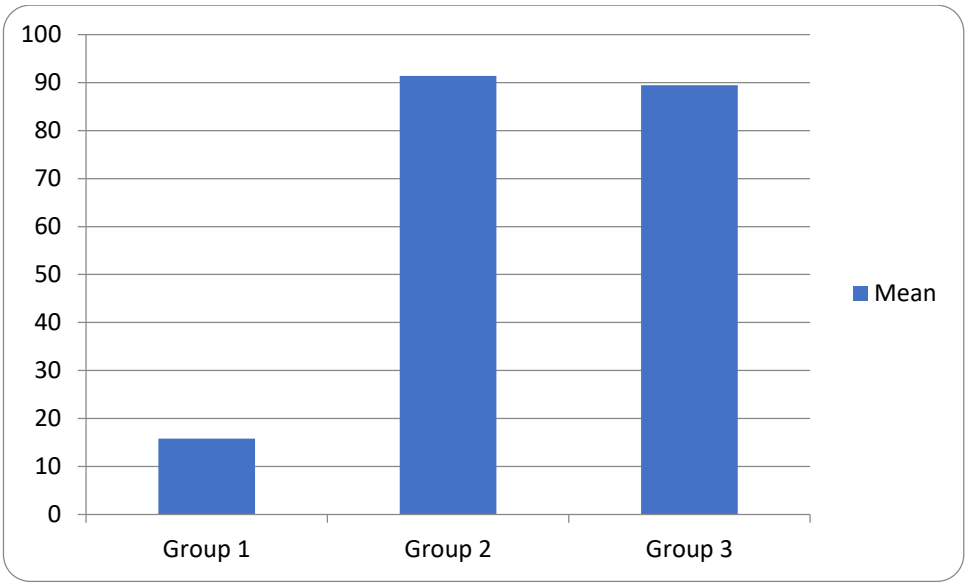


Mean UACR level in group 1 - 123.32, in group 2 - 10.9 & 9.12 in group 3. difference was statistically significant.

Table 16. study groups according to EGFR levels

EGFR	Group 1	Group 2	Group 3	F value	P value, sig
Mean ± SD	15.8 ± 9.4	91.4 ± 4.9	89.46 ± 4.36	1437.157	0.000, Sig

Chart 16. study groups according to EGFR levels



Mean EGFR level in group 1 was 15.8, group 2 was 91.4 and in group 3 it was 89.46. This difference was statistically significant.

DISCUSSION

DISCUSSION

These include prevalent chronic conditions such as hypertension and T2DM, both exert additional pressure on the kidneys over time. Other factors include autoimmune diseases (e.g., lupus), advancing age, specific racial and ethnic groups, a familial history of kidney disease, previous occurrences of acute kidney injury, and anatomical abnormalities in renals or urinary tract. The global impact of CKD has escalated considerably over recent decades. In 1990, it was the 27th cause of death worldwide. CKD is a long-term detoriate in kidney function caused by underlying health conditions and anatomical defects. It is characterised by progressive kidney deterioration that might eventually lead to ESRD, which requires dialysis or kidney transplantation for survival. Notably, CKD is more than just a kidney problem; it is also a substantial risk factor for cardiovascular problems, including heart attacks, strokes, and premature death. Several risk factors have been discovered that influence the start and progression of CKD. This rising trend reflects not just improved diagnostic skills, but also an increase in risk factors in older populations. Access to CKD therapy, particularly in the latter stages, varies greatly by area(72). The bulk of treatment options, including dialysis and transplantation, are available in high-income countries, which tend to have older populations and better sophisticated healthcare systems. Conversely, low- and middle-income nations generally fail to provide effective CKD care, resulting in higher rates of untreated ESRD and related death. In disadvantaged areas, relatively few people obtain treatment, owing to a lack of patient acceptability of renal replacement treatments (RRTs), which are becoming increasingly common(73) . The global incidence of T2DM has been steadily increasing, imposing a significant health and economic burden on individuals and healthcare systems alike. The condition typically begins with slight elevation in urinary albumin excretion referred to as microalbuminuria and is accompanied by a gradual reduction in the GFR. These early manifestations are clinically important, as they are closely linked to elevation of advanced

renal dysfunction as well as a heightened risk of CV events and MACE in both diabetic and non-diabetic populations. In clinical settings, renal damage is primarily assessed through two key parameters: eGFR and the degree of albuminuria. These indicators are essential for the diagnosis, classification, and ongoing monitoring of CKD(74). Emerging research suggests that arterial stiffness and PWV is currently the top non-invasive method used to assess aortic stiffness can predict the onset of microalbuminuria and correlate with yearly changes in eGFR in CKD patients. This association is greater in CKD individuals than normal population, indicating a unique vascular-renal interaction in the setting of the illness. Recent studies have underlined the need of introducing treatment strategies in the early stages of kidney injury, maybe before proteinuria starts. Early therapy for microvascular and vascular abnormalities may assist to avoid or dramatically decrease the evolution of diabetic nephropathy(75). These first vascular abnormalities, which are generally asymptomatic, have been related to organ failure and may serve as early warning signs of cardiovascular problems, particularly I increased risk patients such as diabetics. The Renal Resistive Index (RI) is a diagnostic measure developed from duplex Doppler ultrasonography that evaluates blood flow dynamics inside the renal arteries. It reflects resistance in the renal vascular system and provides information on both dynamic and structural changes in intrarenal arteries. The RI has garnered interest because of its association with vascular changes such as renal arteriolosclerosis, as well as increased cardiovascular risk. Normal RI values in people typically range from 0.47 to 0.70, with modest differences (5% to 8%) observed between the two kidneys. An high RI is commonly regarded as an indication of renal vascular or interstitial injury, and it has been associated to rapid elevation of kidney disease, particularly in those with T2DM. In setting diabetic kidney disease, higher RI values have been linked to decreased renal function and an increased risk of progressing to more severe stages of CKD. Despite its promise as a prediction tool, there is ongoing dispute concerning its effectiveness in guiding treatment

options. While RI may be a useful sign of underlying renal dysfunction, its therapeutic implications are unknown, and further study is needed to better characterise its use in clinical settings .

Age Distribution

In this study, the highest proportion of participants 41.2% aged between 51- 60 years, follows by 55.9% in the 41–50 age group, and 47.1% in the 31–40 age range. The age-related differences were statistically significant. These findings are in line with Sistani et al., who reported an average participant age of 51 years(75). Similarly, Li et al. documented mean ages of 52.04 years in patients with NDKD and 51.32 years in those with DKD(. In a separate investigation by Jenewari et al., the mean age was noted to be 55.9 years(78).

Gender Distribution

Female participants predominated across all three study groups, although the variation was not significant. In contrast, Sistani et al. found a male predominance (75), a pattern also reported by Li et al. in both DKD and NDKD categories. Jenewari et al. similarly observed a higher representation of males(76).

Duration of Diabetes

The average duration of diabetes in group 1 was 9.7 years, compared to 7.8 years in group 2; was not significant. Li et al. stated average diabetes duration of 28.1 months in NDKD and 94.6 months in DKD(77). Jenewari et al. reported a mean duration of 9.6 years(78).

Occupational Status

A sedentary lifestyle was common among participants, accounting for 64.7% of group 1, 61.8% of group 2, and 55.9% of group 3. These differences did not reach statistical significance.

Smoking and Alcohol Use

In terms of smoking, 11.8% of group 1, and 5.9% in both group 2 and group 3 were active smokers, with no statistically significant differences. Regarding alcohol consumption, usage was reported by 2.9% in group 1, and 5.9% in both group 2 and 3, again without significant variation across groups.

Physical Activity

Engagement in physical activity was reported by 73.5% of group 1, 67% of group 2, and all participants (100%) in the group 3. This difference found as statistically significant.

Medication Usage

Among study participants, 50% of group 1 and 47.1% of group 2 were on oral hypoglycemic agents. The variation observed was statistically significant.

Anthropometric Measurements

The average height was similar across all groups 160.7 cm, 161.88 cm, and 162.0 cm with no significant difference in respective groups. However, weight differences were notable: group 1 had a mean weight of 66.44 kg, group 2 had 59.23 kg, and group 3 had 62.76 kg. This difference was statistically significant.

Glycemic Control (HbA1c)

Mean HbA1c levels were 7.58%, 7.87%, and 5.6% in respective groups, showing a statistically significant difference across all groups. Li et al. reported an average HbA1c of 7.32% in NDKD and 8.01% in DKD patients(77).

Serum Creatinine

The average sr creat level was 2.3 mg/dL, 1.45 mg/dL, and 0.72 mg/dL in respective groups. These differences were statistically significant. Li et al. noted serum creatinine levels of 102.41 µmol/L in NDKD and 137.5 µmol/L in DKD(77).

Blood Urea Nitrogen (BUN)

BUN levels averaged 46.2 mg/dL, 33.3 mg/dL, and 31.0 mg/dL in respective groups, with statistically significant differences among the groups. Li et al. found BUN values of 372.42 µmol/L and 381.0 µmol/L in NDKD and DKD, respectively(77).

Kidney Disease Stage

Nearly 47.1% of group 1 had stage 3 chronic kidney disease, while group 2 primarily included patients with stage 1 disease. This difference was statistically significant. Sistani et al. reported that approximately 37% of their study population presented with stage 3 diabetic nephropathy(75).

Renal Resistive Index (RRI)

The mean RRI was 0.9, 0.74, and 0.56. These findings were statistically significant. Sistani et al. reported an average RRI of 0.76, with higher values noted in advanced-stage kidney disease. Li et al. found an RRI of 0.63 in NDKD and 0.7 in DKD(77) while Jenewari et al. reported an average RRI of 0.6 and observed elevated RRI in 25.3% of participants[78].

Urine Albumin-to-Creatinine Ratio (UACR)

UACR levels averaged 123.32 mg/g, 10.9 mg/g, and 9.12 mg/g in respective groups with significant variation across all groups. Jenewari et al. documented a mean UACR of 153.65 mg/g.

Estimated Glomerular Filtration Rate (eGFR)

Group 1 had a mean eGFR of 15.8 ml/min/1.73 m^2, compared to 91.4 ml/min/1.73 m^2 in group 2 and 89.46 ml/min/1.73 m^2 in group 3. The differences were statistically significant. Li et al. observed an average eGFR of 82.67 ml/min in NDKD and 61.32 ml/min in DKD. Jenewari et al. reported a mean eGFR of 79.18 ml/min/1.73 m^2 .

CONCLUSION

CONCLUSION

This study evaluated the renal resistive index (RRI) across three groups: diabetic patients with nephropathy, those without nephropathy and healthy controls. findings revealed that both groups of diabetic individuals exhibited higher RRI values compared to the control group, with the highest indices observed in those with established diabetic kidney disease. Additionally, diabetic nephropathy was associated with elevated clinical markers such as reduced eGFR, increased UACR, and more advanced stages of renal impairment. These results highlights the potential of RRI as an first indicator of renal involvement in diabetes. While the present study offers valuable contributions to understanding the role of RRI in diabetic kidney disease, further large-scale and longitudinal research is warranted to confirm its prognostic utility and broader clinical application ., after 24 hours, the absence of elevated LAR strongly predicts ICU survival.

LIMITATIONS

LIMITATIONS

The limitations of our study include done in a single hospital setting, There can be individual variability in RI, and it can't differentiate early and late kidney changes..

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ANNEXURES

PROFORMA

Study of Renal Resistive Index in Diabetic kidney Disease

NAME	
AGE	
GENDER	
DATE OF ADMISSION	
PRESENTING COMPLIANTS	
Is the patient already a known case of Diabetes	
If Yes then details about treatment history	
Treatment history	
DURATION OF STAY IN HOSPITAL	
TIMES OF READMSSION FOR THE SAME COMPLAINTS	
COMORBIDITIES	

INVESTIGATIONS	**1) SERUM ELECTROLYTES AND RFT** **2)RENAL RESISTIVE INDEX -**

DATE	UREA	CREAT	SODIUM	POTASSIUM

INFORMED CONSENT FORM

Date:

I, Mr/Mrs _____, have been explained in my own vernacular language that I/We will be included in **Study of Renal Resistive Index in Diabetic kidney Disease,** hereby I/We give my valid written informed consent without any force or prejudice for recording the observations of haematological and clinical parameters . The nature and risks involved have been explained to me, to my satisfaction. I have been explained in detail about the study being conducted. I have read the patient information sheet and I have had the opportunity to ask any question. Any question that I have asked, have been answered to my satisfaction. I provide consent voluntarily to allow myself / my relative as a participant in this research. I hereby give consent to provide history, undergo physical examination, undergo the procedure, undergo investigations and provide its results and documents etc to the doctor / institute etc. For academic and scientific purpose the operation / procedure, etc may be video graphed or photographed. All the data may be published or used for any academic purpose.

_____

Name of Patient/Guardian

(Relation with patient)

_____

(Signature of Patient / Attendant)

(Signature & Name of Research doctor)

ಮಾಹಿತಿ ಒಪ್ಪಿಗೆ ನಮೂನೆ

ಶ್ರೀ/ಶ್ರೀಮತಿ _____ ಅವರನ್ನು ನನ್ನದೇ ಆದ ಸ್ಥಳೀಯ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ, ನಾನು/ನಾವು ಮಧುಮೇಹ ಮೂತ್ರಪಿಂಡ ಕಾಯಿಲೆಯಲ್ಲಿ ಮೂತ್ರಪಿಂಡ ನಿರೋಧಕ ಸೂಚ್ಯಂಕ ಅಧ್ಯಯನದಲ್ಲಿ ಸೇರಿಸಲ್ಪಡುತ್ತೇವೆ, ಈ ಮೂಲಕ ನಾನು/ನಾವು ರಕ್ತಶಾಸ್ತ್ರೀಯ ಮತ್ತು ವೈದ್ಯಕೀಯ ನಿಯತಾಂಕಗಳ ಅವಲೋಕನಗಳನ್ನು ದಾಖಲಿಸಲು ಯಾವುದೇ ಬಲವಂತ ಅಥವಾ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ನನ್ನ ಮಾನ್ಯ ಲಿಖಿತ ಮಾಹಿತಿಯುಕ್ತ ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡುತ್ತೇನೆ. ಒಳಗೊಂಡಿರುವ ಸ್ವರೂಪ ಮತ್ತು ಅಪಾಯಗಳನ್ನು ನನಗೆ ತೃಪ್ತಿಪಡಿಸುವಂತೆ ವಿವರಿಸಲಾಗಿದೆ. ನಡೆಸಲಾಗುತ್ತಿರುವ ಅಧ್ಯಯನದ ಬಗ್ಗೆ ನನಗೆ ವಿವರವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ. ನಾನು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಯಾವುದೇ ಪ್ರಶ್ನೆಯನ್ನು ಕೇಳಲು ನನಗೆ ಅವಕಾಶ ಸಿಕ್ಕಿದೆ. ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ತಕ್ಕಂತೆ ಉತ್ತರಿಸಲಾಗಿದೆ. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ನನ್ನನ್ನು / ನನ್ನ ಸಂಬಂಧಿಯನ್ನು ಭಾಗವಹಿಸಲು ಅನುಮತಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ. ಇತಿಹಾಸವನ್ನು ಒದಗಿಸಲು, ದೈಹಿಕ ಪರೀಕ್ಷೆಗೆ ಒಳಗಾಗಲು, ಕಾರ್ಯವಿಧಾನಕ್ಕೆ ಒಳಗಾಗಲು, ತನಿಖೆಗಳಿಗೆ ಒಳಗಾಗಲು ಮತ್ತು ಅದರ ಫಲಿತಾಂಶಗಳು ಮತ್ತು ದಾಖಲೆಗಳನ್ನು ಇತ್ಯಾದಿಗಳನ್ನು ವೈದ್ಯರು / ಸಂಸ್ಥೆಗೆ ಒದಗಿಸಲು ನಾನು ಈ ಮೂಲಕ ಸಮ್ಮತಿಸುತ್ತೇನೆ. ಶೈಕ್ಷಣಿಕ ಮತ್ತು ವೈಜ್ಞಾನಿಕ ಉದ್ದೇಶಗಳಿಗಾಗಿ ಕಾರ್ಯಾಚರಣೆ / ಕಾರ್ಯವಿಧಾನ ಇತ್ಯಾದಿಗಳನ್ನು ವೀಡಿಯೊ ಗ್ರಾಫ್ ಮಾಡಬಹುದು ಅಥವಾ ಛಾಯಾಚಿತ್ರ ಮಾಡಬಹುದು. ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಪ್ರಕಟಿಸಬಹುದು ಅಥವಾ ಯಾವುದೇ ಶೈಕ್ಷಣಿಕ ಉದ್ದೇಶಕ್ಕಾಗಿ ಬಳಸಬಹುದು.

_____

ರೋಗಿಯ / ಪೋಷಕರ ಹೆಸರು
(ರೋಗಿಯೊಂದಿಗಿನ ಸಂಬಂಧ)

_____

(ರೋಗಿಯ / ಪರಿಚಾರಕರ ಸಹಿ)

(ಸಹಿ ಮತ್ತು ಸಂಶೋಧನಾ ವೈದ್ಯರ ಹೆಸರು)

PATIENT INFORMATION SHEET

Study title : Study of Renal Resistive Index in Diabetic kidney Disease

Principal investigator: DR Y SUNAYANA

I DR Y SUNAYANA, Post graduate student in Department of general medicine at Sri Devraj Urs Medical College, will be conducting a study titled **"Study of Renal Resistive Index in Diabetic kidney Disease** . This study will be useful for Early management of diabetic nephropathy. The funds needed for the Renal doppler and renal function test will be done at my own expense .2 ml of blood will be drawn for estimation of renal function test, from each of the participating patients in this study.This study will be done under the guidance of Dr PRABHAKAR K Professor of Department of GENERAL MEDICINE .

All the data will be kept confidential and will be used only for purpose specified by the institution. You are free to provide consent for the participation of yourself in this study. You can also withdraw yourself from the study at any point of time without giving any reasons whatsoever. Your refusal to participate will not prejudice you to any present or future care at this institution.

In case of any clarifications are needed you are free to contact me on this mobile number - 8500872475

Name and Signature of the Principal Investigator

Date-Patient or patient by standers Signature

ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಮಧುಮೇಹ ಮೂತ್ರಪಿಂಡ ಕಾಯಿಲೆಯಲ್ಲಿ ಮೂತ್ರಪಿಂಡ ನಿರೋಧಕ ಸೂಚ್ಯಂಕದ ಅಧ್ಯಯನ

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: ಡಾ. ವೈ. ಸುನಯನ

ಶ್ರೀ ದೇವರಾಜ್ ಅರಸ್ ವೈದ್ಯಕೀಯ ಕಾಲೇಜಿನಲ್ಲಿ ಸಾಮಾನ್ಯ ವೈದ್ಯಕೀಯ ವಿಭಾಗದಲ್ಲಿ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿನಿ ಡಾ. ವೈ. ಸುನಯನ ಅವರು "ಮಧುಮೇಹ ಮೂತ್ರಪಿಂಡ ಕಾಯಿಲೆಯಲ್ಲಿ ಮೂತ್ರಪಿಂಡ ನಿರೋಧಕ ಸೂಚ್ಯಂಕದ ಅಧ್ಯಯನ" ಎಂಬ ಶೀರ್ಷಿಕೆಯ ಅಧ್ಯಯನವನ್ನು ನಡೆಸಲಿದ್ದಾರೆ. ಈ ಅಧ್ಯಯನವು ಮಧುಮೇಹ ನೆಫ್ರೋಪತಿಯ ಆರಂಭಿಕ ನಿರ್ವಹಣೆಗೆ ಉಪಯುಕ್ತವಾಗಿರುತ್ತದೆ. ಮೂತ್ರಪಿಂಡ ಡಾಪ್ಲರ್ ಮತ್ತು ಮೂತ್ರಪಿಂಡದ ಕಾರ್ಯ ಪರೀಕ್ಷೆಗೆ ಅಗತ್ಯವಿರುವ ಹಣವನ್ನು ನನ್ನ ಸ್ವಂತ ಖರ್ಚಿನಲ್ಲಿ ಮಾಡಲಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ಭಾಗವಹಿಸುವ ಪ್ರತಿಯೊಬ್ಬ ರೋಗಿಯಿಂದ ಮೂತ್ರಪಿಂಡ ಕಾರ್ಯ ಪರೀಕ್ಷೆಯ ಅಂದಾಜುಗಾಗಿ 2 ಮಿಲಿ ರಕ್ತವನ್ನು ತೆಗೆದುಕೊಳ್ಳಲಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನವನ್ನು ಸಾಮಾನ್ಯ ವೈದ್ಯಕೀಯ ವಿಭಾಗದ ಪ್ರಾಧ್ಯಾಪಕ ಡಾ. ಪ್ರಭಾಕರ್ ಕೆ ಅವರ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ಮಾಡಲಾಗುತ್ತದೆ.

ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಗೌಪ್ಯವಾಗಿಡಲಾಗುತ್ತದೆ ಮತ್ತು ಸಂಸ್ಥೆಯು ನಿರ್ದಿಷ್ಟಪಡಿಸಿದ ಉದ್ದೇಶಕ್ಕಾಗಿ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಗೆ ನೀವು ಒಪ್ಪಿಗೆಯನ್ನು ನೀಡಲು ಸ್ವತಂತ್ರರು. ಯಾವುದೇ ಕಾರಣಗಳನ್ನು ನೀಡದೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು. ಭಾಗವಹಿಸಲು ನೀವು ನಿರಾಕರಿಸುವುದು ಈ ಸಂಸ್ಥೆಯಲ್ಲಿನ ಯಾವುದೇ ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಆರೈಕೆಗೆ ಹಾನಿ ಮಾಡುವುದಿಲ್ಲ.

ಯಾವುದೇ ಸ್ಪಷ್ಟೀಕರಣಗಳ ಅಗತ್ಯವಿದ್ದರೆ ನೀವು ಈ ಮೊಬೈಲ್ ಸಂಖ್ಯೆ - 8500872475 ನಲ್ಲಿ ನನ್ನನ್ನು ಸಂಪರ್ಕಿಸಬಹುದು.

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ

ದಿನಾಂಕ-ರೋಗಿ ಅಥವಾ ರೋಗಿಯ ಪ್ರತಿವಾದಿಯ ಸಹಿ

MASTER CHART

Sl no	Group	Age	Sex	of Diabetes	Weight(kg)	HbA1c	um creatin	BUN	HDL	LDL	VLDL	um Cholest	of kidney d	RRI	UACR	EGFR
1	1	48	F	17	72	7.4	2.5	43	36	85	45	184	2	0.8	121	26.5
2	1	42	F	16	58	8	3.1	39	38	65	38	249	2	0.9	151	6.7
3	1	40	F	9	65	6.4	2.9	34	40	80	40	150	3	1.1	144	14.4
4	1	42	M	3	58	8	2.4	26	36	78	39	267	4	1.2	102	5.5
5	1	46	M	11	60	8.5	2.5	52	30	78	27	144	3	0.9	270	9.4
6	1	47	F	2	62	8.9	2.6	96	34	84	34	272	2	0.8	28	31.4
7	1	67	M	23	68	6.5	2.8	104	41	98	46	180	3	0.6	46	23.2
8	1	52	M	3	68	6.8	2.4	45	42	182	52	276	2	0.8	18	10.8
9	1	49	M	8	59	7.2	3.8	55.7	40	192	28	260	3	0.9	108	7.1
10	1	65	F	14	61	8.8	2.6	39	38	120	36	150	4	0.8	52	6.3
11	1	65	F	25	72	8.4	2.9	30	36	75	40	186	3	0.9	49	7.8
12	1	72	F	28	68	7.2	3.4	26	42	97	91	230	2	0.8	45	23
13	1	56	M	5	65	7.1	3.1	18	38	124	88	146	3	0.9	189	18
14	1	47	F	7	70	6.6	2.5	26	40	133	24	197	4	1.1	42	23
15	1	54	F	6	75	6.5	2.4	20	38	34	22	98	3	1	220	12.8
16	1	66	F	15	58	6.4	2.6	28	36	126	32	152	2	0.8	243	35
17	1	56	F	4	58	8.4	1.8	32	40	130	36	200	3	0.9	32	18
18	1	44	F	5	69	7.1	1.9	30	35	110	40	150	2	1.1	136	14.4
19	1	46	F	4	65	7.2	2.6	30	40	50	36	240	3	1.2	225	12.5
20	1	58	F	7	70	6.6	1.8	20	35	40	26	200	4	0.9	32	6.8
21	1	63	M	13	70	6.8	1.6	116	35	100	40	300	3	0.8	203	18.4
22	1	59	M	6	75	8.8	2.1	22	41	124	22	173	2	0.6	33	5.7
23	1	40	M	2	75	6.9	1.2	96	45.4	221	45	312	3	0.8	276	16.8
24	1	54	M	15	66	6.7	1.3	52	40	240	146	336	2	0.9	35	9.3
25	1	53	F	8	69	6.4	1.5	96	40.9	110	12	163	3	0.8	41	24.5
26	1	56	F	7	59	8.8	1.8	104	59.5	144	16	219	4	0.9	42	9.2
27	1	55	F	11	69	9.4	1.6	45	59.5	144	16	219	3	0.8	199	18.2
28	1	58	M	5	73	6.6	1.2	39	60	80	22	150	2	0.8	39	40
29	1	52	M	1	71	6.4	1.8	43	36	127	30	198	3	0.9	128	4.9
30	1	44	F	9	69	7.8	2.6	20	38	102	26	164	4	1.1	37	32
31	1	57	F	12	59	8.2	2.4	42	40	184	45	263	3	1.2	243	6.4
32	1	45	M	4	58	11.9	3.2	30	38	146	236	208	2	0.9	276	18
33	1	59	F	17	66	7.9	1.5	39	41	125	34	192	2	0.8	199	14.4
34	1	40	F	7	79	7.3	1.8	32	43	143	27	215	3	0.8	189	5.5
1	2	48	F	2	52	7.2	1	55.7	40	96	48	136	1	0.7	4	80
2	2	42	F	1	50	8	1.2	39	42	145	50	230	2	0.6	6	97.3
3	2	45	F	3	55	7.2	1.5	30	36	88	30	130	1	0.7	8	98
4	2	48	F	8	57	8	1.3	26	36	88	35	126	2	0.8	6	97.3
5	2	53	F	3	55	8.3	1.4	18	38	90	38	170	1	0.8	12	91.5
6	2	45	F	10	59	8.3	1.2	26	35	86	36	150	2	0.7	14	97.3
7	2	54	F	2	60	6.7	0.9	20	46	172	46	292	1	0.7	6	97.3
8	2	50	M	1	53	7.9	0.8	28	45	84	48	107	1	0.7	8	87
9	2	55	M	3	68	10	1.1	32	38	140	52	222	1	0.7	14	86
10	2	48	M	9	65	7.2	1.6	30	38	132	50	152	1	0.8	12	85.3
11	2	40	M	6	55	6.5	1.7	30	30	78	46	137	2	0.8	6	84.3
12	2	45	F	11	53	7.3	1.5	20	40	86	38	179	1	0.7	4	90.2
13	2	45	F	2	79	7.5	1.2	116	38	90	36	207	2	0.7	7	87
14	2	58	F	8	61	6.8	1.1	22	39	133	18	190	1	0.8	8	93.4
15	2	58	M	7	60	9.5	1	24	47	61	56	164	2	0.8	1	92.1
16	2	40	M	3	60	7.5	1.6	32	38	152	47	237	1	0.7	2	91.5
17	2	40	F	8	50	8.3	1.2	26	40	75	49	164	2	0.9	5	97.3
18	2	44	F	15	70	13.1	1.2	25	39	113	19	171	1	0.8	13	89
19	2	62	M	10	54	6.5	2.1	39	47	158	24	229	2	0.7	14	91.5
20	2	57	F	1	55	10.8	2.3	32	32	130	16	178	1	0.7	16	97.3
21	2	49	F	9	59	8	1.2	39	35	80	24	125	2	0.8	14	91.5
22	2	44	F	14	62	7.6	1.5	22	43	80	53	176	1	0.7	5	97.3
23	2	48	F	11	58	7.3	1.6	15	45	60	60	130	1	0.8	14	91.5
24	2	52	M	2	58	7.1	2.5	30	42	86	34	229	1	0.7	15	97.3
25	2	42	M	16	60	7.8	1.7	20	35	90	52	168	2	0.8	16	87
26	2	64	F	11	50	6.7	2.6	32	32	76	46	229	1	0.7	14	86
27	2	51	M	22	60	7.8	1.6	22	42	51	42	257	2	0.7	18	85.3
28	2	48	M	18	55	7.4	1.5	26	36	47	45	250	1	0.7	16	84.3
29	2	40	F	12	58	7.9	1.2	96	40	35	40	160	2	0.8	25	90.2
30	2	40	M	3	61	7.3	1.6	32	46	40	46	270	1	0.7	41	92.1
31	2	49	F	8	52	8.2	1.8	17	40	75	40	250	2	0.7	4	93.4
32	2	42	M	4	70	7.9	1.1	30	35	80	35	160	1	0.7	6	91.5
33	2	43	M	14	75	7.3	1.3	43	40	85	40	168	2	0.8	8	97.3
34	2	47	M	9	65	6.8	1.2	39	40	89	43	173	1	0.7	9	91.5
1	3	38	F	3	72	4.9	2.6	17	40	84	37	162	0	0.5	8	97.3

Sl no	Group	Age	Sex	of Diabete	Weight(kg)	HbA1c	um creatin	BUN	HDL	LDL	VLDL	um Cholest	of kidney d	RRI	UACR	EGFR
2	3	42	F	10	75	5.9	0.6	30	54	128	43	203	0	0.6	6	87
3	3	45	F	8	65	5.7	1	43	50	89	176	145	0	0.5	8	86
4	3	38	F	18	64	6.3	1	39	41	88	34	103	0	0.6	6	85.3
5	3	23	F	9	68	5.8	0.6	43	40	34	37	112	0	0.5	12	84.3
6	3	45	F	4	59	4.9	1.4	39	46.5	104	40	191	0	0.7	14	87
7	3	24	F	3	58	5.5	0.2	34	40	127	26	193	0	0.6	6	86
8	3	30	M	11	65	6	0.5	26	51	92	11	155	0	0.5	8	85.3
9	3	35	M	10	70	5.8	0.8	24	57	118	19	195	0	0.5	14	84.3
10	3	38	M	8	70	5.9	0.2	32	47	38	169	255	0	0.6	12	90.2
11	3	40	M	2	74	5.7	0.8	26	37	74	24	136	0	0.4	6	87
12	3	45	F	6	69	6	1	25	47	38	170	255	0	0.5	6	93.4
13	3	25	F	9	61	6.3	0.5	39	44	113	14	172	0	0.6	8	92.1
14	3	38	F	2	60	5.5	0.6	32	48	38	170	255	0	0.5	6	91.5
15	3	38	M	5	60	5.7	0.6	39	37	128	19	184	0	0.7	12	97.3
16	3	40	M	1	50	5.7	0.2	22	28.8	153	247	246	0	0.6	14	87
17	3	40	F	10	70	4.5	0.5	15	36.2	117	25	231	0	0.5	6	86
18	3	44	F	11	54	4.9	0.5	30	45	146	23	214	0	0.5	8	85.3
19	3	62	M	12	55	5.6	0.9	20	44	85	25	135	0	0.6	14	84.3
20	3	27	F	8	59	5.5	1.4	32	35.5	119	43	200	0	0.7	12	90.2
21	3	39	F	15	62	6.1	1.7	22	59	145	68	218	0	0.5	6	87
22	3	44	F	10	58	5.7	0.6	26	34	82	24	156	0	0.6	4	93.4
23	3	38	F	10	58	5.8	0.3	39	41	104	29	189	0	0.5	7	92.1
24	3	32	M	12	60	5.5	2.5	43	39	90	25	169	0	0.5	8	91.5
25	3	42	M	16	50	6.4	1	39	45	80	30	160	0	0.5	1	97.3
26	3	64	F	2	60	5.9	0.3	34	40	65	34	206	0	0.6	2	89
27	3	51	M	3	55	5.7	0.2	26	40	100	30	160	0	0.7	5	91.5
28	3	48	M	18	58	5.1	0.3	24	40	100	30	170	0	0.5	13	97.3
29	3	40	F	2	61	5.8	0.2	32	40	92	30	158	0	0.5	14	91.5
30	3	30	M	15	52	5	0.2	26	43	103	31	236	0	0.6	16	97.3
31	3	39	F	14	70	5.4	0.4	25	16	75	23	238	0	0.5	14	91.5
32	3	32	M	4	75	5.9	0.4	39	36	117	25	231	0	0.6	5	86
33	3	43	M	5	65	5.7	0.2	32	44	94	45	184	0	0.5	14	85.3
34	3	37	M	6	72	5.1	0.3	39	148	128	33	223	0	0.6	15	84.3