"COMPARISON OF PULMONARY FUNCTION TESTS IN PATIENTS WITH DIABETIC NEPHROPATHY AND WITHOUT NEPHROPATHY"

BY

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IN PARTIAL FULFILLMENT OF THE REQUIREMENTS FOR THE DEGREE OF

DOCTOR OF MEDICINE (M.D.)

UNDER THE GUIDANCE OF

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COMPARISON OF PULMONARY FUNCTION TESTS IN PATIENTS WITH DIABETIC NEPHROPATHY AND WITHOUT NEPHROPATHY

Abstract

Background

The pulmonary complications of type 2 diabetes mellitus have gotten little attention, despite the fact that cardiovascular issues, nephropathy, diabetic retinopathy, and neuropathy have garnered a lot of attention. This study seeks to discover pulmonary function abnormalities in diabetic nephropathy patients. The lung's large connective tissue and microvascular circulation lend credence to the notion that it could be a "target organ" in diabetes patients.

Material and Methods

A cross-sectional study was conducted in the department of General Medicine at RLJH - A Tertiary Care Centre Tamaka, Kolar for 18 months, from May 2023 to October 2024, among all adult patients diagnosed with Type 2 Diabetes Mellitus, with and without diabetic nephropathy. The minimal sample size is 30. But in this study, 50 subjects in each group were considered.

Results

In this study, patients aged 41 to 50 accounted for 36.0% of cases with diabetic nephropathy and 44.0% of cases without it. In diabetic nephropathy cases, the mean HbA1c was 7.5 gm%, but in non-diabetic nephropathy cases, it was 7.7 gm%. In patients with diabetic nephropathy, forced expiratory volume 1 was 1.72 litres, while in those without, it was 1.66 litres. In instances with diabetic nephropathy, the mean functional vital capacity was 3.61 litres, while in those without, it

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COMPARISON OF PULMONARY FUNCTION TESTS IN PATIENTS WITH DIABETIC NEPHROPATHY AND WITHOUT NEPHROPATHY Abstract Background The pulmonary complications of type 2 diabetes mellitus have gotten little attention, despite the fact that cardiovascular issues, nephropathy, diabetic retinopathy, and neuropathy have garnered a lot of attention. This study seeks to discover pulmonary function abnormalities in diabetic nephropathy patients. The lung's large connective tissue and microvascular circulation lend credence to the notion that it could be a "target organ" in diabetes patients. Material and Methods A cross-sectional study was conducted in the department of General Medicine at RLJH - A Tertiary Care Centre Tamaka, Kolar for 18 months, from May 2023 to October 2024, among all adult patients diagnosed with Type 2 Diabetes Mellitus, with and without diabetic nephropathy. The minimal sample size is 30. But in this study, 50 subjects in each group were considered. Results In this study, patients aged 41 to 50 accounted for 36.0% of cases with diabetic nephropathy and 44.0% of cases without it. In diabetic nephropathy cases, the mean HbA1c was 7.5 gms%, but in non-diabetic nephropathy cases, it was 7.7 gm%. In patients with diabetic nephropathy, forced expiratory volume 1 was 1.72 litres, while in those without, it was 1.66 litres. In instances with diabetic nephropathy, the mean functional vital capacity was 3.61 litres, while in those without, it was 3.02 litres. The peak expiratory flow rate in diabetic nephropathy patients was 216.32, compared to 198.78 in diabetic nephropathy patients. The mean FEV1/FVC ratio in diabetic nephropathy patients was 0.50, compared to 0.56 in diabetic nephropathy patients. Conclusion This study found that diabetic nephropathy patients had worse FEV1, FVC, FEV1/FVC, and PEFR values than diabetic patients without nephropathy. Keywords: Diabetic nephropathy, HbA1c, Functional vital capacity, Forced expiratory volume, FEV1/FVC ratio INTRODUCTION Diabetes is a vital cause of kidney damage, myocardial ischemia, cerebro-vascular accident, retinal damage and limb injuries due to its microvascular and macrovascular problems that affect organs like the retina, kidney, nerve, and cardiovascular system. Because of their extensive connective tissue and pulmonary capillary network, the lungs may be impacted by chronic hyperglycemia even though they are not a typical organ involved in diabetes1. Persistent hyperglycemia and changes in the degradation of proteins, glucose, starch, and fatty foods. Reduced tissue sensitivity to insulin, insufficient insulin production, or a combination of the two causes certain metabolic disorders. Type 2 diabetes mellitus can cause permanent damage, dysfunction, or failure to many organs, and its consequences are usually caused by microvascular and macrovascular damage2,3. The target organs of diabetic complications have a similar microangiopathic origin, i.e lung parenchyma, retina, nephron and nerves4. As we already known that cardiovascular problems, nephropathy, diabetic retinopathy, and neuropathy receive a lot of attention, yet, lung damage till now received little attention. The idea that the lung could be a target organ for diabetic microangiopathy has recently gained a lot of interest. DM of type 2 is characterized by incessant levels of high glucose and changes in the degradation of proteins, glucose, starch, cellulose, and fatty contents. Reduced tissue sensitivity to insulin, insufficient insulin production, or a combination of the two causes certain metabolic disorders. Type 2 diabetes mellitus can cause permanent damage, dysfunction, or failure to many organs, and its consequences are usually caused by microvascular and macrovascular damage5. This investigation aims to identify pulmonary function anomalies in Diabetic nephropathy patients. The idea that the lung could also be a "target organ" in diabetic patients is increased by the lung's extensive connective tissue and microvascular circulation. Diabetics' lungs experience several histological alterations as a result of chronic hyperglycemia. Alveolar epithelium thickening and pulmonary capillary basal lamina thickening are examples of this. These modifications ultimately lead to a decrease in lung volume and elastic recoiling capacity. The change of connective tissue caused by non-enzymatic glycosylation in the lung parenchyma is most likely the cause6. In a study by Liu et al, Systolic blood pressure, Fasting and Post-prandial Blood Glucose, HbA1c, Blood Urea Nitrogen, Serum creatinine, Total Leucocyte count, Vital capacity, FEV1/FVC, Maximal voluntary ventilation, DLCO, and DLCO/VA investigations showed that all these influence the clinical outcome of diabetic patient acquiring nephropathy; hence lowering blood pressure, glucose, fats and enhancing lung perfusion & ventilation, and improving diffusion function will help in better prevention and delay the occurrence of Nephropathy. In diabetic nephropathy patients, Renal function tests slowly increased, associated with a decreased in lung perfusion and ventilation function7. Only a relatively small number of research have been conducted, according to the literature, to explain the harmful consequences of diabetes mellitus on pulmonary functioning. As a result, this study will add to the growing body of knowledge about pulmonary functioning in people with diabetes mellitus. The most widely used

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Date: Signature of candidate

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COMPARISON OF PULMONARY FUNCTION TESTS IN PATIENTS WITH DIABETIC NEPHROPATHY AND WITHOUT NEPHROPATHY

ABSTRACT

BACKGROUND : The pulmonary complications of type 2 diabetes mellitus have gotten little attention, despite the fact that cardiovascular issues, nephropathy, diabetic retinopathy, and neuropathy have garnered a lot of attention. This study seeks to discover pulmonary function abnormalities in diabetic nephropathy patients. The lung's large connective tissue and microvascular circulation lend credence to the notion that it could be a "target organ" in diabetes patients.

MATERIALS AND METHODS : A cross-sectional study was conducted in the department of General Medicine at RLJH - A Tertiary Care Centre Tamaka, Kolar for 18 months, from May 2023 to October 2024, among all adult patients diagnosed with Type 2 Diabetes Mellitus, with and without diabetic nephropathy. The minimal sample size is 30. But in this study, 50 subjects in each group were considered.

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CONCLUSION : This study found that diabetic nephropathy patients had worse FEV1, FEV1/FVC, and similar FVC, PEFR values compared to diabetic patients without nephropathy.

KEYWORDS : Diabetic nephropathy, HbA_{1c}, Functional vital capacity, Forced expiratory volume, FEV_1/FVC ratio

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ABBREVIATIONS

Abbreviations (in Alphabetical order)	
ACR	Albumin/ Creatinine Ratio
AGE	Advanced Glycation End products
AMPK	AMP activated protein kinase
ATP	Ademosine Triphosphate
BUN	Blood Urea Nitrogen
CAT	Catalase
DAG	Diacyglycerol
DKD	Diabetic Ketoacidosis
DLCO	Diffusion Capacity of Lung for Carbon Dioxide
DN	Diabetic Nephropathy
ETC	Electron Transport Chain
eNOS	Endothelial nitric oxide synthase
FADH	Flavin Adeniine Dinucleotide
FBG	Fasting Blood glucose
FFA	Free Fatty Acid
FEV	Forced Expiratory volume
FVC	Functional Vital Capacity
GFR	Glomerular Filtration Rate
GPx	Glutathione Peroxidase

GLUT4	Glucose Transporter – 4
GSSG	Oxidized Glutathione
HbA_{1c}	Glycosylated Haemoglobin
H_2O_2	Hydrogen Peroxide
IL	Interleukin
ICAM-1	Intercellular adhesion molecule
IRS	Insulin Resistance
MCP-1	Monocyte chemotactic Protein
mtDNA	Mitochodrial Deoxy Ribo Nucleic acid
mTORC	Mamallian Target Rapamycin Complex
MVV	Maximum Voluntary Ventilation
NAD	Nicotinamide Adenine Dinucleotide
NADH	Nicotinamide Adenine Dinucleotide
NO	Nitric oxide
NOX	NADPH oxidase
NRF	Nuclear eythroid 2 related factor
OH	Hydroxy radical
OS	Oxidative Stress
O_2	Molecular Oxygen
PAI-1	Plasminogen Activator inhibitor – 1
PEFR	Peak Expiratory Flow Rate
PFT	Pulmonary Function Test
PGC-1α	Peroxidase Porliferator activated gamma coactivator 1 – Alpha

PKC	Protein Kinase C
2hPG	2 hours Post Prandial Glucose
RAAS	Renin – angiotensin aldosterone system
RLJH	R.L Jalppa Hospital
RONS	Reactive oxygen and nitrogen species
ROS	Reactive Oxygen Species
SBP	Systolic Blood Pressure
Ser	Serum Creatinine
SNS	Sympathetic Nervous System
SOD	Superoxide Dismutase
TGF	Tumor growth factor
TLC	Total Leucocyte Count
TNF	Tumour Necrosis Factor
VC	Vital Capacity
VACM	Vascular cell adhesion molecule

INTRODUCTION

INTRODUCTION

Diabetes is a vital cause of kidney damage, myocardial ischemia, cerebro-vascular accident, retinal damage and limb injuries due to its microvascular and macrovascular problems that affect organs like the retina, kidney, nerve, and cardiovascular system. Because of their extensive connective tissue and pulmonary capillary network, the lungs may be impacted by chronic hyperglycemia even though they are not a typical organ involved in diabetes[1]. Persistent hyperglycemia and changes in the degradation of proteins, glucose, starch, and fatty foods. Reduced tissue sensitivity to insulin, insufficient insulin production, or a combination of the two causes certain metabolic disorders. Type 2 diabetes mellitus can cause permanent damage, dysfunction, or failure to many organs, and its consequences are usually caused by microvascular and macrovascular damage[2,3].

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This investigation aims to identify pulmonary function anomalies in Diabetic nephropathy patients. The idea that the lung could also be a "target organ" in diabetic patients is increased by the lung's extensive connective tissue and microvascular circulation. Diabetics' lungs experience several histological alterations as a result of chronic hyperglycemia. Alveolar epithelium thickening and pulmonary capillary basal lamina thickening are examples of this. These modifications ultimately lead to a decrease in lung volume and elastic recoiling capacity. The change of connective tissue caused by non-enzymatic glycosylation in the lung parenchyma is most likely the cause[6].

In a study by Liu et al, Systolic blood pressure, Fasting and Post-prandial Blood Glucose, HbA_1c, Blood Urea Nitrogen, Serum creatinine, Total Leucocyte count, Vital capacity, FEV_1/FVC, Maximal voluntary ventilation, DLCO, and DLCO/VA investigations showed that all these influence the clinical outcome of diabetic patient acquiring nephropathy; hence lowering blood pressure, glucose, fats and enhancing lung perfusion & ventilation, and improving diffusion function will help in better prevention and delay the occurrence of Nephropathy. In diabetic nephropathy patients, Renal function tests slowly increased, associated with a decreased in lung perfusion and ventilation function[7].

Only a relatively small number of research have been conducted, according to the literature, to explain the harmful consequences of diabetes mellitus on pulmonary functioning. As a result, this study will add to the growing body of knowledge about pulmonary functioning in people with diabetes mellitus.

The most widely used pulmonary function test - which calculates the functional capacity of lung, specifically the amount of air and the force of speed of air that can be inhaled and exhaled is called as – Spirometry (i.e. breath measure)

This study aims to broadcast as to how chronic hyperglycemia effects lung functions, mainly centering on mechanical aspects of lung dysfunction – calculating modalities like Forced Vital Capacity, Forced expiratory volume in 1 second, Peak expiratory flow rate and the ratio between FEV1 & FVC to be specific[8].

Early assessment and primordial prevention goes a long way in the management of Diabetic nephropathy, hence the need for PFT in patients with Diabetic Nephropathy.

AIMS & OBJECTIVES

AIMS & OBJECTIVES

OBJECTIVES OF THE STUDY –

1. To estimate PFT in patients with Diabetic nephropathy.
2. To compare PFT in patients with nephropathy and without nephropathy.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

The primary cause of both macrovascular and microvascular problems linked to diabetes mellitus is chronic hyperglycemia.

The pathophysiology of diabetic kidney disease, the world's largest cause for dialysis, is greatly influenced by excess glucose, which causes imbalance of oxidation and triggers systemic inflammatory response along with renal parenchymal inflammation.

The occurrence, progression, and clinical implications of the disease are influenced by numerous factors and mechanisms, making the etiology of the condition intricate, multifaceted, and incompletely understood. The mechanism of the pathways of oxidation and inflammation are generally considered to be responsible for kidney damage associated with diabetes mellitus, despite the fact that these metabolic processes are diverse. There’s been convincing evidence postulating a long-term hyperglycemic condition accelerates progression in action of reactive oxygen species causing stress and also inflammation through modified pathways of metabolism in a self-reinforcing loop. The pathophysiology of diabetic kidney disease, the world's largest cause of dialysis, is greatly influenced by excess glucose, which causes imbalance of oxidation and triggers systemic inflammatory response along with renal parenchymal inflammation.

A debilitating consequence of diabetes mellitus is high morbidity & overall mortality causes due to diabetes nephropathy. Structural abnormalities like diabetic glomerulosclerosis could be seen in diabetic patients, and it is clinically defined by persistent renal dysfunction lasting at least three months, and after a hyperfiltration phase – there will be excretion of albumin >30 mg in urine for about 24 hours, an urinary albumin/creatinine ratio ≥ 30 mg/g value[8].

Approximately 425 million population all over the world are thought to diabetic as of now and by 2045, it is only deemed to increase by 48%. Brazil ranks third globally in terms of the number of patients with DM in 2017, with about 12.5 million people receiving a diagnosis.3. Microvascular and macrovascular problems occur in over 90% of DM patients. Among those with DM Type 2, affecting almost 20–40% of individuals, is Diabetic kidney disease, which is considered as the most dreaded outcome. Currently, the primary reason for dialysis in wealthy nations is DKD[9–11].

The pathophysiology of DKD, a progressive and irreversible illness, has been linked to structural and functional alterations in renal cells as a result of metabolic stress brought on by an excessive inflow of glucose, which is accomplished by activating particular metabolic pathways connected to inflammation and redox imbalance[12].

Many stipulations have been proposed as the cause of the early functional loss of renals and it's related problems, being - molecular and epigenetic pathways, despite the fact that numerous conventional processes implicated in the genesis and progression of DKD have been characterized.

Pathophysiology of DKD

Hyperglycemia in DKD, a chronic metabolic disorder, results in renal and vascular cell failure.

The chronic hyperglycemic condition that causes hemodynamic dysfunction and the activation and alterations of metabolic pathways is the pathogenesis of diabetic kidney disease resulting in end-stage renal disease which ultimately requires hemodialysis.

Few of these changes happen in a coordinated manner, which results in a number of further alterations. We shall discuss the metabolic alterations brought on by both intermittent and continuous exposure to hyperglycemia, even though hyperglycemia secondary to diabetes is a significant yet it isn't essential contributing element for the progression of glomerular lesions in Diabetic kidney disease[13,14].

Diabetic Kidney Cells Uptake Of Glucose

Primary sign of diabetes mellitus and primary cause of chronic complications, such as DKD, is hyperglycemia. Type 1 diabetes is caused by an autoimmune abnormality that leads to the impaired function and cell death of β cells of pancreas, while type 2 diabetes is known to be due to excess synthesis & secretion of insulin despite insulin resistance, primarily linked to overweight/obesity[15,16].

Higher values of free fatty acids and their byproducts i.e cytokines and diacylglycerols cause insulin resistance in obesity, and it's prevalent in people with high risk for diabetes mellitus type 2. Insulin receptor substrate 1 phosphorylation is inhibited by FFA's which stops signals from propagating to plasma membrane via GLUT 4 transporter. It also has an impact on how insulin and its receptor interact, which lowers the amount of glucose that insulin-dependent cells absorb and eventually results in hyperglycemia and hyperinsulinemia[17,18].

Furthermore, excessive fat accumulation in obese diabetics stresses adipocytes through multiple replication and increased size of cells, resulting in decreased oxygen saturation and inflammation sub clinically, increased infiltration of macrophages, and pro-inflammatory cytokines release like - IL-6, IL-1, TNF - α, all of which exacerbate IR[11,13]. By influencing glucose absorption and encouraging insulin resistance, TNF-α in turn helps to release additional substrates and is indirectly activating kappa B which is a transcription factor, which maintains chronic hyperglycemia[19].

Hyperglycemia causing Reactive Oxygen and Nitrogen Species (RONS) generation

When IR or hyperglycemia occurs, as in DM, incapacity of the cells to atone for the elevated blood sugars intake causing glucotoxicity. In non-insulin-dependent cells, triggering of oxidation pathways results in stress in a elevated glucose level state, increased RONS synthesis, and the activation of alternate pathways[20].

Glycolysis is the main source of energy for cells when glycemic homeostasis is maintained and oxygen is not present. Coenzymes that absorb high-energy electrons generated during oxidation-reduction (redox) events and take part in other energy pathways are byproducts of these reactions.

Glycolysis's production of substrates triggers two further energy pathways: the Krebs cycle and Mitochondria's ETC pathway via protein complexes[21].

The hyperglycemic state of diabetes mellitus encourages the hyper stimulation of the 3 primary channels of energy that were discussed beforehand.

In Krebs cycle, the FADH2 & NADH, which were supposed to be low due to ETC, are prepared in higher amounts as a result of the increased glycolysis stimulation[22].

ETC produces Reactive Oxygen species, particularly in renal cells with many mitochondria. Through complexes I and II, generation of higher quantities of ATP will be seen because of increased reduction & oxidation potential of diabetic kidney cells as well as high levels of superoxide anion (O2-•) will be seen.

O2-• produces H2O2, •OH radical & ONOO- and may contribute to the development of the lesions, thus stating that both nonradical and radical ions can participate in the formation of renal lesions[23,24].

Hyperglycemia and hyperlipidemia alter mitochondrial energy in the renal parenchyma of diabetic or obese people, leading to altered function of the power house of the cell and excess ROS that damage DNA of the mitochondria by blocking the protein kinase which was activated by AMP activated Protein and mTORC1 via rising breakage of mitochondria and faulty production, and the alterations impact mitochondrial biogenesis via changing the activation of PGC-1α. It leads to reduced generation of ATP, can affect ECT activities by causing renal damage & cell apoptosis[25].

In addition to that, more glycolysis increases the production of AGEs, PKC activation, and aids in polyol and hexosamine metabolism. Additionally, it leads to lower ATP, and aggravates fragmentation & dysfunction of mitochondria[26].

Polyol pathway

Aldose reductase converts glucose into sorbitol due to increased levels of NADPH, and it is one effect of the cells' enhanced absorption of glucose. Following that, nicotinamide adenine dinucleotide (NAD) transforms sorbitol into fructose. For the antioxidant glutathione (GSH) to regenerate, NADPH is a crucial cofactor.

Consequently, the NADPH scarcity impacts the defense of the antioxidants and causes alterations in reduction & oxidation due to elevated aldose reductase activity. Glyceraldehyde 3-phosphate dehydrogenase (GAPDH) activity is inhibited by a rise in O2•-, which also reduces glycolysis and activates other pathways. PKC is activated when the NADH/NAD ratio rises because more DAG is produced. The pathway's byproduct fructose has lately been linked to indicators of kidney damage[27,28].

Protein Kinase C

Exaggerated elevation of glycolysis byproducts and hyperglycemia both boost the production of glyceraldehyde-3phosphate and its conversion to dihydroxyacetone, which in turn stimulates the production of PKC activating agents like DAG.

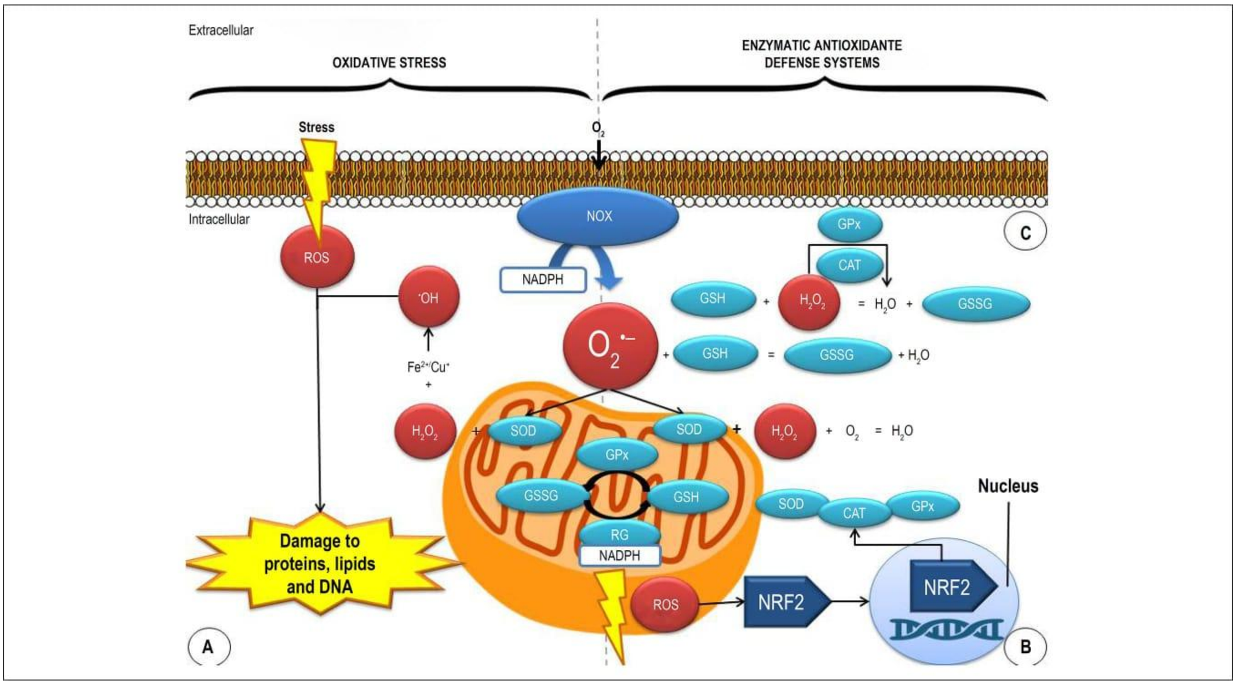


Fig 1: Oxidative stress and protective mechanism in diabetic kidney cells[14].

Here - CAT: Catalase; GPx: Glutathione Peroxidase; GSSG: Oxidized Glutathione; NRF_2: Nuclear Erythroid 2-related factor 2; NOX: NADPH Oxidase; SOD: super oxide dismutase.

In renal cells, elevated PKC triggers a number of processes that lead to kidney damage. In the early stages of diabetic kidney disease, Nitric oxide (NO) availability in the renal parenchyma is made possible by PKC - by inducing and activating endothelial nitric oxide synthase (eNOS). Increased permeability, altered function of endothelium, increased filtration of glomerulus, and excess urinary albumin excretion are caused by elevated NO, which also raises PG E1, activity of angiotensin II, and activation of VEGF[29,30].

Prolonged diabetes causes vasoconstriction as well as glomerular and systemic hypertension because continuous high levels of glucose lowers level of an eNOS cofactor, i.e. tetrahydrobiopterin, along with a corresponding decreased production of nitric oxide[31].

Endothelial dysfunction and the advancement of DKD are brought on by a nitroso-reduction-oxidation imbalance and due to interaction between O2•- and NO, RONs is elevated, raises ONOO- which promote hyperglycemia-related endothelial damage[30].

PKC overexpression triggers the activation of PAI-1 & TGF-β, it increases the degradation of varied collagen types, fibronectin, & matrix of extracellular type, which ultimately causes bigger size of cells of renal parenchyma, sclerosis of the glomerulus, and fibrosis of kidneys[28].

Hexosamines

Hyperglycemia stimulates the pathway for effective functioning, which leads to converting fructose 6-phosphate & synthesis of UDP-GlcNAc which is the final product. The O-GlcNAc transferase then O-glycosylates this product into N-acetylglucosamine (O-GlcNAc).

Cell proteins are stimulated and modified by an overabundance of O-GlcNAc. Genetic expression alterations in DKD lead to an increase in TNF-α transcription, which causes extracellular matrix protein overproduction and kidney injury through OS[32].

Advanced glycation end products (AGEs)

Increased endogenous synthesis brought on by hyperglycemia, food, and inadequate clearance of these products as a result of decreased GFR may all contribute to the development of renal injury.

AGEs are uremic poisons. Glucose, fructose & others, or the intermediary components of glucose metabolites (G-6-P, F-6-P R-5-P, and glyceraldehyde), act with an amines & others resulting in creation of a Schiff base & Amadori products which are the first byproducts. This leads to production of AGEs. Hyperglycemic circumstances speed up the synthesis of the Amadori products, which are extremely reactive with metal ions and amine groups via the glycolysis & oxidation of certain molecules to create glyoxal & methylglyoxal and the simple carbohydrates and its intermediates.

The acclamation amongst these molecules produce AGEs. Increased endogenous synthesis brought on by hyperglycemia, food, and inadequate clearance of these products as a result of decreased GFR may all contribute to the development of renal injury[33–35].

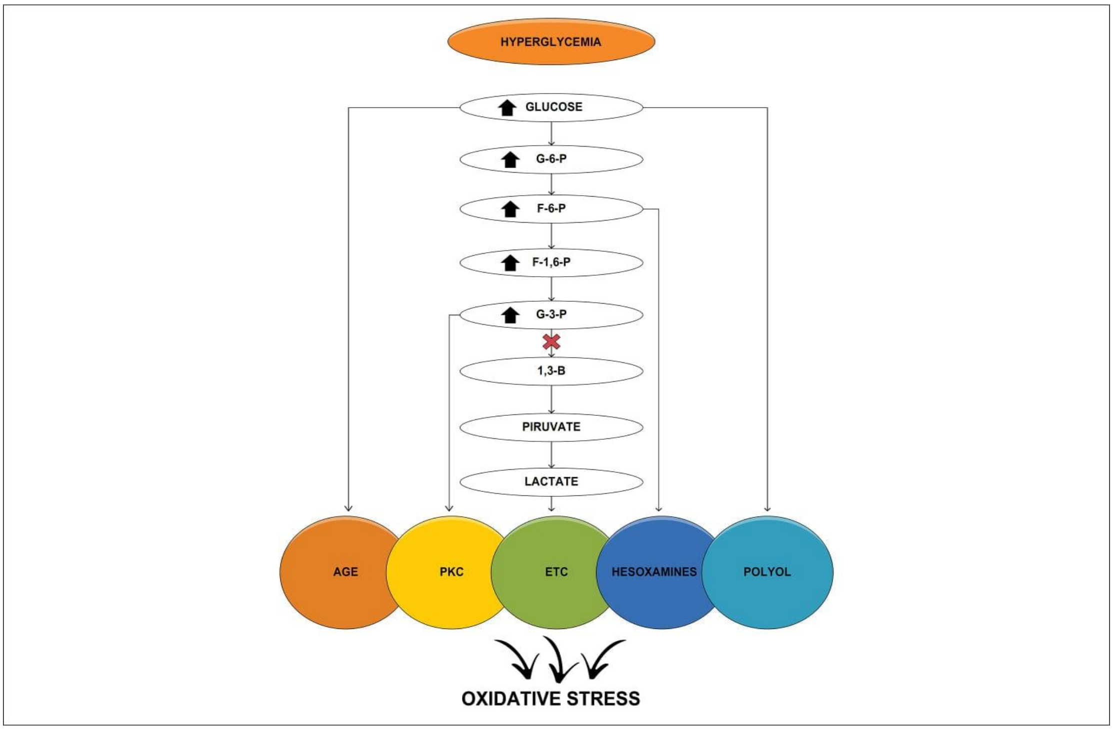


Fig 2 – Pathway before glycolysis & Oxidative stress[14].

Second generation AGEs are of little weight & are soluble & must be eliminated in urine following AGE metabolism and tissue removal. Although they can be extremely reactive substances, renal elimination limits their impact. However, the elimination of AGEs is compromised in renal failure, which leads to elevated levels in tissues and serum[33].

Through interactions between proteins present outside the cell and the macronutrients, an elevation seen in internal pool of AGEs directly damages cells, altering their structure and functionality. Because of the reduced enzymatic hydrolysis of these altered proteins, matrix proteins which are present outside the cell accumulate excessively, sclerosis of glomerulus develops, and shrunken kidneys follows[30].

AGEs react with RAGE receptor in a variety of inflammatory cell types, in addition to directly causing extracellular and intracellular damage. A series of internal reactions are started after the substrate/receptor connection. By activating macrophages through NF-kappa B, these processes control the production of proteins, attached molecules, IL-1, IL-6, and TNF-α, aggravating subclinical tissue inflammation linked to diabetes mellitus in DKD[36,37].

Increased RON synthesis is linked to the AGEs/RAGEs interaction, which also directly activates NOX (via the RAGEs in kidney cells & phagocytosed immune cells) and aids in OS.

Additionally, AGs cause OS in means increasing ONOO-production & reducing NO availability while decreasing eNOS expression and increasing inducible NOS (iNOS) expression. Thus, the production of MCP-1, VCAM-1 & ICAM-1 & TGF-β causes endothelial dysfunction[34,35].

NADPH oxidases

NADPH oxidases are a significant source of reactive oxygen species in diabetes mellitus. It has 7 distinct isoforms: one to five (4 was previously known as "renox") & double oxidases one & two respectively. These are proteins which shift electrons from NADPH to O2, making it into a radical, also prolonging damage to kidney parenchyma. Excess formation & storage of O2 shows there's altered balance in reduction & oxidation and damage to various renal tissues[38].

AG II

Podocytes, mesangial cells, and glomerular cells all produce more Ang II and its receptors in response to chronic hyperglycemia in diabetes mellitus. It raises intrarenal angiotensin levels by increasing mesangial cell expression of renin and angiotensinogen. ROS buildup in adipose tissue, which produces Ang II, aggravates this process[39].

Increases in Ang II lead to aberrant renin-angiotensin-aldosterone system (RAAS) activation, which exacerbates kidney mechanical damage brought on by HTN of vasculature or glomerulus. Along with this, it has extra functions like activation of cytokines & increased regulation causing preceded elevation of cell count, and delayed increased size of kidney tissue cells[40].

Hemodynamic variability of diabetic nephropathy

Differences of dynamics due to elevated glucose levels are indicative of early DKD. Glomerular hyperperfusion, hypertension, and hyperfiltration are the hallmarks of the initial hemodynamic events, which also cause changes in glomerular anatomy & physiology, leading to excessive excretion of albumin in urine, initially an elevated GFR followed by late reduction, also causing hyper proliferation of size of glomerular cells, expansion of mesangium, sclerosis of glomerulus and shrunken kidney following natural course of kidney disease[41,42].

DKD is frequently preceded by hypertension, particularly in DM2. Persistent metabolic disorders, on the other hand, result in persistent hypertension and pressure dysregulation, which can aggravate diabetic kidney damage. The complicated, multifactorial mechanism underlying hypertension in DKD includes aberrant activation of the SNS and RAAS, altered function of endothelium, elevated OS, and impaired sodium control, including renal tubular reabsorption of salt. These mechanisms increase extracellular volume and mediate vasoconstriction, which raises blood pressure[43,44].

The most commonly recognized hemodynamic component for the development of DKD is RAAS, which also contributes to HTN & excess filtration. Blocking RAAS has been demonstrated to slow the disease's progression[45].

The synthesis of Ang II in renal cells is significantly increased due to stress on the vessel which is due to increased blood pressures, elevated blood glucose levels, inflammation & ROS, which also contributes to RAAS hyperactivation. This ultimately leads to increases in BP, pressure of the glomerulus, and injury to parenchyma of kidneys by causing constriction of vessels, reuptake of electrolytes because of reaction with AT1 & aldosterone production[46].

Oxygen radical formation leads to impaired function of endothelium (associated with vascular constriction & dilation function abnormality), which is because of altered balance of oxidation & reduction. The formation of NO, strong vasodilator reacts with BH4, it’s decreased in response to elevated ROS. Additionally, O2•-directly reduces NO and ONOO-, which lowers NO availability and causes persistent vasoconstriction[47].

Together with altered function of endothelium, constriction & resistance of vessels brought on by the OS, Ang II's actions raise afferent arteriole pressure, which raises BP, hyper perfusion and excessive filtration of glomerulus, and excess protein elimination in urine, all of which contribute to the progression of DKD.

Furthermore, in DKD, sodium-hydrogen exchangers (NHEs) have a significant impact on systemic and renal hemodynamics.

Different types of renal cells express NHEs, which control the transfer of sodium & hydrogen, which help in a variety of cell processes, such as maintaining proper acid-base balance, quantity of liquid, and cell viability. Exchangers and their similar forms are involved in the pathophysiology of DKD, specifically in GBM area. They do this by stimulating the inhibition of apoptosis which leads to shrunken kidneys & hypertension within the glomerulus & hyper replication of mesangium[48,49].

Renin & salt sensors are regulated by the NHE2 receptors in macula densa cells. According to the proposed mechanism, Ang II and cell shrinkage brought on by hypertonicity serve as the renin-release signal, which causes the RAAS to overexpress and intraglomerular pressure to rise. This would start a vicious cycle by activating the signaling pathway that causes kidney cells to express more NHE receptors. Additionally, an excess of salt brought on by NHE in the macula densa raises intracellular pH and depolarizes cells, which triggers the NOX enzymes to start producing ROS[50].

Numerous pharmacological treatments target NHEs, such as SGLT and RAAS inhibitors, which block NHE in the kidneys and lower intraglomerular pressure as well as fibrotic and proliferative processes.

ACEi and ARBs, either alone or in combination, demonstrated efficacy in lowering proteinuria and slowing the progression of DKD. As a result, they've known to enhance the lives of patients living with diabetic nephropathy[51,52].

Redox imbalance in DKD

In almost all cell types, OS, the initial stage of DKD, initiates pathogenic pathways. OS arises from a redox imbalance between pro- and antioxidants, where the rise in RONs overpower less effective anti-oxidant structure[53,54].

Numerous cell types in the kidneys as well as invading like macrophages produce RONs. enhanced RON synthesis, which makes up almost 80% of all reactive species, is linked to a significant rise in self-oxidation of glucose, enhanced ETC stimulation & mitochondrium. Together with these pathways, other enzymatic systems, including AG 2 and uncoupled eNOS and NOXs, contribute to the production of ROS in the kidneys of diabetic and obese patients[55,56].

Hyperglycemia-induced RON overproduction lowers amounts of catalase, thioredoxin reductase, & SOD (especially the manganese subtype) It also inhibits formation of reduced GSH. Furthermore, as a result of increasing ROS due to it's high necessity, non-enzymatic antioxidants spontaneously decrease. Since it starts defense by interacting with oxygen radical for hydrogen peroxide creation & is broken down by CAT and glutathione peroxidase, SOD is regarded as the primary physiological defense against ROS[53].

At low doses, RONs alter antioxidant enzyme transcription factors, which are crucial for OS attenuation.

NRF2 translocates to nucleus and activates genes to be copied from DNA to RNA that code for several enzymes by which it inhibits NF-kB. These defenses are ineffective at stopping or averting development of altered balance of reduction & oxidation when ROS overproduction occurs, as in DM[57].

RONs reduce SIRT enzymes which help in modulation & transformation through organic esterification of ETC. These enzymes are necessary for the stimulating PGC-1α & mitochondrial SOD. Additionally, hexosamine-derived O-GlcNAc attenuates SIRT activity, which exacerbates this process in DM. Decreased PGC-1α is linked to increased RON production in diabetic rats' kidneys via exacerbating mitochondrial fragmentation and[58].

A novel area of treatment has emerged as a result of the in vivo description of a progressive decline in antioxidant defenses in chronic renal disease. By supporting enzymatic & non-enzymatic antioxidant defense opposed to toxic substances, recent research has demonstrated the effectiveness of a increased antioxidant intake as an additional for diabetic nephropathy management[59,60].

Clinical features

Significant hazard factors for diabetic kidney disease are uncontrolled hypertension, poor control of blood glucose, & a prolonged duration of diabetes mellitus. High BMI, smoking, elevated lipid and cholesterol levels & diabetes or renal damage history in family are additional risk factors. Additionally, patients may exhibit concomitant diseases such retinopathy, elevated Blood pressure, CAD & PVD. Notably, as was already said, retina & nephron changes of diabetes are very strongly correlated[61,62].

Individuals who have diabetic nephropathy frequently share physical traits with other diabetics. Patients are usually asymptomatic in early stages, and screening for any ailment often reveals increased levels of protein elimination in urine of about 30 - 300 mg/g creatinine. Patients may exhibit symptoms like weariness, pedal edema from hypoalbuminemia and nephrotic syndrome, and foamy urine (a sign of urine protein (>3.5 g/d) as the condition worsens.

Other clinical features commonly seen with DM are[63,64] :

- Generalized tiredness
- Giddiness
- Excess thirst & excess urine
- Increased frequency of micturition
- Visual impairment – diminution of vision
- Paresthesias & peripheral nerve ending defects
- Wounds of feet with delay in healing
- Recurrent infections
- Hyperpigmented neck/axilla/groin regions – typical of type 2 DM
- Weight loss due to no apparent reason – typical of type 1 DM

Diagnosis

Proteinuria

The defining feature of diabetic nephropathy is proteinuria. Since the precise onset of Type 2 DM is sometimes unknown, diagnosing DKD in Type 2 DM is more difficult than in Type 1 DM. Diagnosing diabetic nephropathy in type 2 diabetes requires a thorough history and physical examination. While Type 2 DM patients should be examined for proteinuria when they're initially diagnosed and annually after, Type 1 DM patients are advised to be tested for proteinuria before 5 years of diagnosis. Aggressive treatment is necessary for increased proteinuria, which is a sign of deteriorating kidney function[63].

Diabetic nephropathy is incessant excretion of albumin in urine for about twice or more times, apart a span of 3 months. Excretion should be about 300 mg/d or higher. Moderate excretion can be seen in cases of early renal damage, causing 30-300 mg/d. Severe is described as >300 mg/d of urinary albumin excretion. Another way to describe moderately elevated albuminuria is a spot UACR of 20–200 mg/g[64].

Bio markers of urine

Other molecules are being investigated as possible indicators of diabetic nephropathy due to selective non-specificity & delayed albuminuria. Instead of concentrating only on the glomerulus, there has been an increase in interest in researching indicators of tubulointerstitial damage in recent years. DKD is very closely linked to non-albuminuric proteinuria, a sign of tubulointerstitial damage. Proximal tubular damage may even happen before glomerular injury, according to some data[65].

The most extensively researched markers for DKD are periostin, KIM-1, NGAL. Supposedly NGAL has 76% sensitivity & 55% specificity, KIM-1 had a 63% sensitivity and a 90% specificity, and periostin had an 80% sensitivity and a 66% specificity. Combining these biomarkers may improve the early identification of diabetic nephropathy, even though many of them are not yet generally accessible outside of research settings[66].

Treatment

When started before the onset of diabetes complications, intensive glycemic control is most beneficial; when started later, its effectiveness is diminished. Early, strict glycemic control is therefore strongly advised. Even after the study ended, T2D patients with better control of blood sugars & HbA_1c continued to have better microvascular outcomes and lower mortality, according to the United Kingdom Prospective Diabetes Study (UKPDS), even though their HbA_1c values were convergent between the two groups[67].

Similar outcomes were observed in Type 1 DM participants in DCCT. If HbA_1c is maintained <6.5% within the 1st year, the long-term advantages of early glucose-lowering medication have "legacy effect" or "metabolic memory." Although, some research has indicated that T2D patients have worse cardiovascular and all-cause outcomes as a result of hypoglycemia episodes linked to strict glycemic control. An HbA_1c target of roughly 7.0% is advised by the KDOQI and KDIGO guidelines in order to prevent the emergence of microvascular problems[67].

Fructosamine and glycated albumin are two additional, less popular techniques for assessing glycemic control; however, these tests lack adequate validation. Although HbA_1c is the best indicator of long-term glycemic management, it might not be a reliable indicator of severe hyperglycemia or hypoglycemia episodes, common in CKD. NKF-KDOQI recommendations propose a HbA_1c approximately 7.0%, however, specific goals should be set according on the patient's overall clinical status[68].

Use of ACEi & ARBs

The KDIGO guidelines allow for customization depending on patient-specific characteristics and recommend a blood pressure of <120/80 mm Hg for diabetics. Unless otherwise indicated, all diabetic individuals with hypertension are recommended to take ACEIs or ARBs. These drugs should also be titrated to the maximum amount that is tolerated. Insufficient research has been done on the use of ACEIs or ARBs in cases of albumin excretion in urine without HTN; therefore, each case should be evaluated separately. As part of their treatment, kidney transplant patients with T2DM & HTN require inhibition of RAAS.

Since stopping ACEIs or ARBs can increase risk of cardiac mortality, MI, & CVA, there is evidence to recommend the usage of these drugs in hypertensive dialysis patients. According to KDIGO guidelines, ACEI/ARB-associated hyperkalemia should be managed with rigorous dietary adherence and, if required, potassium binders[69].

The RENAAL & IDNT trials provide evidence of the advantages of ARBs slowing the damage of kidneys. Benefits of BP control on relative comorbidities, such as mortality, CVS events, & vessel damages, were emphasized by the UKPDS. Nonetheless, there were no appreciable variations in cardiovascular outcomes or the development of ESRD between intensive systolic blood pressure control (<120 mm Hg)[70].

The efficacy of ACEIs in lowering CKD with a eGFR of >60 mL/min/1.73m^2 was validated by the HOPE, LIFE, and ALLHAT trials. Furthermore, research like IRMA2 has demonstrated how beneficial ARBs are at preventing proteinuria in microalbuminuria patients. ACEIs have been demonstrated to reduce the progression of diabetic nephropathy in people with Type 1 DM and overt proteinuria. In T2D patients, the IDNT and RENAAL investigations showed comparable advantages. These studies offer compelling proof that, regardless of their impact on blood pressure, RAAS-blocking drugs aid in slowing the development of diabetic nephropathy. Nevertheless, it is no longer advised to use more than one RAS-blocking medication because this can result in negative effects, such as acute renal failure. Furthermore, it is not recommended to use RAAS inhibition to treat diabetic patients without albuminuria or hypertension.

Metformin & GLP-1 Receptor Agonists

T2D patients with CKD & a eGFR >30 mL/min/1.73m^2, ADA & KDIGO recommendations recommend metformin in addition to dietary changes as first-line treatment. Metformin has shown notable improvements in cardiovascular outcomes, all-cause mortality, and the progression of CKD. However, because they run the risk of developing lactic acidosis people of eGFR <45 mL/min/1.73m^2 should not begin using metformin. For eGFR 45 to 60 mL/min/1.73 m^2, the metformin dosage is be given only half the dose. In order to avoid problems from possible renal insults, metformin should also be avoided during inpatient stays. Strict surveillance of metformin is advised as there's a chance it may reduce vitamin B12 & folate levels[71].

Mineralocorticoid Antagonists

In cardiac and renal disorders, activation of mineralocorticoid receptors has been closely linked to fibrosis, inflammation, and unfavorable hemodynamic remodeling. Steroid mineralocorticoid antagonists, such as spirolactone and eplerenone, have shown promise, especially in people with HFrEF. Additionally, the substances demonstrated to successfully lower urinary protein loss in CKD, similar advantages in urinary protein loss brought on by DM & other[72].

A nonsteroidal mineralocorticoid antagonist that is specific and authorized to the management of CKD linked to Type 2 DM is finrenone. The mineralocorticoid receptor is passively and bulkily antagonistic to finrenone. Numerous trials, such as FIDELITY-DKD, FIGARO-DKD, and FINEARTS-HF, have indicated that the medicine improves kidney parenchymal & CVS outcomes and lowers albuminuria in individuals with kidney disease[73].

Although the Food and Drug Administration (FDA) has not approved esaxerenone, which functions similarly to finerenone, used in Japan & other nations and demonstrated to lower urinary albumin loss in people with T2D.

Na-Glucose Cotransporter-2 Inhibitors

In the proximal tubule, SGLT2Is inhibit glucose reabsorption, which results in decreased albuminuria, GFR loss, and metabolic strain on nephrons, as well as increased glucosuria and decreased capillary hypertension. Additionally, they reduce glomerular hypertension and energy expenditure by mitigating macula densa salt hypersensitivity. Stimulation of hypoxia-inducible factors (HIFs), promote the generation of erythropoietin, is another important mechanism.

Beyond regulating blood sugar, SGLT2Is have other positive effects. These drugs encourage a change in metabolism from the use of carbohydrates to the use of fats, which reduces visceral and subcutaneous fat and causes weight loss in general. As a result of this process, free fatty acids are transformed into ketone bodies, which provide cardiac and renal cells with energy. Blocking glucose reuptake lowers concomitant dissolution of salt, chloride & water, is another renoprotective action of SGLT2Is. This decrease maintains GFR by reducing the glomerular hyperfiltration that is frequently seen in diabetes[74].

Renal Replacement

When eGFR reaches 10 to 15 mL/min/1.73 m2, ESRD may occur and RRT could be necessary. Dialysis alternatives are kidney transplantation, HD, peritoneal dialysis. When a patient's GFR drops to about 20 mL/min/1.73m^2, they should be referred to a transplant clinic. Renal transplantation is typically recommended for patients with good functional status. According to a study, 47% of patients waiting for a kidney transplant also have diabetes; this number is predicted to rise.

Prognosis

Diabetic kidney disease has high chances of long term morbidity & mortality. Urinary albumin loss is a vital risk factor for CVS risk, & most of them eventually develop ESRD. It is mostly seen with retinal changes secondary to diabetes as well.

REVIEW OF RELATED LITERATURE ON PFTS IN PEOPLE WITH DIABETIC KIDNEY DISEASE

In a study by Chintalapati et al, Mean FEV 1% was 98.12 (+/-10.06), 75.88 (+/-14.10) and 57.64 (+/-13.49), Mean FVC% was 86.78 (+/- 8.77), 69.82(+/-13.88) and 53.02(+/-13.41), the difference observed was statistically significant (p value <0.001). Patients with diabetes showed restrictive pattern in PFT & seen to be more severe in the group with diabetic nephropathy. Among patients with diabetes for <10 years, 10 to 20 years, & >20 years, respectively, the mean FEV1% was 71.03 (+/-13.19), 66.74 (+/-18.34), and 60.29 (+/-15.25), while the mean FVC% was 65.66 (+/-11.06), 60.79 (+/-18.00), and 56.38 (+/-16.55). The mean FEV1% and FVC% differences with DM duration were not statistically significant. In the diabetes mellitus group, PAH was present in 3 patients (6%) while it was prevalent in 20 patients (40%) of diabetic nephropathy & difference being statistically significant (p < 0.001) in both groups. According to this study, diabetics have a restrictive pattern in their pulmonary function tests, and their impairment is particularly severe in those who have nephropathy. Diabetes duration has no effect on the dysregulation of pulmonary functioning. In the group of people with diabetic nephropathy, pulmonary hypertension was more prevalent[79].

In one study by Shah et al, except FEV1/FVC, the PFTs were considerably lower in diabetes patients than in healthy controls. There was no connection between HbA1c, FVC, or FEV1 and the length of the disease. DM also affects the lungs, leading to restrictive type in PFT, reduced elastic recoil, & inflammatory changes. Authors discovered that glucose levels & disease duration are probably not main factors influencing lung damage, indicating a need for additional investigation[4].

Another study by Gupta et al, age ranged 61 to 70 years saw the greatest number of patients. The control group's mean age was 55.84 years, compared to the patients' mean age of 59.38 years. The patients' gender distribution revealed a ratio of 66:34 for men to women, compared to 64% men to 36% women in the control group. HbA1c levels in cases (8.23) were substantially higher than in the control group (5.02). In comparison to controls, who had a mean of 102.24 & range of 82.13-122.35, Type 2 DM patients had a mean of 67.96 with a range of 48.19-87.73. HbA1c revealed a substantial correlation with age distribution. The age range with the greatest HbA1c was above 70 years old (10.73), followed by 51 to 60 years (7.92), while the age range with the lowest HbA1c was under 40 years (4.33). They concluded that, compared to controls, Type-2 DM people showed lower FVC and FEV1 values, yet raised FEV1/FVC% ratio. Length of DM and the FEV1% Pred & FVC% Pred were negatively correlated. Increasing length and FEV1/FVC% have a linear connection, which suggests a restrictive lung condition. The spirometry values FVC and FEV1 continuously declined as the HbA1c level rose. In order to identify pulmonary problems, patients with type 2 DM are advised to undergo PFTs periodically[75].

Another study by Kaur et al, in diabetics correlated to controls, all PFT measures (FVC%, FEV1%, & FEV1/FVC) significantly decreased. Hence, diabetics exhibit a mixed pattern in PFT. Moreover, a significant positive connection between FBS & FEV1/FVC was observed in patients with DM. But, the study group's body mass index (BMI) was more compared to controls, & difference wasn't statistically significant. Also, to minimise complications & improve QOL, pulmonary function tests for diabetics should be required because the condition does affect the lungs[76].

MATERIALS AND METHODS

MATERIAL AND METHODS

A study which was of cross sectional type was done at RLJH in the General Medicine Department in a high-level teaching hospital in Tamaka, Kolar which lasted for a period of 1 and a half year - between May 2023 to October, 2024. Among all adults, patients screened for and diagnosed as Diabetes Mellitus Type 2 were included in the study, with and without diabetic nephropathy. The ethical committee of the institution provided clearance before the start of the study. Before including the patients in the study, they were informed in detail and proceeded with their consent.

Sample Size: 100.

It was evaluated by using the disparity in Mean FEV_1% betwixt Diabetic Nephropathy & Diabetics without nephropathy from a previous article Ashrith et. al. which has 57.64 ± 13.49 min and 98.12 ± 10.06. At 99% Confidence limit & power sample of 90%, a sample size of 27 is calculated for each group by the Med calc software and using the aforementioned formula. Including the 10% non-response, we arrived at a sample size of 27 + 2.7 ≈ 30 subjects were finalized as the study size in each group.

Minimal sample size is 30. But in this study, 50 subjects each were considered for the two groups.

Estimation Formula Of Sample Size –

$$N = \frac{2\,SD^2(Z\alpha_{/2} + Z\beta)^2}{d^2}$$

1. Where Z alpha/$_2$ - critical value of the normal distribution at $_{\alpha/2}$ (i.e. for a confidence level of 99%, **α** will be 0.01 & the critical value is 2.57).
2. Z beta- critical value of the normal distribution at β (i.e. for a power of 90%, ß is 0.1 & the critical value is 1.28),
3. SD - Standard Deviation of variance from study population of previous study
4. d - largest difference between two mean values

The criteria of inclusion and exclusion was -

Criteria of inclusion –

Patients above the age of 18.

Patients diagnosed as Diabetes Mellitus Type 2

Criteria of exclusion –

1. Nephropathy through other causes
    - ❖ Glomerulopathies
    - ❖ Obstructive uropathy
    - ❖ Renal Macrovascular Disease
    - ❖ Chronic Tubulointerstitial nephropathies
    - ❖ Obesity related nephropathy

Methodology:

1. This study was done after obtaining ethical clearance from the institutional ethical committee as well as consents from the Individuals / study subjects.
2. 50 individuals in each group fulfilling inclusion & exclusion criteria were included.
3. Detailed and thorough history was recorded and examination was done.
4. The patient underwent all the necessary investigations.
5. All patients admitted with Type 2 DM coming to Department of Medicine OPD was taken into study and estimating the prevalence of patients with Nephropathy.
6. Pulmonary Function Test was assessed in Diabetic Nephropathy Patients
7. Comparing PFT in patients with & without nephropathy

Statistical Analysis Of The Study :

After entering data into Microsoft excel & analyzing using SPSS 22 version software, the data which was obtained was categorically exhibited in the form of proportions and frequencies. Significance was checked using the Chi-square test. Mean and standard deviation were the modes used to express the data collected. To identify the mean difference between the two parties, Independent t test was used. The study was deemed to be considered as statistically significant if P value <0.05.

RESULTS

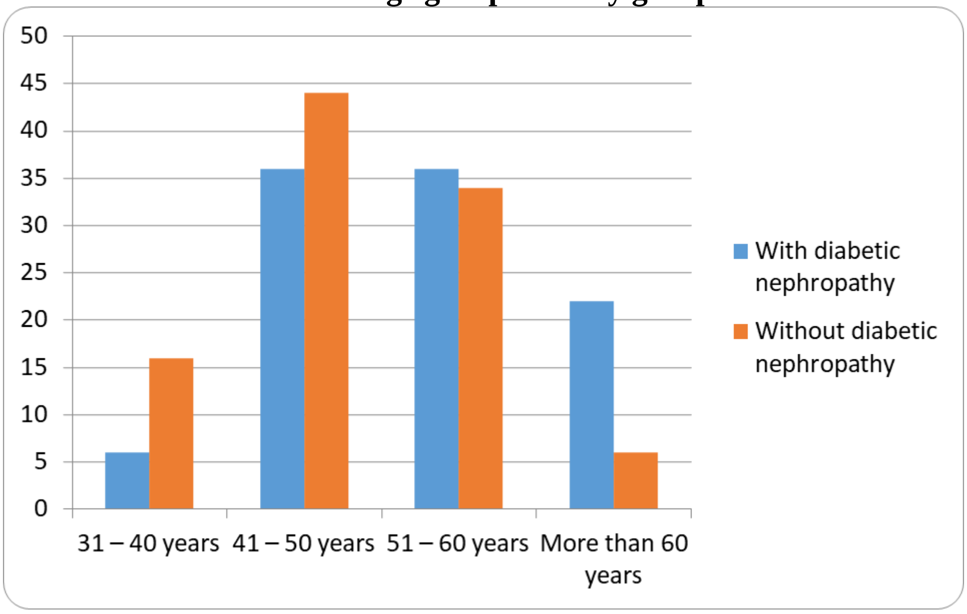
RESULTS

Table 1. Age group of study groups

Age group	With diabetic nephropathy n (%)	Without diabetic nephropathy n (%)
31 – 40 years	3 (6.0)	8 (16.0)
41 – 50 years	18 (36.0)	22 (44.0)
51 – 60 years	18 (36.0)	17 (34.0)
More than 60 years	11 (22.0)	3 (6.0)
Total	50 (100)	50 (100)

χ^2 value = 7.273 df = 3 p value, Sig = 0.064, NS

Chart 1. Age group of study groups



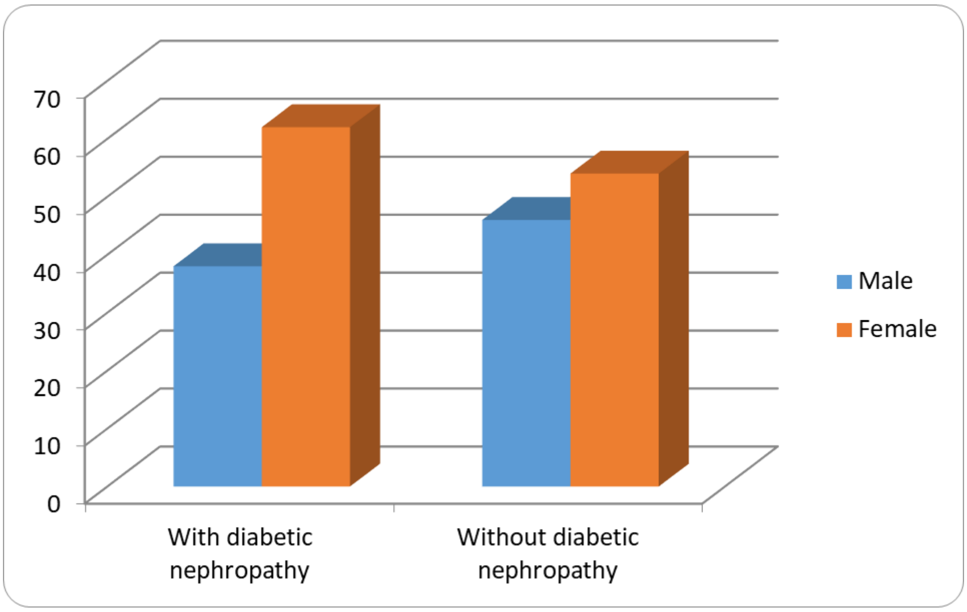
In this study, people aged between 41 – 50 years are - about 36.0% of cases are of diabetic nephropathy and 44/0% cases are of without diabetic nephropathy. Statistically, this difference was not significant among the two above mentioned study groups.

Table 2. Sex of study groups

Sex	With diabetic nephropathy n (%)	Without diabetic nephropathy n (%)
Male	19 (38.0)	23 (46.0)
Female	31 (62.0)	27 (54.0)
Total	50 (100)	50 (100)

χ^2 value = 0.657 df = 1 p value, Si g = 0.418, NS

Chart No 2 - Sex of the study groups

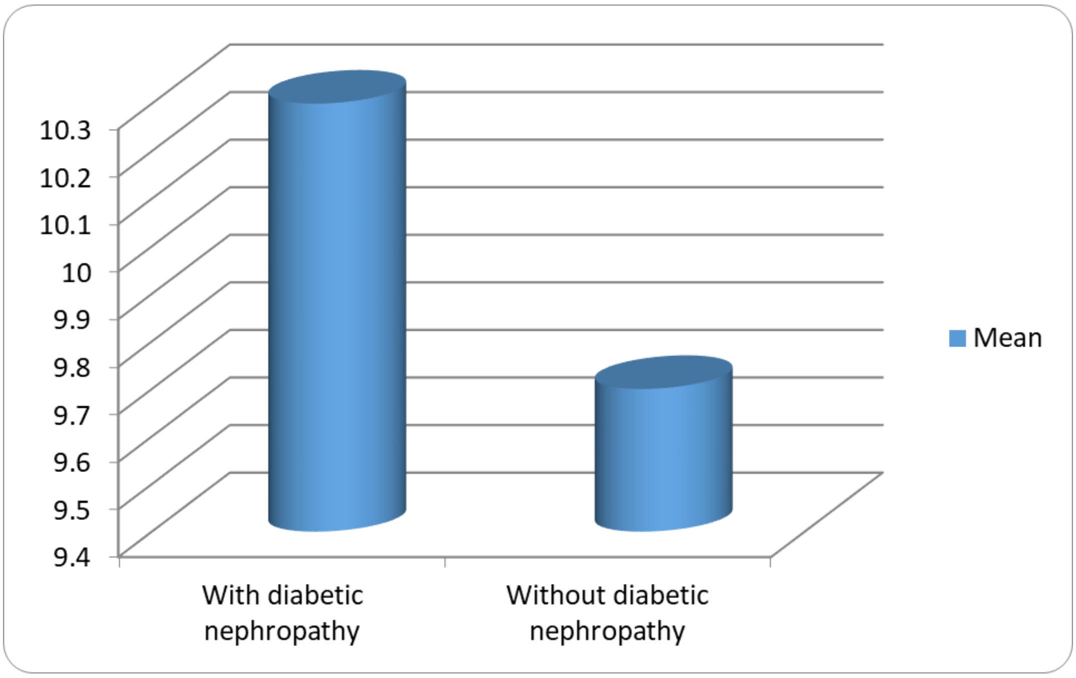


This study had shown that, about 62.0% of the cases with diabetic nephropathy and 54.0% without diabetic nephropathy were of female population. Statistically, this difference was not significant among the two above mentioned study groups.

Table 3. Duration of diabetes mellitus in study groups

Duration of diabetes mellitus	With diabetic nephropathy	Without diabetic nephropathy	T value	P value, Sig
Mean ± SD	10.3 ± 7.4	9.7 ± 7.3	0.394	0.694, NS

Chart 3. Duration Of Diabetes Mellitus In The Study Groups



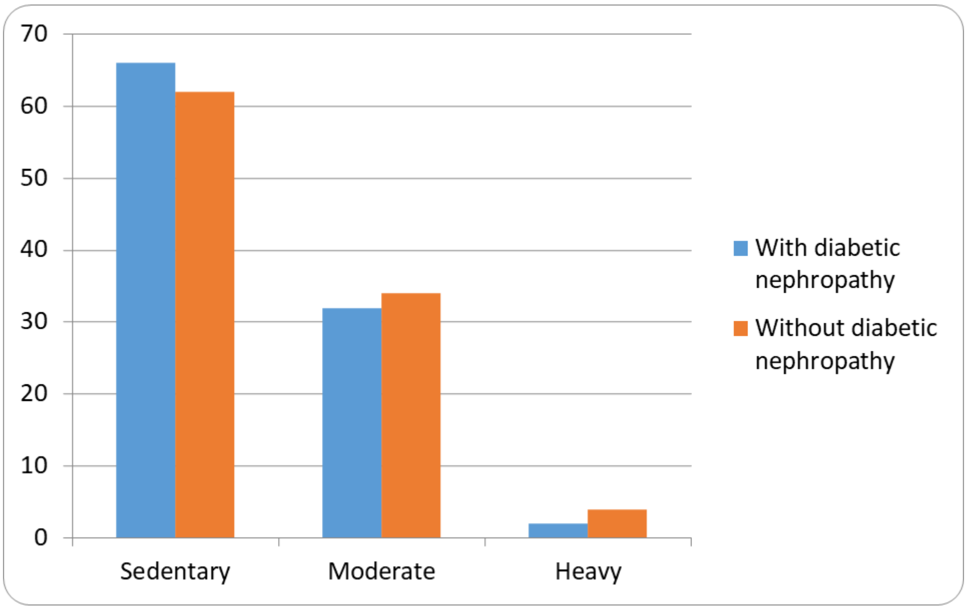
Mean duration of diabetes mellitus in cases with diabetic nephropathy was 10.3 years and in cases without diabetic nephropathy was 9.7 years. Statistically, this difference was not significant among the two above mentioned study groups.

Table 4. Occupation of the study groups

Profession	With diabetic nephropathy n (%)	Without diabetic nephropathy n (%)
Sedentary	33 (66.0)	31 (62.0)
Moderate	16 (32.0)	17 (34.0)
Heavy	1 (2.0)	2 (4.0)
Total	50 (100)	50 (100)

χ^2 value=0.426 df=2 p value, Sig=0.808, NS

Chart 4. Occupation of study groups



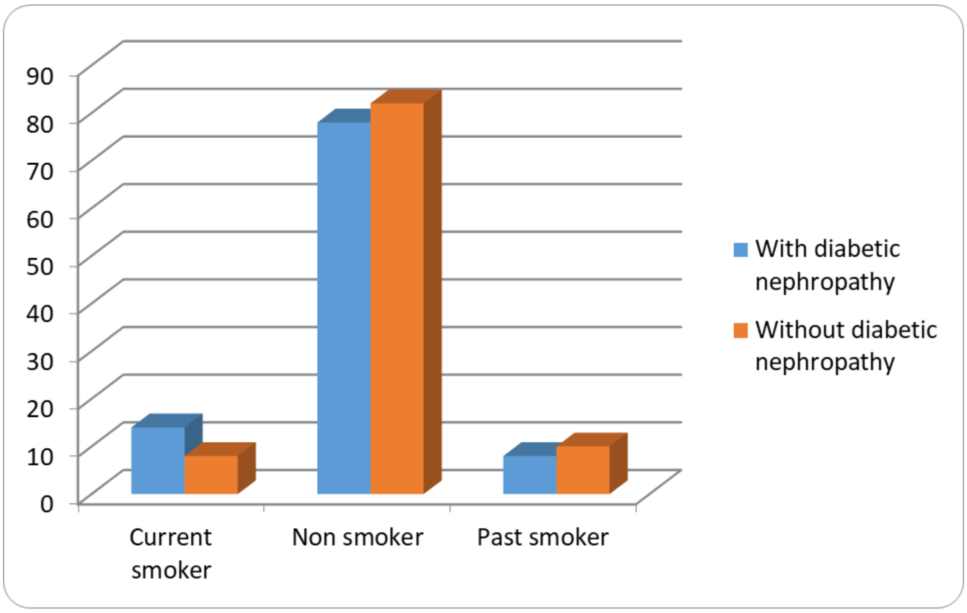
About 66.0% of the cases with diabetic nephropathy and 62.0% without diabetic nephropathy were having a sedentary lifestyle. Statistically, this difference was not significant among the two above mentioned study groups.

Table 5. Smoking in study groups

Smoking	With diabetic nephropathy n (%)	Without diabetic nephropathy n (%)
Current smoker	7 (14.0)	4 (8.0)
Non smoker	39 (78.0)	41 (82.0)
Past smoker	4 (8.0)	5 (10.0)
Total	50 (100)	50 (100)

χ^2 value = 0.979 df = 2 p value, Sig = 0.613, NS

Chart No. 5 - Smoking in study groups



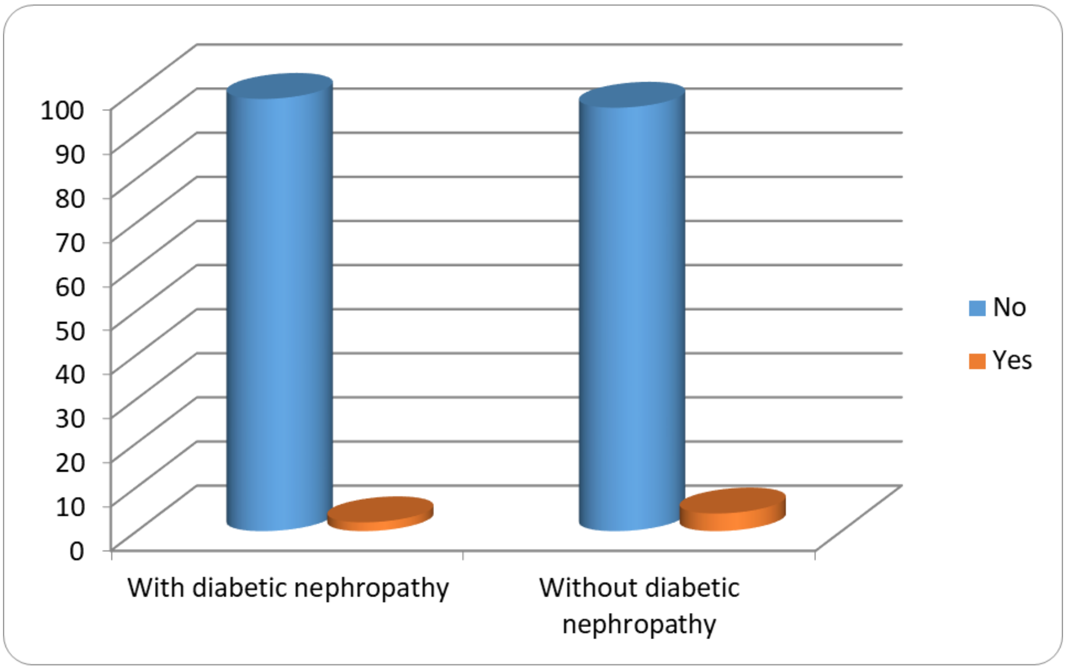
About 14.0% of the cases with diabetic nephropathy and 8.0% without diabetic nephropathy were currently still smoking. Statistically, this difference was not significant among the two above mentioned study groups.

Table 6. Alcohol use in study groups

Alcohol consumption	With diabetic nephropathy n (%)	Without diabetic nephropathy n (%)
No	49 (98.0)	48 (96.0)
Yes	1 (2.0)	2 (4.0)
Total	50 (100)	50 (100)

χ^2 value = 0.344 df=1 p value, Sig = 0.558, NS

Chart 6. Alcohol use in study groups



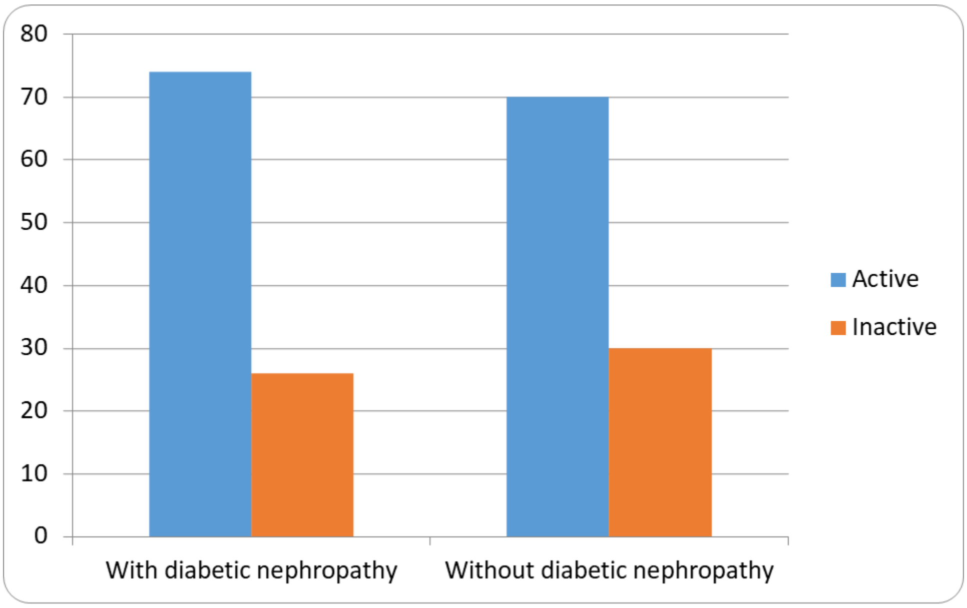
About 2.0% of the cases with diabetic nephropathy and 4.0% without diabetic nephropathy were using alcohol. Statistically, this difference was not significant among the two above mentioned study groups.

Table 7. Physical activity in study groups

Physical activity	With diabetic nephropathy n (%)	Without diabetic nephropathy n (%)
Active	37 (74.0)	35 (70.0)
Inactive	13 (26.0)	15 (30.0)
Total	50 (100)	50 (100)

χ^2 value = 0.198 df = 1 p value, Sig = 0.658, NS

Chart 7. Physical activity in study groups



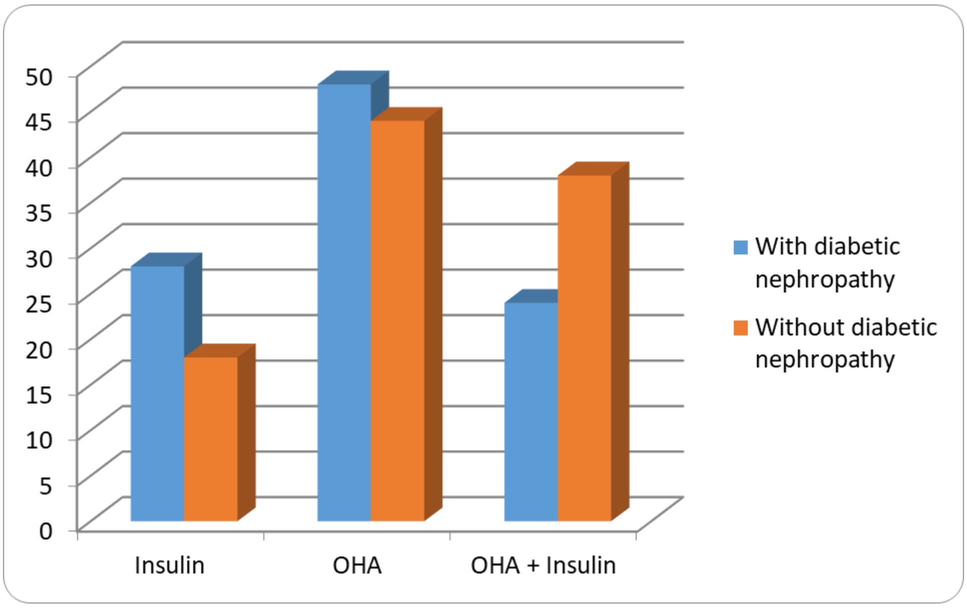
About 26.0% of the cases with diabetic nephropathy and 30.0% without diabetic nephropathy were physically inactive. This difference was not statistically significant.

Table 8. Antidiabetic medication in the study groups

Drug	With diabetic nephropathy n (%)	Without diabetic nephropathy n (%)
Insulin	14 (28.0)	9 (18.0)
OHA	24 (48.0)	22 (44.0)
OHA + Insulin	12 (24.0)	19 (38.0)
Total	50 (100)	50 (100)

χ^2 value=2.755 df=2 p value, Sig=0.252, NS

Chart 8. Antidiabetic medication in the study groups

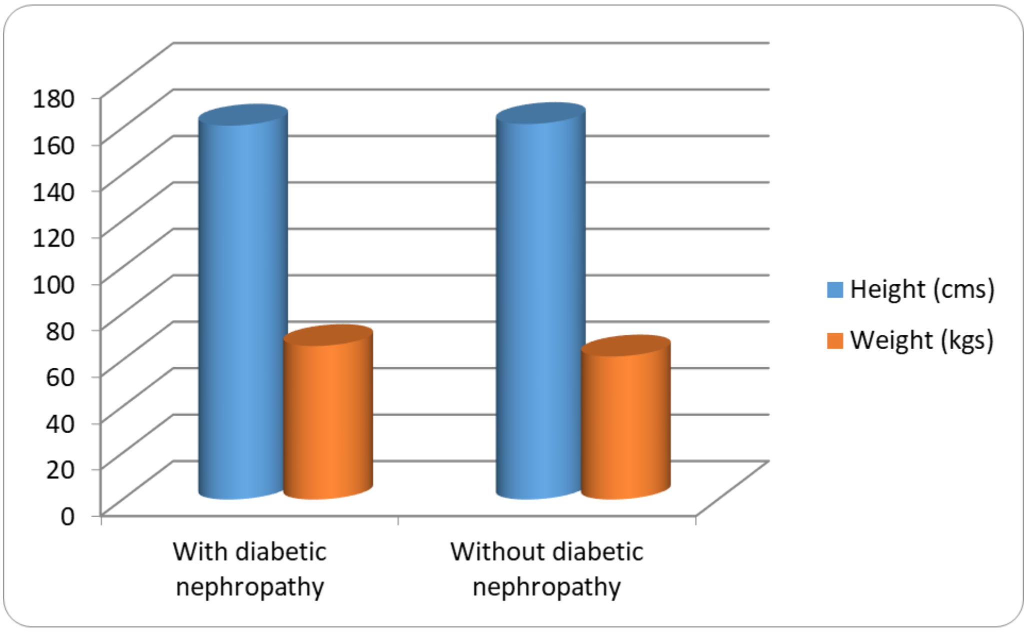


About 28.0% of the cases with diabetic nephropathy and 18.0% without diabetic nephropathy were using insulin. This difference was not statistically significant.

Table 9. Anthropometry in the study groups

Anthropometry (Mean ± SD)	With diabetic nephropathy	Without diabetic nephropathy	T value	P value, Sig
Height (cms)	161.0 ± 4.6	161.7 ± 5.2	0.712	0.478, NS
Weight (kgs)	66.1 ± 5.7	61.6 ± 7.3	3.419	0.001, Sig

Chart 9. Anthropometry in the study groups

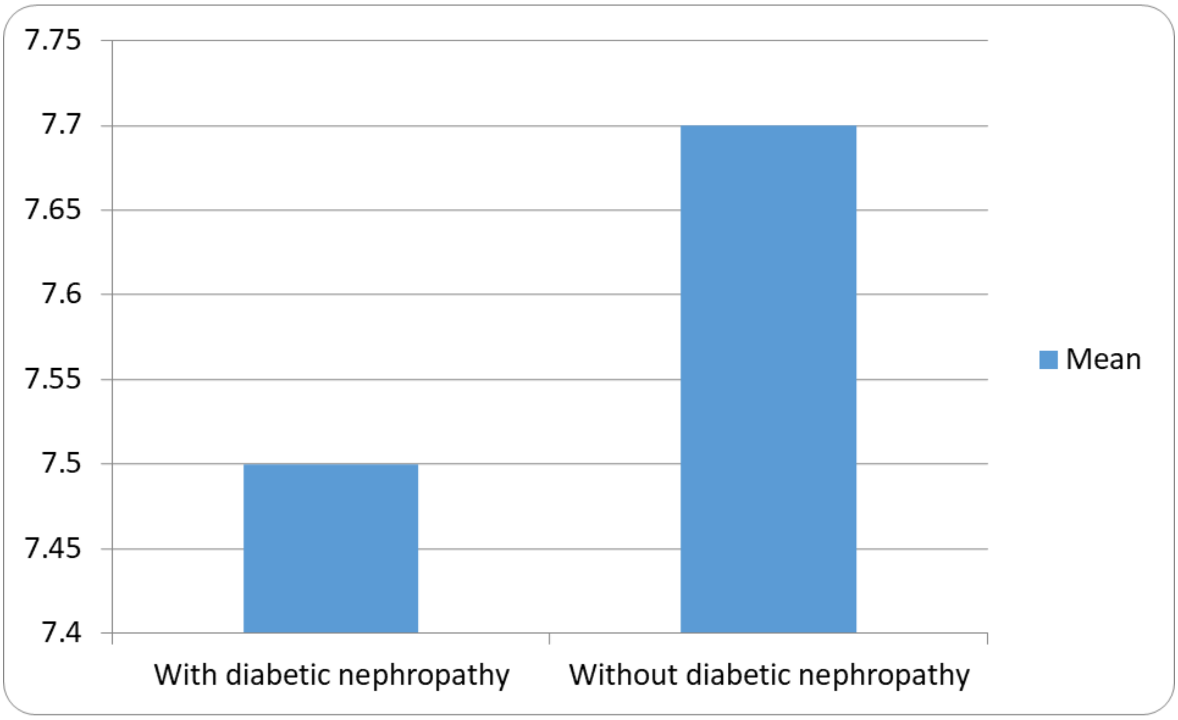


Mean Height in cases with diabetic nephropathy was 161 cms and 161.7 cms in cases without diabetic nephropathy was not statistically significant. Mean weight in cases with diabetic nephropathy was 66.1 kgs and 61.6 kgs in cases without diabetic nephropathy. This difference was statistically significant.

Table 10. HbA_1c in the study groups

HbA_1c (%)	With diabetic nephropathy	Without diabetic nephropathy	T value	P value, Sig
Mean ± SD	7.5 ± 1.1	7.7 ± 1.2	0.597	0.552, NS

Chart 10. HbA_1c in the study groups

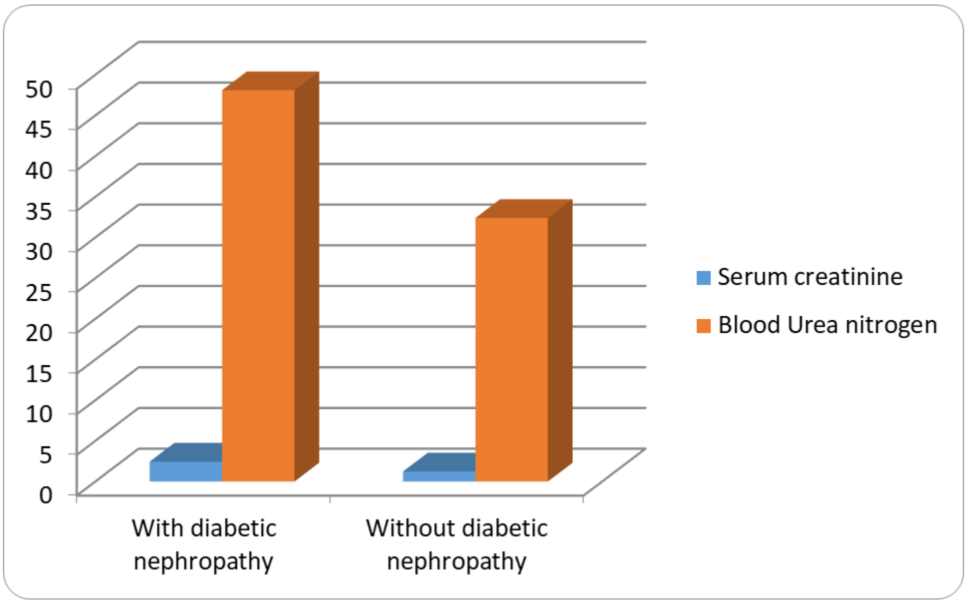


Mean HbA_1c in cases with diabetic nephropathy was 7.5 gms% and 7.7 gms% in cases without diabetic nephropathy. Statistically, this difference was not significant among the two above mentioned study groups.

Table 11. Renal function in the study groups

Renal function tests (Mean ± SD)	With diabetic nephropathy	Without diabetic nephropathy	T value	P value, Sig
Serum creatinine	2.44 ± 0.62	1.23 ± 0.6	10.129	0.000, Sig
Blood Urea nitrogen	48.1 ± 28.8	32.4 ± 12.1	3.553	0.001, Sig

Chart 11. Renal function in the study groups



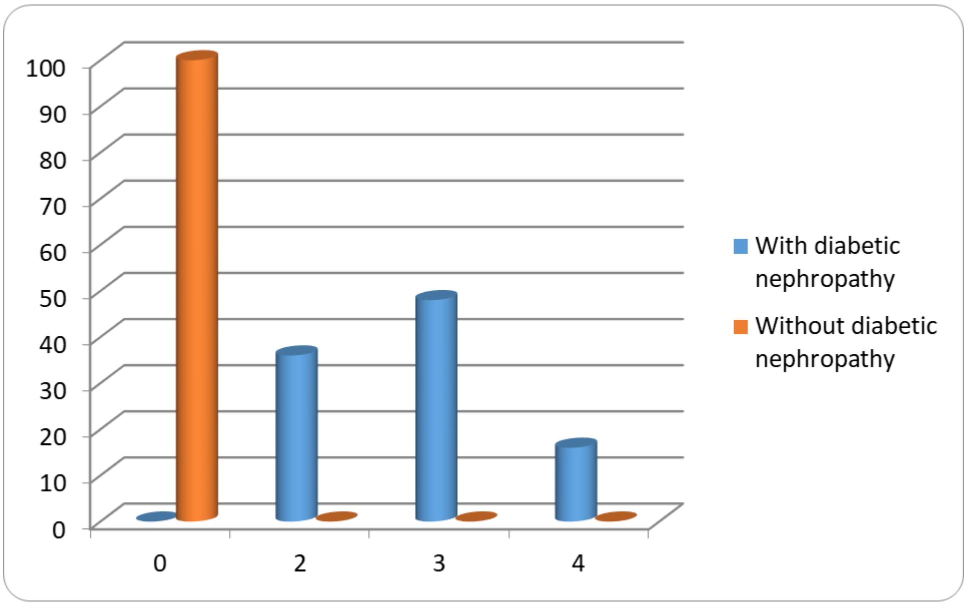
The mean value of creatinine in diabetic nephropathy cases group was 3.44 mg/dl and 1.23 mg/dl in cases without diabetic nephropathy. Statistically, this difference was significant among the two above mentioned study groups.

Table 12. Stage Of Kidney Disease In The Study Groups

Stage of kidney disease	With diabetic nephropathy n (%)	Without diabetic nephropathy n (%)
0	0	50 (100.0)
2	18 (36.0)	0
3	24 (48.0)	0
4	8 (16.0)	0
Total	50 (100)	50 (100)

χ^2 value=100.0 df=3 p value, Sig = 0.000, Sig

Chart 12. Stage Of Kidney Disease In The Study Groups

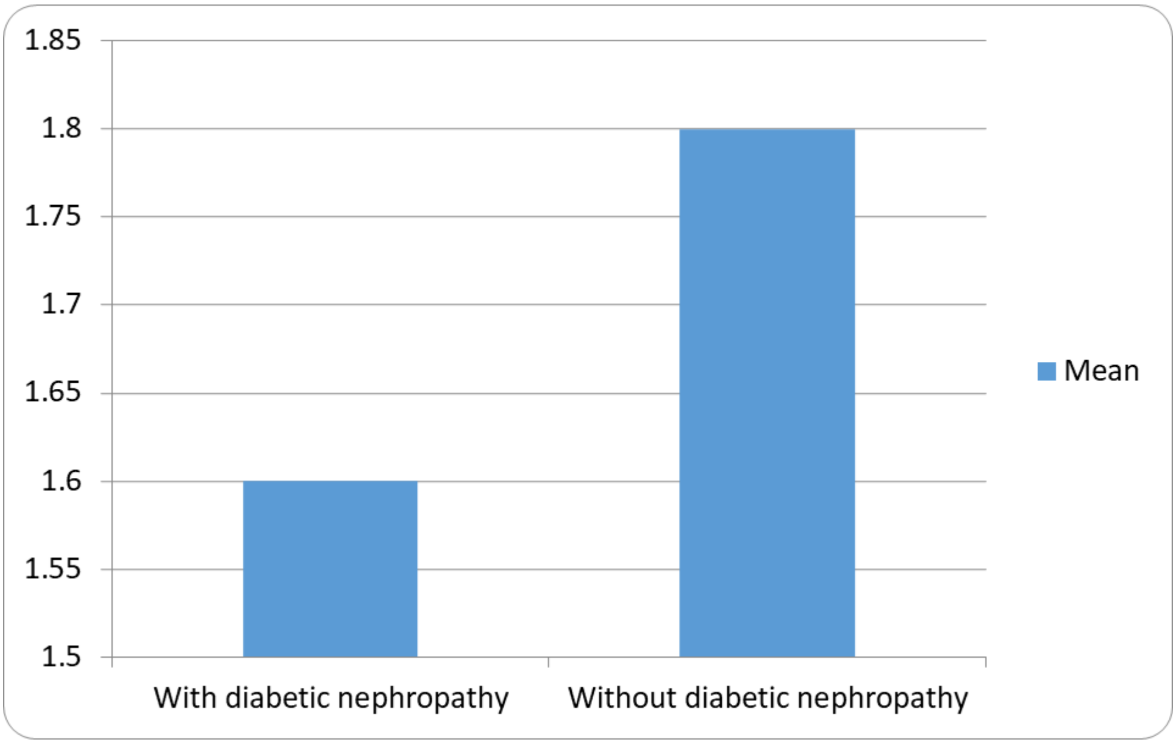


About 36.0% of the cases with diabetic nephropathy had stage 2 kidney disease, 48.0% had stage 3 and 16.0% had stage 4 kidney disease in this study. This difference was statistically significant between the two groups.

Table 13. FEV_1 in the study groups

FEV_1	With diabetic nephropathy	Without diabetic nephropathy	T value	P value, Sig
Mean ± SD	1.6 ± 0.51	1.8 ± 0.55	2.164	0.033, Sig

Chart 13. FEV_1 in the study groups

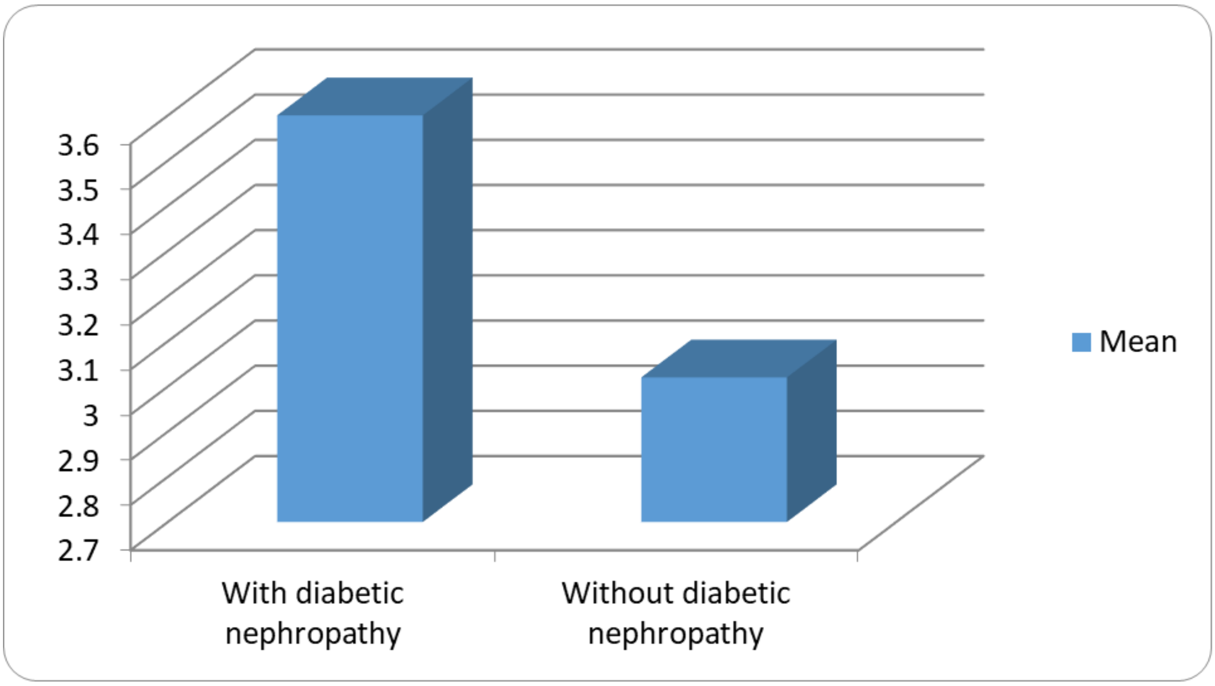


Forced expiratory volume 1 was 1.6 litres in cases with diabetic nephropathy and 1.8 litres in cases without diabetic nephropathy. Statistically, this difference was not significant among the two above mentioned study groups.

Table 14. FVC in the study groups

FVC	With diabetic nephropathy	Without diabetic nephropathy	T value	P value, Sig
Mean ± SD	3.6 ± 1.53	3.02 ± 0.92	2.332	0.000, Sig

Chart 14. FVC in the study groups

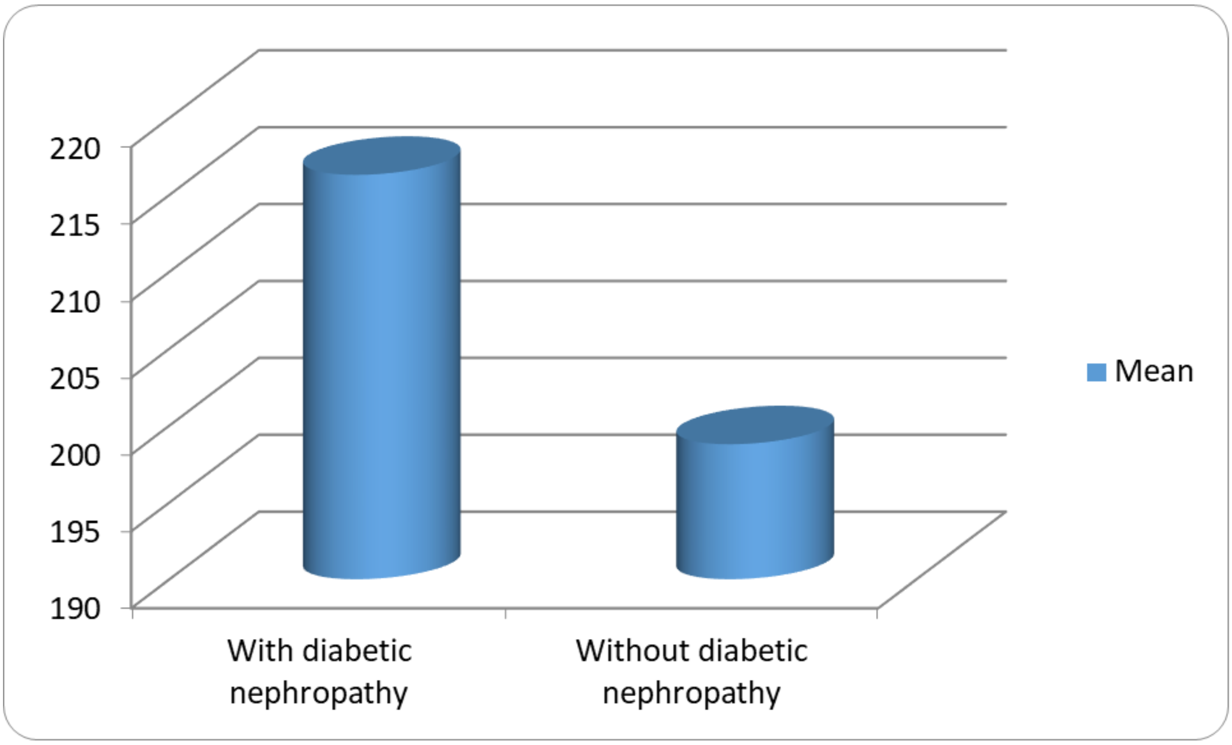


Mean functional vital capacity was 3.6 litres in cases with diabetic nephropathy and 3.02 litres in cases without diabetic nephropathy. This difference was a statistically significant between the two groups.

Table 15. PEFR in the study groups

PEFR	With diabetic nephropathy	Without diabetic nephropathy	T value	P value, Sig
Mean ± SD	216.3 ± 44.81	198.78 ± 47.74	1.894	0.061, NS

Chart 15. PEFR in the study groups

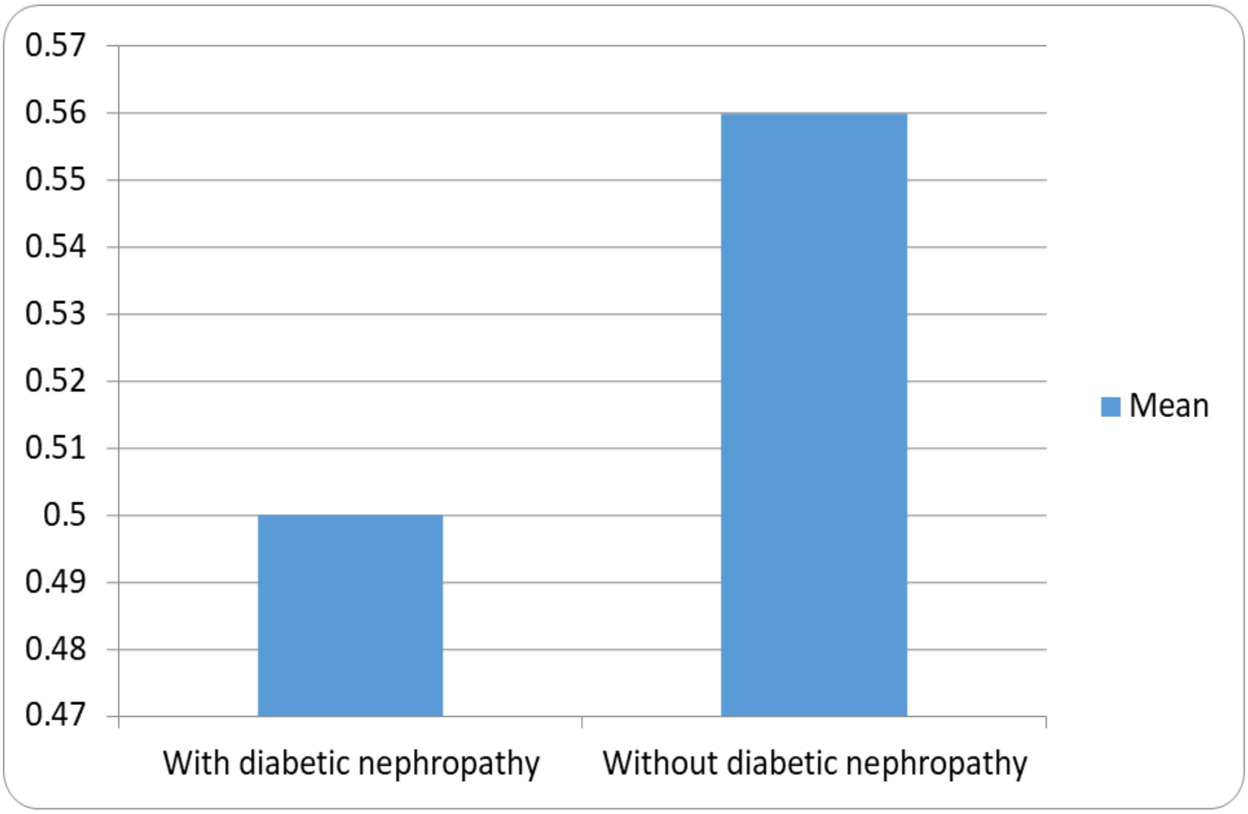


Peak expiratory flow rate in cases with diabetic nephropathy was 216.3 and 198.78 in cases without diabetic nephropathy. This difference was not statistically significant between the two groups

Table 16. FEV_1/FVC ratio in the study groups

FEV_1/ FVC	With diabetic nephropathy	Without diabetic nephropathy	T value	P value, Sig
Mean ± SD	0.50 ± 0.15	0.56 ± 0.11	2.271	0.025, Sig

Chart 16. FEV_1/FVC ratio in the study groups



Mean FEV_1/FVC ratio in cases with diabetic nephropathy was 0.50 and 0.56 in cases without diabetic nephropathy. This difference was statistically significant between the two groups.

DISCUSSION

DISCUSSION

DM is defined by elevated serum glucose levels which are observed due to defect in insulin functioning or production, or both. The secondary pathophysiologic alterations in several organ systems is observed due to altered regulation of metabolism in diabetic people which is causing a significant burden in the structure of medical care. Diabetes-related chronic hyperglycemia is linked to a number of metabolic abnormalities that, over time, cause harm, dysfunction, and leading to multi-system failure – cardiomyopathy, atherosclerosis, nephropathy, retinopathy and neuropathies[76]. Although this is widely known, less attention has been paid to DM's pulmonary consequences. Knowledge regarding the cellular and molecular pathophysiology of this illness is growing. It is increasingly believed that diabetes should be treated as an endothelial illness rather than only a metabolic disease, and that the numerous manifestations of the multiorgan involvement are mostly caused by endothelial dysfunction[77].

Microangiopathic complications of DM involving kidney, nerve & eye have long been understood and treated from a variety of angles. However, diabetes-related microangiopathy may have an impact on the lung's alveolar capillary network, a sizable microvascular unit which hasn't been studied in detail yet. ANS acting on phrenic nerve, causing reduced tone of the diaphragmatic muscle associated with loss of control of diaphgram has been found to contribute to the pulmonary function abnormalities. Additionally, this microangiopathy in the lungs can cause changes in alveolar function, which can show up as abnormalities in pulmonary function[78].

The primary goal of aforementioned study is to give us a brief idea on how lungs function in diabetic kidney disease cases.

Age group

In this study, people aged 41 to 50 made up about 36.0% of the cases with diabetic nephropathy and 44.0% of the cases without it. In one study by Ashrith et al, average age of cases without DM, w/o diabetic nephropathy & with diabetic nephropathy was 58.6 years, 59.8 years and 59.96 years respectively [79] . Another study by Selvaraj et al, showed that among the cases about 30% were aged between 41 – 45 years [80] .

Gender

According to this study, women made up roughly 62.0% of cases with diabetic nephropathy and 54.0% of cases without it. In a study by Ashrith et al, the proportion of males and females was same [79] . Another study by Selvaraj et al, showed that among the cases, about 54% were males [81] .

Duration of diabetes mellitus

The average duration of diabetes mellitus was 9.7 years for those without diabetic nephropathy and 10.3 years for those with it. In a study by Ashrith et al, average duration of diabetes mellitus of cases without nephropathy found to be 10.42 years & 14.18 years in cases with diabetic nephropathy [79] .

Type of work

Sedentary workers accounted for roughly 66.0% of individuals with diabetic nephropathy and 62.0% of those without it.

Smoking

Current smokers made up about 8.0% of individuals without diabetic nephropathy and 14.0% of cases with it.

Alcohol use

Alcohol use was present in about 2.0% of individuals with diabetic nephropathy and 4.0% of cases without it.

Physical activity

Physical inactivity was present in about 30.0% of cases without diabetic nephropathy and 26.0% of cases with it.

Antidiabetic used

Insulin was used in about 18.0% of individuals without diabetic nephropathy and 28.0% of those with it.

Anthropometry

The mean height in individuals with diabetic nephropathy was 161 cm, while the mean height in individuals without the condition was 161.7 cm. The average weight of individuals with diabetic nephropathy was 66.1 kg, while those without the condition weighed 61.6 kg.

HbA_1c levels

The mean HbA_1c was 7.5 gms% in diabetic nephropathy cases and 7.7 gms% in non-diabetic nephropathy cases.

Serum creatinine

Mean serum creatinine in cases with diabetic nephropathy was 3.44 mg/dl & it was 1.23 mg/dl in those without diabetic kidney disease.

Stage of kidney disease

About 36.0% of the cases with diabetic nephropathy had stage 2 kidney disease, 48.0% had stage 3 and 16.0% had stage 4 kidney disease in this study.

Forced Expiratory Volume

Forced expiratory volume 1 was 1.6 litres in cases with diabetic nephropathy and 1.8 litres in cases without diabetic nephropathy. In one study by Ashrith et al, average FEV_1 found to be 98.12% in cases without diabetes mellitus, 75.88% in cases without diabetes nephropathy and 57.64% in cases with diabetic nephropathy[79]. Another study by Selvaraj et al, mean FEV_1 was 68.37% in cases of DM of type 2 & 77.27% in non-diabetics[80]. Another study by Patel et al, average FEV_1 of diabetic nephropathy cases was 2.45 L and 2.65 litres in healthy controls[81].

Functional Vital Capacity

Mean functional vital capacity was 3.6 litres in cases with diabetic nephropathy and 3.02 litres in cases without diabetic nephropathy. In a study by Ashrith et al, the mean FVC% was 86.78 in cases with no diabetes, 69.82 in cases with diabetes mellitus with no nephropathy and 53.02 in cases with diabetic nephropathy[79]. In a study by Selvaraj et al, the mean FVC% was 68.18 in cases with diabetes mellitus and 84.52% in non-diabetic cases[80]. In a study by Patel et al, the mean FVC was 2.95 in diabetic nephropathy cases and 3.25 in healthy controls[81].

Peak Expiratory Flow Rate

Peak expiratory flow rate in cases with diabetic nephropathy was 216.32 and 198.78 in cases without diabetic nephropathy. Mean PEF% in a study by Ashrith et al was 88.62% in cases with no diabetes, 59.4% in cases with diabetes mellitus without nephropathy and 48.96% in cases with diabetes mellitus with nephropathy[79]. In a study by Patel et al, the mean peak expiratory flow rate was 450 L/min in diabetic nephropathy cases and 500 L/min in controls[81].

FEV_1/FVC ratio

Mean FEV_1/FVC ratio in cases with diabetic nephropathy was 0.50 and 0.56 in cases without diabetic nephropathy. In a study by Ashrith et al, mean FEV_1/FVC% was 91.74 in cases without diabetes mellitus, 88.88% in cases with diabetes mellitus without nephropathy and 89.48% in cases with diabetes mellitus and nephropathy[79]. In a study by Selvaraj et al, the mean FEV_1/FVC% was 100.66 in type 2 diabetes mellitus cases and 90.8% in non-diabetes cases[80]. In a study by Patel et al, FEV_1/FVC ratio was 82.3% in cases with diabetic nephropathy and 83.5% in healthy controls[81].

LIMITATIONS

LIMITATIONS

- This study was limited to one centre only.
- This study had not used any sampling methods.
- The sample size was small to generalize the result.

CONCLUSION

CONCLUSION

The study was undergone in order to examine how pulmonary functions will be affected in diabetic nephropathy cases in comparison with cases without nephropathy.

This study shown that, the FEV_1, FEV_1/FVC were low in diabetic nephropathy cases when compared with diabetic cases without nephropathy while FVC, PEFR was about the same in both the groups.

This study results were similar to the study results of the studies reviewed and has brought out important facts about PFTs in cases of diabetic nephropathy.

RECOMMENDATIONS

- A multicentric study can be conducted in order bring out more facts about pulmonary function tests.

SUMMARY

SUMMARY

- The study was undergone in order to examine how pulmonary functions will be affected in diabetic nephropathy cases in comparison with cases without nephropathy.
- In this study, people aged 41 to 50 made up about 36.0% of the cases with diabetic nephropathy and 44.0% of the cases without it.
- According to this study, women made up roughly 62.0% of cases with diabetic nephropathy and 54.0% of cases without it.
- The average duration of diabetes mellitus was 9.7 years for those without diabetic nephropathy and 10.3 years for those with it.
- Sedentary workers accounted for roughly 66.0% of individuals with diabetic nephropathy and 62.0% of those without it.
- Current smokers made up about 8.0% of individuals without diabetic nephropathy and 14.0% of cases with it.
- Alcohol use was present in about 2.0% of individuals with diabetic nephropathy and 4.0% of cases without it.
- Physical inactivity was present in about 30.0% of cases without diabetic nephropathy and 26.0% of cases with it.
- Insulin was used in about 18.0% of individuals without diabetic nephropathy and 28.0% of those with it.
- The mean height in individuals with diabetic nephropathy was 161 cm, while the mean height in individuals without the condition was 161.7 cm.
- The average weight of individuals with diabetic nephropathy was 66.1 kg, while those without the condition weighed 61.6 kg.
- The mean HbA_1c was 7.5 gms% in diabetic nephropathy cases and 7.7 gms% in non-diabetic nephropathy cases.

- Mean serum creatinine in cases with diabetic nephropathy was 3.44 mg/ dl and 1.23 mg/ dl of cases without diabetic kidney disease.
- About 36.0% of the cases with diabetic nephropathy had stage 2 kidney disease, 48.0% had stage 3 and 16.0% had stage 4 kidney disease in this study.
- Forced expiratory volume 1 was 1.6 litres in cases with diabetic nephropathy and 1.8 litres in cases without diabetic nephropathy.
- Mean functional vital capacity was 3.3 litres in cases with diabetic nephropathy and 3.3 litres in cases without diabetic nephropathy.
- Peak expiratory flow rate in cases with diabetic nephropathy was 213.9 and 201.2 in cases without diabetic nephropathy.
- Mean FEV_1/FVC ratio in cases with diabetic nephropathy was 0.51 and 0.55 in cases without diabetic nephropathy.

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ANNEXURES

PROFORMA

Comparison Of Pulmonary Function Tests in patients with Diabetic Nephropathy and without Nephropathy

NAME: AGE/SEX:

OCCUPATION: ADDRESS :

DURATION OF DIABETES: : DIABETIC RELATED HISTORY

DECREASED VISION TINGLING/NUMBNESS

BLURRING OF VISION GIDDINESS/BLACKOUTS

INCREASED SWEATING INCONTINENCE OF URINE/FAECES

TRANSIENT LOC LOWER URINARY TRACT SYMPTOMS

H/O ACUTE ONSET OF FEVER FAMILY H/O DIABETES

PHYSICAL EXERCISE

TREATMENT HISTORY: 1.OHA 2.INSULIN 3.OHA+INSULIN

GENERAL EXAMINATION: P/Ict/Cy/Cl/LN/PE

HEIGHT: WEIGHT: BMI:

WAIST CIRCUMFERENCE: PULSE:

BLOOD PRESSURE— SUPINE: STANDING:

Cardiovascular system Abdominal system

Respiratory system Central nervous system

INVESTIGATIONS FBS: FUNDOSCOPY:

PPBS: HbA1C:

B.UREA: S.CREATININE:

U.MICROALBUMIN: ECG:

CHEST X-RAY: FUNDOSCOPY :

ULTRASOUND ABDOMEN AND PELVIS :

PULMONARY FUNCTION TESTS

PARAMETERS	MEASURED	PREDICTED
FVC		
FEV_1		
FEV_1		
FEV_1/ FVC		
FEF 25% - 75%		

INFORMED CONSENT FORM

Date:

I, Mr/Mrs ______________________________, have been explained in my own vernacular language that I/We will be included in **"Comparison Of Pulmonary Function Tests in patients with Diabetic Nephropathy and without Nephropathy",** hereby I 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 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

____

(Signature of Patient)

(Signature & Name of Research doctor)

<u>ರೋಗಿಯ ಮಾಹಿತಿ ಪತ್ರಿಕೆ</u>

ಅಧ್ಯಯನ ಶೀರ್ಷಿಕೆ: "ಮಧುಮೇಹ ನೆಫ್ರೋಪತಿ ಮತ್ತು ನೆಫ್ರೋಪತಿ ಇಲ್ಲದ ರೋಗಿಗಳಲ್ಲಿ ಶ್ವಾಸಕೋಶದ ಕಾರ್ಯ ಪರೀಕ್ಷೆಗಳ ಹೋಲಿಕೆ"

ದಿನಾಂಕ:

ನಾನು, ಶ್ರೀ / ಶ್ರೀಮತಿ __________, ಡಯಾಬಿಟಿಕ್ ನೆಫ್ರೋಪತಿ ಮತ್ತು ನೆಫ್ರೋಪತಿ ಇಲ್ಲದ ರೋಗಿಗಳಲ್ಲಿ ಶ್ವಾಸಕೋಶದ ಕಾರ್ಯ ಪರೀಕ್ಷೆಗಳ ಹೋಲಿಕೆ ಅಧ್ಯಯನ ಮಾಡಲು ನಾನು / ನಮ್ಮನ್ನು ಸೇರಿಸಲಾಗುವುದು ಎಂದು ನನ್ನದೇ ಆದ ಸ್ಥಳೀಯ ಭಾಷೆಯಲ್ಲಿ ವಿವರಿಸಲಾಗಿದೆ. ಹೆಮಟೊಲಾಜಿಕಲ್ ಮತ್ತು ಕ್ಲಿನಿಕಲ್ ನಿಯತಾಂಕಗಳ ಅವಲೋಕನಗಳನ್ನು ದಾಖಲಿಸಲು ಯಾವುದೇ ಬಲ ಅಥವಾ ಪೂರ್ವಾಗ್ರಹವಿಲ್ಲದೆ ತಿಳುವಳಿಕೆಯುಳ್ಳ ಒಪ್ಪಿಗೆ. ಒಳಗೊಂಡಿರುವ ಸ್ವರೂಪ ಮತ್ತು ಅಪಾಯಗಳನ್ನು ನನ್ನ ತೃಪ್ತಿಗೆ ವಿವರಿಸಲಾಗಿದೆ ನಡೆಸುತ್ತಿರುವ ಅಧ್ಯಯನದ ಬಗ್ಗೆ ನನಗೆ ವಿವರವಾಗಿ ವಿವರಿಸಲಾಗಿದೆ. ನಾನು ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆಯನ್ನು ಓದಿದ್ದೇನೆ ಮತ್ತು ಯಾವುದೇ ಪ್ರಶ್ನೆ ಕೇಳುವ ಅವಕಾಶ ನನಗೆ ಸಿಕ್ಕಿದೆ. ನಾನು ಕೇಳಿದ ಯಾವುದೇ ಪ್ರಶ್ನೆಗೆ ನನ್ನ ತೃಪ್ತಿಗೆ ಉತ್ತರಿಸಲಾಗಿದೆ. ಈ ಸಂಶೋಧನೆಯಲ್ಲಿ ಪಾಲ್ಗೊಳ್ಳುವವನಾಗಿ ನನ್ನ / ನನ್ನ ಸಂಬಂಧಿಯನ್ನು ಅನುಮತಿಸಲು ನಾನು ಸ್ವಯಂಪ್ರೇರಣೆಯಿಂದ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ. ಇತಿಹಾಸವನ್ನು ಒದಗಿಸಲು, ದೈಹಿಕ ಪರೀಕ್ಷೆಗೆ ಒಳಗಾಗಲು, ಕಾರ್ಯವಿಧಾನಕ್ಕೆ ಒಳಗಾಗಲು, ತನಿಖೆಗೆ ಒಳಗಾಗಲು ಮತ್ತು ಅದರ ಫಲಿತಾಂಶಗಳು ಮತ್ತು ದಾಖಲೆಗಳನ್ನು ಡಾಕ್‌ಗೆ ಒದಗಿಸಲು ನಾನು ಈ ಮೂಲಕ ಒಪ್ಪಿಗೆ ನೀಡುತ್ತೇನೆ

______

ರೋಗಿಯ ಹೆಸರು / ರಕ್ಷಕ

(ರೋಗಿಯೊಂದಿಗಿನ ಸಂಬಂಧ)

______

(ರೋಗಿಯ / ಅಟೆಂಡೆಂಟ್‌ನ ಸಹಿ)

(ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ)

PATIENT INFORMATION SHEET

Study title : "Comparison Of Pulmonary Function Tests in patients with Diabetic Nephropathy and without Nephropathy"

Principal investigator: Dr K HARSHITHA/ DR VIDYASAGAR

I, Dr. K HARSHITHA, Post graduate student in Department of general medicine at Sri Devraj Urs Medical College, will be conducting a study titled **"Comparison Of Pulmonary Function Tests in patients with Diabetic Nephropathy and without Nephropathy"**

This study will be useful for further management of Diabetic Nephropathy and Diabetes as a whole. This study will be done under the guidance of Dr. VIDYASAGAR, Professor, Department of GENERAL MEDICINE.

The investigations that are done for the study are - Pulmonary function Test, FBS, PPBS, HbA1c, Serum electrolytes, Renal function test, Complete Blood Picture, Chest Radiograph, Urine Routine, 24 Hour Urinary Protein, Fundoscopy and Ultrasound Abdomen And Pelvis. All the investigations will be done free of cost, by the principal investigator.

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 - 9550795808

Name and Signature of the Principal Investigator

Date-

Patient Signature

<u>ರೋಗಿಯ ಮಾಹಿತಿ ಹಾಳೆ</u>

ಅಧ್ಯಯನದ ಶೀರ್ಷಿಕೆ: ಡಯಾಬಿಟಿಕ್ ನೆಫ್ರೋಪತಿ ಮತ್ತು ನೆಫ್ರೋಪತಿ ಇಲ್ಲದ ರೋಗಿಗಳಲ್ಲಿ ಶ್ವಾಸಕೋಶದ ಕಾರ್ಯ ಪರೀಕ್ಷೆಗಳ ಹೋಲಿಕೆ

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿ: ಡಾ. ಹರ್ಷಿತಾ ಕೆ/ವಿದ್ಯಾಸಾಗರ್ ಸಿ ಆರ್

ಶ್ರೀ ದೇವರಾಜ್ ಮೆಡಿಕಲ್ ಕಾಲೇಜಿನಲ್ಲಿ ಸಾಮಾನ್ಯ ಔಷಧಿ ವಿಭಾಗದಲ್ಲಿ ಸ್ನಾತಕೋತ್ತರ ವಿದ್ಯಾರ್ಥಿ ಡಾ. ಹರ್ಷಿತಾ ಕೆ/ "ಡಯಾಬಿಟಿಕ್ ನೆಫ್ರೋಪತಿ ಮತ್ತು ನೆಫ್ರೋಪತಿ ಇಲ್ಲದ ರೋಗಿಗಳಲ್ಲಿ ಶ್ವಾಸಕೋಶದ ಕಾರ್ಯ ಪರೀಕ್ಷೆಗಳ ಹೋಲಿಕೆ" ಎಂಬ ಅಧ್ಯಯನವನ್ನು ನಡೆಸಲಿದ್ದೇನೆ. ಈ ಅಧ್ಯಯನವು ಡಯಾಬಿಟಿಕ್ ನೆಫ್ರೋಪತಿ ಮತ್ತು ಒಟ್ಟಾರೆಯಾಗಿ ಮಧುಮೇಹದ ಹೆಚ್ಚಿನ ನಿರ್ವಹಣೆಗೆ ಉಪಯುಕ್ತವಾಗಿದೆ. ಜನರಲ್ ಮೆಡಿಸಿನ್ ವಿಭಾಗದ ಪ್ರಾಧ್ಯಾಪಕ ಡಾ.ವಿದ್ಯಾಸಾಗರ್ ಅವರ ಮಾರ್ಗದರ್ಶನದಲ್ಲಿ ಈ ಅಧ್ಯಯನ ನಡೆಯಲಿದೆ.

ಅಧ್ಯಯನಕ್ಕಾಗಿ ಮಾಡಲಾದ ತನಿಖೆಗಳೆಂದರೆ - ಪಲ್ಮನರಿ ಫಂಕ್ಷನ್ ಟೆಸ್ಟ್, FBS, PPBS, HbA1c, ಸೀರಮ್ ಎಲೆಕ್ಟ್ರೋಲೈಟ್‌ಗಳು, ಮೂತ್ರಪಿಂಡದ ಕಾರ್ಯ ಪರೀಕ್ಷೆ, ಸಂಪೂರ್ಣ ರಕ್ತದ ಚಿತ್ರ, ಎದೆಯ ರೇಡಿಯೋಗ್ರಾಫ್, ಮೂತ್ರದ ದಿನಚರಿ, 24 ಗಂಟೆಗಳ ಮೂತ್ರದ ಪ್ರೋಟೀನ್, ಫಂಡೋಸ್ಕೋಪಿ ಮತ್ತು ಅಲ್ಟ್ರಾಸೌಂಡ್ ಹೊಟ್ಟೆ ಮತ್ತು ಸೊಂಟ. ಎಲ್ಲಾ ತನಿಖೆಗಳನ್ನು ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಗಳು ಉಚಿತವಾಗಿ ಮಾಡುತ್ತಾರೆ.

ಎಲ್ಲಾ ಡೇಟಾವನ್ನು ಗೌಪ್ಯವಾಗಿ ಇರಿಸಲಾಗುತ್ತದೆ ಮತ್ತು ಸಂಸ್ಥೆಯು ನಿರ್ದಿಷ್ಟಪಡಿಸಿದ ಉದ್ದೇಶಕ್ಕಾಗಿ ಮಾತ್ರ ಬಳಸಲಾಗುತ್ತದೆ. ಈ ಅಧ್ಯಯನದಲ್ಲಿ ನಿಮ್ಮ ಭಾಗವಹಿಸುವಿಕೆಗೆ ಒಪ್ಪಿಗೆ ನೀಡಲು ನೀವು ಸ್ವತಂತ್ರರಾಗಿದ್ದೀರಿ. ಯಾವುದೇ ಕಾರಣಗಳನ್ನು ನೀಡದೆ ನೀವು ಯಾವುದೇ ಸಮಯದಲ್ಲಿ ಅಧ್ಯಯನದಿಂದ ಹಿಂದೆ ಸರಿಯಬಹುದು. ಭಾಗವಹಿಸಲು ನಿಮ್ಮ ನಿರಾಕರಣೆಯು ಈ ಸಂಸ್ಥೆಯಲ್ಲಿ ಯಾವುದೇ ಪ್ರಸ್ತುತ ಅಥವಾ ಭವಿಷ್ಯದ ಕಾಳಜಿಗೆ ನಿಮ್ಮನ್ನು ಪೂರ್ವಾಗ್ರಹ ಮಾಡುವುದಿಲ್ಲ.

ಯಾವುದೇ ಸ್ಪಷ್ಟೀಕರಣಗಳ ಅಗತ್ಯವಿದ್ದಲ್ಲಿ ನೀವು ಈ ಮೊಬೈಲ್ ಸಂಖ್ಯೆಗೆ ನನ್ನನ್ನು ಸಂಪರ್ಕಿಸಲು ಮುಕ್ತರಾಗಿದ್ದೀರಿ - 9550795808

ಪ್ರಧಾನ ತನಿಖಾಧಿಕಾರಿಯ ಹೆಸರು ಮತ್ತು ಸಹಿ

ದಿನಾಂಕ-

ರೋಗಿಯ ಅಥವಾ ರೋಗಿಯ ವೀಕ್ಷಕರ ಸಹಿ

MASTER CHART

Sl no	Age	Sex	HbA1c	Serum creatinine	BUN	Stage of kidney disease	PRE FEV1	FVC	PEFR	PRE FEV1/ FVC
1	42	F	7.4	2.5	43	2	2.7	4.2	205	0.64
2	40	F	8	3.1	39	2	2	3.1	216	0.64
3	42	M	6.4	2.9	34	3	2.52	3.8	236	0.67
4	46	M	8	2.4	26	4	1.9	2.9	138	0.66
5	47	F	8.5	2.5	52	3	2.8	4.9	252	0.57
6	67	M	8.9	2.6	96	2	2	3.4	234	0.58
7	52	M	6.5	2.8	104	3	2.1	3.5	178	0.6
8	49	M	6.8	2.4	45	2	2.2	3.9	150	0.57
9	65	F	7.2	3.8	55.7	3	1.6	2.8	255	0.57
10	65	F	8.8	2.6	39	4	1.4	3.3	169	0.42
11	72	F	8.4	2.9	30	3	2.4	4.6	208	0.52
12	56	M	7.2	3.4	26	2	2	3.5	260	0.57
13	47	F	7.1	3.1	18	3	1.6	3.0	224	0.53
14	54	F	6.6	2.5	26	4	1.4	3.1	155	0.45
15	66	F	6.5	2.4	20	3	1.2	3.2	199	0.38
16	56	F	6.4	2.6	28	2	0.8	2.1	232	0.38
17	44	F	8.4	1.8	32	3	1.45	5.8	246	0.25
18	46	F	7.1	1.9	30	2	1.3	2.7	188	0.48
19	58	F	7.2	2.6	30	3	1.9	3.3	288	0.57
20	63	M	6.6	1.8	20	4	2	3.5	268	0.57
21	59	M	6.8	1.6	116	3	1.3	2.0	222	0.65
22	40	M	8.8	2.1	22	2	2	3.2	199	0.62
23	54	M	6.9	1.2	96	3	2.2	3.4	176	0.64
24	53	F	6.7	1.3	52	2	2.4	3.5	201	0.68
25	56	F	6.4	1.5	96	3	2.5	3.7	266	0.67
26	55	F	8.8	1.8	104	4	2.6	3.8	169	0.68
27	58	M	9.4	1.6	45	3	1.5	2.4	152	0.63
28	52	M	6.6	1.2	39	2	1.8	3.0	155	0.6
29	44	F	6.4	1.8	43	3	2.3	3.7	226	0.63
30	57	F	7.8	2.6	20	4	0.7	3.0	243	0.23
31	45	M	8.2	2.4	42	3	1.2	3.2	254	0.38
32	42	F	11.9	3.2	30	2	1.4	2.1	186	0.68
33	40	F	7.9	1.5	39	2	1.9	3.0	216	0.63
34	42	M	7.3	1.8	32	3	2.2	3.3	260	0.66

Sl no	Age	Sex	HbA1c	Serum creatinine	BUN	Stage of kidney disease	PRE FEV1	FVC	PEFR	PRE FEV1/ FVC
35	46	M	8.5	2.5	52	3	2	3.4	286	0.58
36	47	F	8.9	2.6	96	2	1	3.6	272	0.28
37	67	M	6.5	2.8	104	3	1.5	2.2	168	0.67
38	52	M	6.8	2.4	45	2	2.1	4.6	286	0.46
39	49	M	7.2	3.8	55.7	3	1.4	3.4	224	0.41
40	65	F	8.8	2.6	39	4	1.6	3.6	262	0.45
41	65	F	8.4	2.9	30	3	2.6	3.8	152	0.68
42	72	F	7.2	3.4	26	2	1.3	2.1	144	0.62
43	56	M	7.1	3.1	18	3	1.8	2.6	132	0.68
44	47	F	6.6	2.5	26	4	2.8	4.2	306	0.66
45	54	F	6.5	2.4	20	3	1.2	3.2	171	0.38
46	66	F	6.4	2.6	28	2	1.1	3.2	181	0.34
47	56	F	8.5	2.5	52	3	1.4	2.1	197	0.67
48	44	F	8.9	2.6	96	2	2	3.5	251	0.57
49	46	F	6.5	2.8	104	3	2.4	4.1	253	0.58
50	58	F	6.8	2.4	45	2	0.8	3.1	184	0.26
51	63	M	7.2	1	39	0	2.3	3.7	239	0.63
52	59	M	8.8	1.2	17	0	2	3.4	157	0.58
53	40	M	8.4	1.5	30	0	2.5	4.4	195	0.57
54	54	M	7.2	1.3	43	0	1.6	2.6	186	0.62
55	53	F	7.1	1.4	39	0	2.4	3.8	155	0.64
56	56	F	6.6	1.2	43	0	2	3.6	147	0.55
57	55	F	6.5	0.9	39	0	1.1	2.0	325	0.55
58	58	M	6.4	0.8	34	0	2.1	3.4	141	0.61
59	52	M	6.5	1.1	26	0	1.4	3.0	211	0.46
60	44	F	6.8	1.6	24	0	1.1	3.1	203	0.35
61	57	F	7.2	1.7	32	0	1	3.2	243	0.31
62	45	M	8.8	1.5	26	0	0.6	0.9	147	0.64
63	59	F	8.4	1.2	25	0	2.2	11.0	135	0.2
64	40	F	7.2	1.1	39	0	1.4	3.1	201	0.45
65	48	F	7.1	1	32	0	1.6	3.2	171	0.5
66	42	F	6.5	1.6	39	0	1	3.3	186	0.3
67	45	F	7.2	1.2	26	0	0.6	1.3	227	0.456

Sl no	Age	Sex	HbA1c	Serum creatinine	BUN	Stage of kidney disease	PRE FEV1	FVC	PEFR	PRE FEV1/ FVC
68	48	F	8	1.2	25	0	2.3	9.2	275	0.25
69	53	F	7.2	2.1	39	0	2	3.1	157	0.64
70	45	F	8	2.3	32	0	2.3	3.3	157	0.69
71	54	F	8.3	1.2	39	0	1.5	3.4	189	0.44
72	50	M	8.3	1.5	22	0	1	3.4	196	0.29
73	55	M	6.7	1.6	15	0	2.1	3.5	151	0.6
74	48	M	7.9	2.5	30	0	1.92	3.1	273	0.62
75	40	M	10	1.7	20	0	1.13	1.9	188	0.61
76	45	F	7.2	2.6	32	0	2.2	6.9	131	0.32
77	45	F	6.5	1.6	22	0	1.28	3.8	212	0.34
78	58	F	7.3	1.5	26	0	2.11	3.5	151	0.6
79	58	M	7.5	1.2	96	0	1.3	2.0	218	0.66
80	40	M	6.8	1.6	32	0	2.38	3.5	254	0.68
81	40	F	9.5	1.8	17	0	1.78	3.6	131	0.49
82	44	F	7.5	1.1	30	0	1.92	3.3	265	0.59
83	62	M	8.3	1.3	43	0	1.32	2.7	274	0.49
84	57	F	13.1	1.2	39	0	1.72	2.7	239	0.64
85	49	F	6.5	2.6	17	0	1.45	2.7	209	0.53
86	44	F	10.8	0.6	30	0	1.58	2.8	252	0.56
87	48	F	8	1	43	0	1.57	4.1	224	0.38
88	52	M	7.6	1	39	0	0.61	2.5	165	0.24
89	42	M	7.3	0.6	43	0	0.95	1.5	172	0.62
90	64	F	7.1	1.4	39	0	1.75	2.7	200	0.66
91	51	M	7.8	0.2	34	0	1.47	2.2	205	0.66
92	48	M	6.7	0.5	26	0	1.8	3.1	222	0.59
93	40	F	7.8	0.8	24	0	1.65	2.7	321	0.61
94	40	M	7.4	0.2	32	0	1.18	2.6	260	0.46
95	49	F	7.9	0.8	26	0	1	2.6	192	0.39
96	42	M	7.3	1	25	0	1.21	2.5	213	0.49
97	43	M	8.2	0.5	39	0	1.68	2.6	178	0.64
98	47	M	7.9	0.6	32	0	1.12	2.6	209	0.43
99	40	M	7.3	0.6	39	0	1.56	4.2	144	0.37
100	45	F	6.8	0.2	22	0	1.1	2.0	164	0.56