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NAIBA EMMANUEL

SU91-MSAHW-S23-070

FAHS

**Sonographic Markers in the Early Detection of Renal
Dysfunction in Diabetic Adults**



SUPERIOR UNIVERSITY

Thesis Submitted to

The Superior University Lahore

In Partial Fulfillment of the

Requirement for the Degree

Master of Science in Allied Health Sciences

By

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Roll No. SU91-MSAHW-S23-070

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Supervisor Name: Sadia Sabir

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This is to certify that the research work presented in this thesis, titled **“sonographic markers in the early detection of renal dysfunction in diabetic adults”** was conducted by **“Naiba Emmanuel”** under the supervision of **“Miss. Sadia Sabir”**

No part of this thesis has been submitted anywhere else for any other degree. This thesis is submitted to the Faculty of Allied Health Sciences, The Superior University, Lahore in partial fulfillment of the requirements for the degree of **MS Diagnostic Ultrasound** in the field of **“Radiological Imaging Technology”** in Faculty of Allied Health Sciences at The Superior University, Lahore.

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DEDICATION

In the name of GOD, the most Gracious, the most Merciful.

I am extremely thankful to Almighty 'GOD' Who is the entire source of knowledge and wisdom endowed to mankind, for providing me with the acumen and vision to complete this endeavor.

I would like to express my profound gratitude to my supervisor for her wise counsel and encouraging attitude towards this study. I am extremely grateful to her for immensely facilitating me during my study period by ensuring the provision of favorable circumstances and conducive environment. This project would not have been possible without her support and expert guidance.

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Student Name

NAIBA EMMNAUEL

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LIST OF ABBREVIATION

Abbreviation not explain in the text

SCr	Serum Creatinine
GFR	Glomerular Filtration Rate
BMI	Body Mass Index
CKD	Chronic Kidney Disease
ESRD	end-stage renal diseases
DM	Diabetes Mellitus
US	Ultrasound
MRI	Magnetic Resonance Imaging

ABSTRACT

Determining kidney function in adult diabetic populations is essential for nephrology and medical interventions. A key indicator of the health and function of the kidneys is its size; variations from normal values, which usually signify the existence of underlying renal illnesses such as renal tumors, polycystic kidney disease, or kidney cysts. Since renal dysfunction usually develops over several years and has an extended latent phase during which the disorder is clinically quiet, biomarkers that measure kidney function are the primary basis for diagnosis, evaluation, and treatment. The current study comprised a sample of 250 persons. The prevalence of kidney size anomalies, such as variations in parenchymal thickness and cortical echogenicity, in connection to gender and BMI is also examined in this study. The results showed that male participants had higher serum creatinine and eGFR levels with increasing BMI. Serum creatinine ranged from 0.876 ± 0.341 mg/dL in males with BMI <18.5 kg/m² to 1.325 ± 0.677 mg/dL in those with BMI >30 kg/m², with an overall mean of 0.997 ± 0.503 mg/dL ($p=0.113$). eGFR increased from 77.257 ± 11.959 mL/min/1.73 m² to 101.125 ± 16.762 mL/min/1.73 m², with a significant p-value of 0.001. In females, serum creatinine ranged from 0.790 ± 0.310 mg/dL in the <18.5 BMI group to 1.097 ± 1.004 mg/dL in the >30 BMI group, with a mean of 0.963 ± 0.798 mg/dL ($p=0.014$). eGFR ranged from 62.200 ± 10.464 mL/min/1.73 m² to 84.595 ± 13.180 mL/min/1.73 m², with a significant p-value of <0.001 .

Key Words: kidney dysfunction, ultrasonography, sonographic markers, diabetes

Chapter 1:

INTRODUCTION

One of the body's two most important excretory organs, the kidneys are essential for preserving electrolytes and water balance. They also have endocrine organ functions. Renal size evaluation is important since kidney enlargement as well as decrease is a symptom of many diseases. Although anatomy suggest that the diameter lengthwise and according to width of a person's kidney are 12 centimetres and 6 centimetres, respectively, research indicates that these measurements differ between ethnic communities and are impacted by other variables like body size, gender, and age (1).

In nephrology and medical care, it is crucial to estimate kidney function among adult diabetes populations. Kidney size is a crucial metric for evaluating the well-being and functioning of the kidneys; deviations from normal levels frequently indicate the presence of underlying renal diseases including renal tumours, polycystic kidney disorder, or the kidneys cysts.

According to the International Diabetes Federation (IDF), there are about 537 million people worldwide having diabetes mellitus (DM) during 2021 and by 2045, the figure is predicted to increase nearly 783 million. Diabetes Mellitus is one among the most prevalent chronic illnesses in the world. Diabetic nephropathy, one of the many consequences linked to diabetes, is a major contributor to the development of end-stage renal diseases (ESRD) as well as chronic kidney disease (CKD), which together account for a substantial worldwide healthcare burden. Prevention of permanent kidney damage through early detection and prompt treatments for diabetic nephropathy will greatly lower mortality as well as morbidity risks in diabetes patients (2, 3).

Accurate estimation of kidney function is central to the detection, evaluation, and treatment. Glomerular filtration rate (GFR) is widely accepted as the best overall measure of kidney function. For this reason, Chronic Kidney Disease is defined based on the level of Glomerular Filtration Rate, and clinical recommendations for individuals at increased risk of renal dysfunction and for patients at each stage of Chronic Kidney Disease include assessment of Glomerular filtration rate. A normal urine albumin-to-creatinine ratio (uACR) is typically less than 30 milligrams per gram (mg/g). (4).

Renal dysfunction typically evolves over many years, with a long latent period when the disease is clinically silent and therefore diagnosis, evaluation and treatment is based mainly on biomarkers that assess kidney function. Glomerular filtration rate (GFR) remains the ideal marker of kidney function. Unfortunately measuring GFR is time consuming and therefore GFR is usually estimated from equations that take into account endogenous filtration markers like serum creatinine (SCr) (5).

It is estimated that 850 million individuals worldwide, or 9.5% from the population, suffer from chronic kidney diseases (CKD). The prevalence of Chronic Kidney Disease is expected to increase further in light of ageing populations along with the rising incidence of multimorbidity, even among younger individuals. It is anticipated that Chronic Kidney Disease would rank as the fifth most common cause of mortality worldwide by 2040. Dialysis alone accounts for more than half of the money spent on CKD, despite renal failure being a rare side effect of the disease. About 40,000 early demises in the United Kingdom are caused by Chronic Kidney Disease, and those who have the disease have significantly higher percentages of hospitalisation and hospital stays for prolonged periods of time. The direct as well as indirect effects of Chronic Kidney Disease cause disability and decreased productivity, which reduces the working population and raises sick pay. Although not every instance of renal failure or chronic renal disease may be avoided, but risk reduction techniques can be both economical and successful if Chronic Kidney Disease is identified and treated promptly in the course of the illness. Only 25% of nations have Chronic Kidney Disease management plans in place, and less than half acknowledge Chronic Kidney Disease as a global health concern. The significant cost of this condition and the general lack of knowledge about ways to treat individuals with chronic kidney disease further hinder appropriate and prompt care (6).

Numerous imaging techniques are employed to estimate renal size. Although magnetic resonance imaging (MRI) and computed tomography (CT) are well-established methods for measuring kidney volume, they have disadvantages such as expense, radiation exposure, and restricted accessibility. With minimal inter-observer variability, the ultrasound of the kidneys may be performed effectively, swiftly, economically, widely, and right on the bedside of the patients to give the physician vital kidney anatomical data. The safety of

utilizing ultrasonography for diagnostics indications is widely known. Without exposing the person being examined to radiations, the use of ultrasound is non-invasive exploratory method that gives sufficient structural characteristics to diagnose disorders of the kidney (7).

Bipolar renal diameters are nearly always recorded at renal ultrasound because they are correlated with renal function in chronic kidney disease (CKD). According to earlier research, renal volume determined by ultrasonography provides a more precise indication of a functional kidney compared to its length. Although renal length has relatively little specificity for predicting impaired renal function, a more recent study found that the kidneys length as well as volume significantly connected with elderly's approximate glomerular filtration rate (eGFR) (8). It is challenging to determine the actual kidney volume with ultrasound, though. Although the ellipsoid technique can be utilised for determining volume, this approach has a flaw in that the kidney is not ellipsoid. Furthermore, the central sinus fat, which varies from participant person to person and doesn't have functional renal tissue, would be included alongside the ellipsoid volume. These elements most likely influenced the results of a recent study that found a weak but favourable correlation between several glomerular filtration rate (GFR) indices and kidney volume as measured by sonography (9).

Overview Nephron loss and irreversible sclerosis are hallmarks of chronic kidney disease (CKD). According to the underlying cause, the renal mass gradually decreases over an extended period of time. Patients with CKD are divided into five phases. Changes in the kidney's markers in bloodstream and urine, histopathological anomalies, and imaging tests will all be used to diagnose chronic kidney disease (CKD). Ultrasound is the best imaging method because it is non-invasive, makes the kidney easily accessible and visible, and provides enough information on its anatomy without exposing the patient to radiation or contrast. All of these elements aid in the preliminary diagnosis, kidney function anticipating, and therapeutic decision-making. The best way to evaluate kidney disease is by ultrasonography, which is the only tool available to most those with chronic kidney disease. (10) . Irreversible kidney injury is indicated by a small kidney with a thin echogenic cortex or parenchyma. Alteration in renal echogenicity are represented by

ultrasonographic findings such as longitudinal length, parenchymal, and cortical thickness. Additionally, ultrasonography is a superior method for determining how the disease is progressing. It offers details on the degree of kidney impairment, its potential for recovery, and the choice to have a renal biopsy. A straightforward and widely used technique for estimating GFR is serum creatinine. However, tubular creatinine production and individual differences in the serum creatinine depending on muscle mass are disadvantages of serum creatinine-based GFR. In addition, any notable increase in serum creatinine indicates a roughly 50% decline in GFR (11).

Chronic kidney diseases also known as CKD, is defined as a rise in creatinine levels over several months. Reduced glomerular filtration rate (GFR) (less than 60 ml/min per 1.7 m²) for more than three months is used to determine the degree of kidney injury. CKD is one of the major causes of kidney failure which gives rise to end stage renal disease (ESRD). It entails a gradual decline within the kidney's functioning and physical structure over several months, either with drop or without any drop in glomerular filtration rate (GFR). Pathological irregularities, fluctuations in both urine and blood content of renal function indicators (Glomerular Filtration Rate, serum creatinine), or imaging tests can all be used to detect chronic renal disease (12).

Given their correlation with general body shape and structure, anthropometric measures including heights and weight of the body are frequently suggested as possible predictors of kidney volume. Numerous studies have examined the connection between kidney volume and anthropometric data, indicating that given parameters could be helpful stand-ins for estimating renal size. This has been demonstrated that kidney volume is more precise and is associated better with the overall anthropometry of the subjects (BMI, height and weight). Gender, height, weight, any signs of renal illness, and the nutritional health of the individual are all known to have an impact on the kidney size or diameters of children. One commonly cited indicator of a person's nutritional condition is their body mass index (BMI). Individuals can be classified as underweight using calculated BMI values (13).

For persons who have diabetes or is at an elevated risk for developing chronic kidney disease, this research will provide the most recent recommendations. Individuals in

economically challenged or low-resource areas have greater access to promptly kidney problem identification attributed to the cost-effectiveness of ultrasound-based examinations which improves treatment equity. A continuous assessment concerning the renal' functioning and structure could be obtained by sonographic markers, that are helpful to track the course of the condition over time. Because medical professionals can modify drugs or therapies determined by the findings of routine ultrasound evaluation, diabetic people may benefit from more individualised treatment, which can help delay or stop the further development of the illness. Enhanced quality of living, fewer hospital stays, and reduced general healthcare expenses are all results of fewer problems. Patients can enjoy healthier, more active lifestyles because they are less burdened by maintaining advanced illness stages. Instead of depending on late-stage indicators, which frequently indicate irreparable damage, individuals with diabetes mellitus may gain from earlier management approaches that concentrate on preventing renal damage (14).

Sonographic markers tend to be more specific on the initial phases of renal damage because diabetic adults having early renal failure show greater renal cortical echogenicity along with abnormal kidney size upon sonography, that can be detected before significant rises in serum creatinine as well reductions in Glomerular Filtration Rate. Compared to conventional biomarkers, having evidence of aberrant sonographic indicators at baseline (such as increased echogenicity) more effectively predicts prospective progression to CKD, or chronic kidneys disease, in persons with diabetes.

There is no significant difference between sonographic markers and traditional biochemical markers (e.g., serum creatinine, GFR) in the early detection of renal dysfunction in diabetic adults. This hypothesis sets the foundation for research aimed at proving the effectiveness and importance of sonographic markers in the early, non-invasive detection of renal dysfunction among diabetic patients, with the goal of improving early diagnosis and intervention strategies.

AIMS AND OBJECTIVES

- To evaluate the sensitivity and specificity of sonographic markers with traditional biochemical markers in diabetic adults.
- To identifying early renal impairment across different BMI categories and between gender.

Chapter 2

LITERATURE REVIEW

The kidneys are situated in retroperitoneal region on both sides of the spine. Since the liver is located above the right kidney in the abdominal region, the left kidney is slightly more elevated than right kidney (15). Males and females weigh between 125 to 175 grammes and 115 to 155 grams, respectively, for both bean-shaped organs. On average, the kidney is between 11 and 14 centimetres long, 6 centimetres wide, and 4 centimetres thick. The muscle mass, fat, and rear ribs surround the kidneys (16).

The kidneys are shielded and kept in form by the renal capsule, a thin, robust, fibrous external layer. The perirenal fat surrounds this, serving as a cushion of protection and helping to maintain the kidney's structural integrity. The ureters, lymph nodes, vascular system, and nerves all pass through the kidney through the hilum, an oval in shape notched area on the inner side of organs. Furthermore, the kidneys are attached to the abdominal walls and other surrounding structures by the kidneys fascia, an extra layer of elastic connective tissue (17).

Nephrons, the structural components in charge of filtration, are found in cortex of kidneys, the organ's outermost surface. The kidneys medulla, which is composed of twelve cone-like renal pyramids and thirteen the kidneys columns, is located underneath the cortex. The pyramidal part of medulla is where the cortex expands. These pyramids represent the organisation of the the kidneys the medulla featuring the base of stacked structure facing its outer layer and the pointed peak, called the renal papilla, towards kidney pelvis. Urine is transported from the nephrons to the renal pelvis by collecting ducts located in the pyramids of the renal. Urine is collected in the renal pelvis, a funnel-shaped hollow, before it enters the ureter. From there, it branches into major calyces, which in turn split into minor calyces.

The renal arteries provides oxygenated blood to the kidneys from the abdominal aorta, branching into segmental arteries. Within the renal columns, these segmental arteries split into interlobar arteries, which pass between the renal pyramids. The interlobar arteries give rise to arcuate arteries, which branch into interlobular arteries after arching over the pyramid bases. The glomeruli, the network of capillaries in charge of filtration, are fed by

the afferent arterioles that emerge from these interlobular arteries, which also supply the cortex. Lastly, the renal vein transports the kidneys' deoxygenated blood to the inferior vena cava (18).

Urine moves from nephrons towards kidneys through collecting ducts that exist in renal pyramidal systems, which are cone-shaped projections. Each pyramid's top culminates at the kidney's papilla, producing the little calyx and drains into the greater calyx, whilst the base is positioned on its kidneys cortex. These primary calyces enter the urine tract after joining kidney's pelvis. The renal crest is formed by the union of all renal papillae. The kidneys columns, that are inward projections of the renal cortex, differentiate the pyramidal components (19)

The nephron is the structural and functional unit of the kidney, with each kidney containing approximately 1 to 1.5 million nephrons. It plays a crucial role in filtering blood, reabsorbing essential substances, secreting waste, and forming urine. The nephron consists of two major components: the renal corpuscle and the renal tubule. The renal corpuscle is located in the renal cortex and includes the glomerulus and Bowman's capsule. The glomerulus is a ball-like network of capillaries where blood filtration occurs, with blood entering through the afferent arteriole and exiting via the efferent arteriole. Surrounding the glomerulus is the Bowman's capsule, a double-walled, cup-like structure that collects the filtrate (20).

Three essential layers make up the glomerular filtration barrier, which controls the filtration process. Fenestrations (pores) in the glomerular capillary endothelium permit the flow of water, ions, and tiny solutes while obstructing the passage of big proteins and blood cells. Large proteins cannot pass through the negatively charged matrix that makes up the basement membrane. Specialised epithelial cells termed podocytes, which have pedicels that resemble feet, create slit diaphragms that further regulate the filtering of big molecules and proteins. The primary filtrate (ultrafiltrate) that penetrates the renal tubule is formed by the renal corpuscle's ability to filter about 120 mL of plasma each minute thanks to these layers working together (21).

The renal tubes, a continuous, specialised tube that emerges from Bowman's capsule, is crucial for the excretion of waste materials and for the specific reabsorption of water and other solutes. There are four major parts to it. Simplified cubic epithelial cells having a brushed borders of microvilli to enhance surface area for absorption line the Proximal Convoluted Tubule (PCT), which is situated in the kidney's cortex. The PCT secretes hydrogen ions, ammonia, and certain medications into filtrate whilst reabsorbing 65–70% of the filtered water, salt, chloride, amino acids, , glucose, and potassium.

There are two components to the Loop of Henle, which stretches from the cortex through the medulla thus back: the ascending limb, which has thick walls, is impermeable to water, and actively pumps out potassium, sodium, and chloride, and the descending limb, which has thin walls and is permeable to water but not solutes. Reabsorbing salt, chloride, and potassium causes the ascending leg to dilute the filtrate, whereas the descending limb promotes water uptake and concentrates the filtrate. The medulla concentration slope produced by this opposite mechanism is necessary for the collecting duct's water resorption. The cortex contains the Distal Convoluted Tubule (DCT), which has a simple cuboidal epithelium lining it but no brush boundary. Under the influence of hormones like aldosterone and parathyroid hormone (PTH), it contributes to the reabsorption of calcium, sodium, and chloride. It also secretes potassium, hydrogen ions, and ammonium into the filtrate. Last but not least, the Collecting Duct, which absorbs filtrate from several nephrons and runs from the cortex to the medulla, is not actually a part of the nephron. It is essential for the reabsorption of water when antidiuretic hormone (ADH) is present (22)

Filtration, reabsorption, and secretion all depend on the nephron's blood supply. Blood is delivered by an afferent artery to the glomerulus for filtration. Because it is narrower than the afferent arteriole, the efferent arteriole creates pressure for filtration while draining blood from the glomerulus. Capillaries that are peritubular enables the reabsorption and secretion of solutes by encircling the PCT and DCT. Maintaining the medullary concentration gradient depends on the Vasa Recta, a capillary network that encircles the Henle loop (22).

Diabetes

Diabetes is a long-term medical condition marked by high blood glucose levels and abnormal fatty tissue and protein digestion. When the pancreas is unable to create enough insulin and the cells are unable to benefit from the insulin which is produced efficiently, blood glucose levels rise since the glucose is unable to be metabolized in the cells. Type of Diabetes: Diabetes comes in three main forms: Type 1 Diabetes: Type 1 diabetes is an autoimmune disorder where the beta cells present in the pancreas are unable to produce enough insulin needed to convert glucose in blood to energy, which is required by the cells. As a result, there is an increase in blood sugar levels. This can cause a variety of medical issues, including hypoglycaemia (which is a condition characterized by low sugar levels in blood) as well as hyperglycaemia (which is the inverse condition to hypoglycaemia). Type II Diabetes: where the body cells develop resistance to the activity of insulin generated thus insulin production gradually declines over time. Pregnancy-related Diabetes/Gestational Diabetes: which develops during gestation and raises the probability of type II diabetes in expectant mother and weight gain in the fetus. It can also cause difficulties during gestation and during child birth. The difference between typical and diabetes blood sugar levels is not always inevitable, but there are two more types of glucose intolerance: impaired fasting glucose (IFG) and impaired fasting glycemia (IGT). Compared to those with typical blood sugar levels, those with IFG as well as IGT are more likely to develop cardiovascular disease (23).

Amorim RG et al, in 2009 explored The primary cause of both macrovascular and microvascular problems linked to diabetes is prolonged hyperglycemia. Excess hyperglycemia is a major contributor to the pathophysiology of diabetic kidney diseases, the world's largest cause of dialysis, by causing redox imbalance and systemic and intrarenal inflammation. The onset, progression, and the clinical results of these diseases are influenced by numerous factors and mechanisms, making its etiology complicated multifaceted, and still poorly understood. The metabolic processes involving oxidative and inflammatory pathways, which are generally acknowledged, despite the fact that the mechanisms causing kidney damage associated with diabetes mellitus are diverse. There is unmistakable proof that a persistently high blood sugar level causes inflammation with oxidative stress, which are mediated through modified metabolic

pathways via a self-reinforcing cycle. This promotes the development of end-stage renal disease and cell damage. In the pathophysiology of diabetic kidney disease, the current work provides an update about metabolic pathways including redox imbalances and inflammation brought on by prolonged exposition to hyperglycemia (24).

Thongnak L et al, 2020 conducted a research on the development of diabetic renal disease is a complicated process that involves both lipotoxicity from fat buildup and glucotoxicity. Both of the renal tubular and glomerular parts of the kidneys, considered to be the main locations of diabetic nephropathy lesions, exhibit lipid deposits in diabetic individuals. This review's objective was to shed light on the processes that result in renal lipid accumulation along with the consequences of kidney lipotoxicity in people with diabetes. Diabetic kidneys additionally possess a lower number of lipid oxidation genes and a higher number of lipogenic genes, both of which contribute to lipid buildup. Lipid deposits and lipid metabolites are known to induce oxidative stress, inflammatory conditions, fibrosis, as well as apoptosis. A pathogenic process that can result in diabetes-related nephropathy and impaired kidney function is the kidneys lipotoxicity, which is caused by abnormalities in the metabolism of lipids (25).

Chen C-J et al, in 2020 conducted a study for patients with susceptible chronic kidneys disease (CKD), non-invasive ultrasound imaging methods offer an additional clinical strategy for the early detection of Chronic Kidney Disease. For the identification of various stages of CKD, researchers have developed a detection system based on visual computational methods and automated learning methodologies. For successful and efficient studies, fundamental area-proportional and topographical features, as well as support-vector-machine approaches, were used. Based on approximated glomerular filtering rates, a number of clustered populations of patients with chronic kidney disease were assessed and contrasted. According to the results of changing alterations to ultrasound pictures, the suggested method may be applied as supplementary evidence to assist in distinguishing between various clinical conditions (26).

Gooding KM et al, in 2020 evaluated the primary causes of diabetes-related early mortality is still diabetic kidneys disease (DKD). In addition to frequently accessible

statistics, there is a lack of availability for indicators that can be employed in healthcare settings to enhance renal decline estimation, especially in the initial phases. The BEAt-DKD endeavor's iBEAt research seeks to ascertain if renal imagery biomarkers, such as ultrasound (US) and MRI (magnetic resonance imaging), offer information about the pathophysiology and complexity of Diabetic Kidney Disease and because they might have the capacity to be predictive biomarkers in Diabetic Kidney Disease. More than 500 individuals with type II diabetes mellitus and Glomerular Filtration Rate of ≥ 30 ml/min/1.73 m² are being recruited for the prospective multi-center observational cohort trial known as iBEAt. Blood as well as urine will be drawn, clinical exams will be conducted, and health information will be acquired at baseline. For three years, these evaluations will be conducted once a year. Quantitative kidney Magnetic Resonance Imaging and Ultrasound with automated processing of Magnetic Resonance Imaging will also be performed on every subject at baseline. For diagnostics and validation research, the biological specimens were kept in the central laboratory, while data was kept in the central repository. The possible relationships between scanning biomarkers and kidney disease as well as if imaging biomarkers enhance predictive accuracy of Diabetic Kidney Disease progression were investigated by data analysis. The findings might support a more individualised strategy for managing DKD in Type II DM patients (27). Koc AS et al, in 2019 researched the objectives were which to ascertain if patients with diabetic kidney disease (DKD) had higher cortical stiffness (CS) levels as a result of advanced disease stages and to identify the parameters linked to CS values in the same patient population. The study comprised 22 healthy participants and 120 individuals who had Type-II DM who experienced DKD. CS levels were assessed using pSWE with addition to standard laboratory testing and renal ultrasonography (USG). Aortic and carotid intima-media thickness (IMT) measurements were made. A control group had been then included. In every stage, renal Cortical Stiffness values were discovered to be noticeably higher. AUC had been >70% with parathyroid gland hormone (PTH), cortical thickness, and cortical stiffness (CS) when the curve of receiver operating characteristics analysis was used to identify individuals with DKD. The likelihood of elevated CS (>9.0 kPa) in Diabetes Kidney Disease patients was evaluated separately by thickness of cortex and Parathyroid levels, which were also found to be independently linked with kidney

Cortical Stiffness in kidney disease patients ($P < 0.05$). Reduced cortical thickness while PTH levels are closely linked to a surge in kidney CS as kidney disease stage increases (28).

Variable	AUROC Curve	P	Cut-off	Sensitivity	Specificity
Parathyroid hormone (pg/mL)	0.914 (0.866–0.962)	<0.001	100	86.7%	84.8%
Internal carotid IMT (mm)	0.742 (0.657–0.827)	<0.001	0.6	72.2%	66.7%
NT-proBNP (pg/mL)	0.740 (0.658–0.823)	<0.001	300	63.0%	61.5%
Common carotid IMT (mm)	0.730 (0.643–0.817)	<0.001	0.7	71.7%	65.6%
Cortical thickness (mm)	−0.723 (0.622–0.814)	<0.001	11.5	65.3%	71.4%
Cortical stiffness (kPa)	0.708 (0.617–0.800)	<0.001	8.5	65.2%	60.4%

IMT: Carotid intima-media thickness.

Table 3. The parameters associated with renal cortical stiffness and linear regression analysis for parameters significantly correlated with renal cortical stiffness.

Variable	Univariate analysis		Multivariate analysis	
	P	r	P	B
DM duration (years)	0.002	0.276	0.248	0.119
Creatinine (mg/dL)	0.008	0.239	0.615	0.035
eGFR (mL/min/1.73 m ²)	0.003	−0.265	0.636	−0.055
Parathyroid hormone (pg/mL)	<0.001	0.378	<0.001	0.344
Cortical thickness (mm)	0.011	−0.237	0.011	−0.237
Common carotid IMT (mm)	0.033	0.195	0.127	0.120

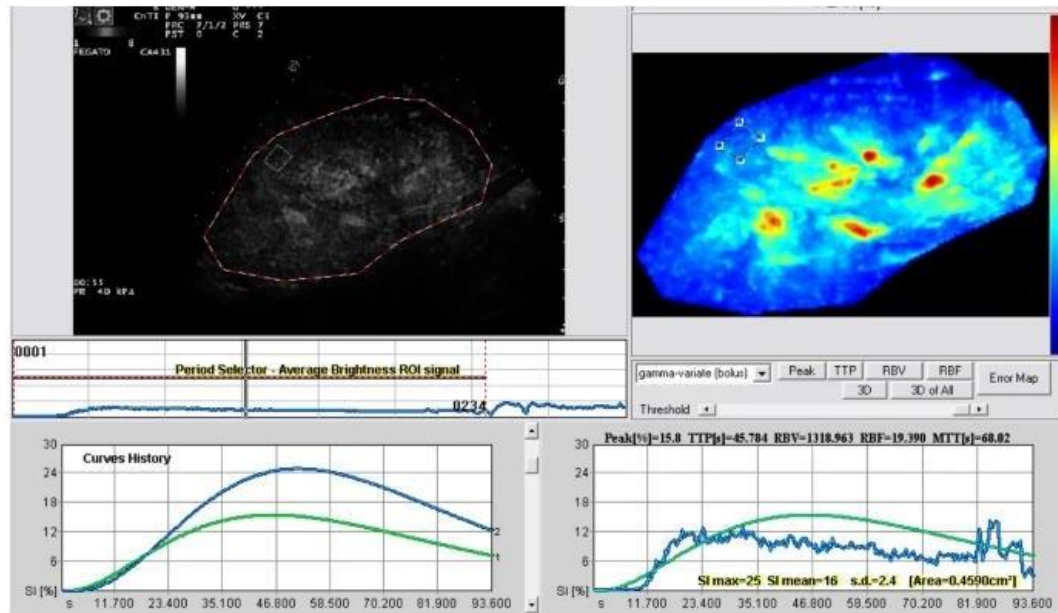
DM: Diabetes mellitus, IMT: Intima-media thickness, $*R^2_{Adjusted} = 0.353$ and control group was excluded in correlation and linear regression analyses.

Figure 2. 1 Renal cortical stiffness parameters

Moloney A et al, in 2020 recruited 86 females with CKD who were followed up with on a secondary referral centre between 2016 to 2019 were included in this retrospective cohort research. Every woman had sonographic placental evaluation in the second trimester, which included uterine arterial Doppler, placental structure, and the umbilicus evaluation. Multiplies on medians (MoM) had been employed to represent continual placenta markers. Sonographic indicators' predictive significance for preeclampsia along with birthweight below the 10th percentile. A specific category of females with chronic kidney

disease (CKD) who are especially at risk for preeclampsia and foetal growth limitation can be identified by a second-trimester sonographic placental scan. Such information could help them with their later prenatal care and help medical professionals make the sometimes difficult distinction between a preeclampsia exacerbation and underlying chronic kidney disease (29).

Druid FM et al, in 2020 found that GFR gradually decreases at a mean average of 8 mL/min/1.73 m²/decade beyond a certain age of thirty. An issue with ageing is that renal function cannot be accurately assessed by measures that are valid for young persons, such as creatinemia. In actuality, even though their renal function has significantly declined, many patients with chronic renal failure may still have serum creatinine values within the normal range. Since renal size as well as parenchymal echogenicity are seen as indicators of kidney function, ultrasonography has become a standard investigative technique in kidney disorders. As such, US is helpful in determining the existence and severity of renal failure. When assessing kidney illness in older people, The CEUS procedure is helpful because the elevated haemodynamic resistance of kidneys microvessels lowers as assessing senile changes, EcoColor along with EcoDoppler also prove helpful. In this case, the distribution of colour data is more reduced and restricted to intersegmental as well as interlobar districts in contrast to the younger adult populations. The resistivity index is one of ecoDoppler indicators that could be used to track the evolution of renal injury; any confounding factors should be taken into consideration. Non-invasive along with practical methods for treating kidney illness include ultrasound, color-Doppler; when combined with laboratory data, they enable quick and accurate clinical, screening, and treatment classification. Additionally, it permits early treatment, which eventually leads to better patient care (30).



Wash in–out curves (CEUS) in acute rejection using a low mechanical index (MI) (range 0.04–0.1) after administration of SonoVue (BraccoTM)

Figure 2. 2

Honda T et al, in 2019 conducted a research that For aerobic creatures to survive, oxygen is necessary. When mitochondria use oxygen to produce energy, reactive oxygen species (ROS) are continuously produced. Oxidative stress is caused by any imbalance between the generation of ROS and associated clearance system. Numerous diseases are known to be linked to oxidative stress, a major cause of tissue damage. irrespective of the primary aetiology, the kidney's function is vulnerable to low oxygen levels, and kidney hypoxia occurs as a frequent final phase of kidney failure. Oxidative stress is made worse by renal hypoxia, while renal hypoxia is made worse by increased oxidative stress. Through a number of processes, persistent kidney disease, particularly diabetic renal disease, also increases oxidative stress. Thus, the course of renal damage is significantly influenced by the vicious loop between oxidative strain and kidney hypoxia. After reviewing current data linking oxidative stress and recurrent hypoxia in kidney failure, this review outlines a number of prospective anti-oxidative stress treatment strategies (31).

Gembillo G et al in 2019 conducted a research that, Diabetes mellitus (DM), whose incidence have been steadily rising in recent years, is a serious global public health

concern. According the predictions of the Institution for Alternate Futures (IAF), during 2015 to 2030, the overall number of Americans with type 1 and type 2 diabetes is predicted to rise by 54 percent. Around one third of the people with diabetes mellitus have diabetic nephropathy (DN), which is presently the most common reason of end-stage kidney dysfunction in Western countries. The intricate relationships between vitamin D and progressive, chronic changes linked to deteriorating renal function are intimately related. The vitamin D depending pathways become dysregulated as consequence of these modifications. In contrast to the rise in glomerular membrane thickening, vitamin D supplementation has been shown in numerous trials to play a vital part in the reversal of albuminuria along with glomerulosclerosis, in addition to in improving renal outcomes. The interaction between endothelium as well as podocytes is crucial for maintaining homeostasis and controlling nephron function. In order to establish the outcomes of continual vitamin D intake in DN while to assess the efficacy of this treatment and the proper dosage, it remains worthwhile to investigate such factors with additional in vitro research and randomised controlled trials in wider patient series, although mounting evidence suggests that vitamin D might have antiproteinuric, protective effects and anti-inflammatory effects for individuals with DN (32).

Table 3. Vitamin D baseline status in DN patients with different values of GFR. Data are expressed as mean $\pm$ standard deviation or median (IQR).

Study, [Reference], (Year)	Populations Characteristics: Adults with CKD (either Secondary to Type 1 or 2 Diabetes Mellitus)	Serum 25 (OH) Vit D Baseline Levels in Different Stages of CKD			
		eGFR <15 mL/min/1.73 m ² (Stage 5) Patients Number (n)	CKD eGFR 15-60 mL/min/1.73 m ² (Stage 3-4) Patients Number (n)	eGFR 60-90 mL/min/1.73 m ² (Stage 2) Patients Number (n)	eGFR >90 mL/min/1.73 m ² (stage 1) Patients Number (n)
Navaneethan Sankar D [61] (2011)	2403 (n) DM with stage 3-4 CKD age 71.5 $\pm$ 11.7 yrs 33% men, 67% women	-	1626 (n) (67.67%): 15-29 ng/mL 777 (n) (32.33%): <15 ng/mL	-	-
Wahl [62] (2012)	1820 (n) DM with eGFR 40.7 $\pm$ 12.8 mL/min/1.73 m ² age 59.5 $\pm$ 9.8 years	-	1820 (n) 23.9 $\pm$ 13.3 ng/mL	-	-
Peng [63] (2015)	144 (n) T2DM and eGFR 45.2 (40.3-53.2) mL/min/1.73 m ² age 65 (IQR 52-75) years, 65.3% men, 34.7% women median diabetes duration 14.5 (IQR 9.5-19.0) years	-	144 (n): 8.5 (6.8-11.3) ng/mL	-	-
Sipahi [64] (2016)	1463 (n) T2DM and CKD age 14-88 years 37% men 63% women serum level of Vitamin D <20 ng/mL reported in 770 (52.0%) serum level $\geq$ 20 and <30 ng/mL in 357 (24.0%) patients.	-	6 (n) (0.8%) (stage 4) <20 ng/mL 1 (n) (0.3%) (stage 4): $\geq$ 20 and <30 ng/mL 99 (n) (7.9%) (stage 3) <20 ng/mL 25 (n) (7.1%): $\geq$ 20 and <30 ng/mL (stage 3)	239 (n) (31.9%): <20 ng/mL 112 (n) (31.9%): $\geq$ 20 and <30 ng/mL	446 (n) (99.5%): <20 ng/mL 212 (n) (80.4%): $\geq$ 20 and <30 ng/mL
			23 (n) (7.1%) (stage 3): $\geq$ 30 ng/mL	106 (n) (32.9%): $\geq$ 30 ng/mL	193 (n) (99.9%): $\geq$ 30 ng/mL
Xiao [71] (2016)	240 (n) with T2DM, persistent microalbuminuria (AER 30-300 mg/24 h) or macroalbuminuria (AER >300 mg/24 h)	60 (n): 7.74 $\pm$ 2.90 ng/mL	60 (n) (stage 4): 8.44 $\pm$ 2.53 ng/mL 60 (n) (stage 3): 10.31 $\pm$ 3.56 ng/mL	60 (n): 12.23 $\pm$ 4.07 ng/mL	-
Ray [71] (2017)	72 (n) with "DM" age 54.2 $\pm$ 11.7 years 44 men, 28 women; 24.2% of CKD subjects were vitamin D deficient (<10 ng/mL) 41.4% having vitamin D insufficiency (10-20 ng/mL).	30 (n): 10.95 (IQR 9.3, 16.4) ng/mL	42 (n) (stage 4): 19.15 (IQR 13.6, 23.4 ng/mL)	-	-

(CI) Confidence interval, (CKD) chronic kidney disease, (eGFR) estimated glomerular filtration rate, (DN) diabetic nephropathy, (DM) diabetes mellitus, interquartile range (IQR), (n) number, (Yrs) years.

Figure 2. 3

Colombo M et al, 2020 evaluated The purpose of the research was to report information regarding the incidence and determinants of renal deterioration in type1 diabetes mellitus (DM) from a current population cohort. 5777 adults with type 1 DM who were 16 years of age or bit older, detected before they reached age of fifty years old, and a representation of Scotland's people with type 1 diabetes community were included as the cohort that we used. During the recruitment process, we took measurements of serum creatinine plus the urinary creatinine ratio (ACR), and afterwards connected the information to the electronic medical records. The average age had been 44.1 years, while the duration of diabetes were 20.9 years. The mean incidence of end-stages renal disease (ESRD) and chronic kidney disease (CKD) phases G1 was 64%, G2 was 29.3%, G3 was 5.4 %, and G4 were 0.7%, respectively. The average annual drop in eGFR during the 11.6-years period of observation was moderate. Heart disease (OR 1.9, $p = 5 \times 10^{-5}$) and retinal (OR 1.3, $p = 0.02$) were more common among such decliners. The prognosis of final eGFR was increased by including HbA1c, preceding cardiovascular illnesses. Prediction was not improved by attempts to identify latent types of decliners or predict linearity in eGFR decline. Compared to previous estimates, these data demonstrate significantly reduced levels of renal disease. Early detection of people who will suffer a substantial drop in eGFR is still difficult, though (33).

Nampoothiri RV et al, in 2019 included 100 nonalcoholic individuals who had hepatic steatosis on ultrasonography were included in the research. The GFR and the evaluation of 24-hour urine protein along with microalbumin were used to estimate the presence of kidney disease. Renal dysfunction was associated with a number of risk factors, such as elements of the metabolic syndrome, the degree of hepatic steatosis (measured by ultrasound), the liver necro-inflammation (measured by hepatic amino acids), and liver fibrosis (measured by intermittent elastography). There was evidence of decreased kidney function among 28 individuals with NAFLD, including 5 (5%) with aberrant GFR, 18 (18%) with severe proteinuria, including 5 (5%) with both. From transient elastography, the diagnosis of type II diabetes mellitus, elevated hepatic transaminases, alongside advanced fibrosis had been determined as distinct predictors of kidney dysfunction. The detection of chronic fibrosis had the best specificity of 90% and elevated hepatic transaminases with sensitivity (89%). When evaluating kidney failure in individuals with

non-alcoholic fatty liver disease (NAFLD), a model that included all three factors performed well. Renal impairment affects about a third of those diagnosed with NAFLD. The degree of liver dysfunction and a history of DM determine the incidence of kidney dysfunction in NAFLD patients (34).

Provenzano M et al, in 2019 found that A high ultrasonography renal resistive indices (RI) has recently been identified as a sign of the responsiveness to nephroprotective medications and is predictive of cardiorenal the results in CKD. Therefore, doctors may find it relevant to have a risk profile of CKD patients with aberrant RI. Clinical and ultrasonography analyses were performed on consecutive individuals who were referred to our non-dialysis CKD clinic between January 1, 2016, and January 12, 2016. Aged >18 years with the presence of chronic kidney disease (CKD), which is recognised as a glomerular estimation <60 mL/min/1.73 m² as well as proteinuria >0.150 g/24 h, were prerequisites for inclusion. The primary conditions for exclusion include acute kidney damage, solitary kidney, and the presence of renal stenosis. The mean of three measurements in each kidney's artery segments was the Renal Index value. To assess relationships between clinical factors and longitudinal RI, univariate data analysis was used. 69.9% of the 73 patients in our study were men. 0.67 ± 0.09 was the mean RI. The median eGFR was 54.1 mL/min/1.73m², and the prevalences of diabetes and cardiovascular disease (CVD) were 19.2% and 20.6%, respectively. eGFR and haemoglobin levels fell from low (<0.65) to intermediate (0.65–0.70) to high (>0.70) RI categories, however diabetes, heart disease (CVD), phosphate, and smokers increased. According to univariate analysis, RI was substantially correlated with age, blood phosphorus, eGFR, the compound urea haemoglobin, diabetes, and cardiovascular disease. Independent of eGFR, the evidence indicates that RI is higher in those with CKD with CVD, DM, smoking, and elevated blood phosphorus. To confirm if a greater RI is indicative of a more intricate intrarenal injury route beyond kidney activity, more research is required (35).

Horton WB et al in 2021 conducted a research that Microvascular impairment in diabetes and cardiovascular disease is examined inclusively in the present review. Arterial and tissue function are typically homeostatically modulated by dynamic interactions among the the vessel walls and resident tissues elements in almost every organs.

Individually (or in combination in cardiometabolic disorder), diabetes mellitus, high blood pressure, being overweight, and dyslipidaemia all impair this control, with dysfunction acting as an early indicator of change. Specifically, we propose that the well-known renal, and neurological consequences of diabetes mellitus are late-stage signs of microvascular damage that starts years earlier and is frequently exacerbated by other aspects of cardiometabolic disorders (e.g., obesity, dyslipidaemia, hypertension). We highlight data showing that anatomic microvascular illness in various organs, in addition to in the cardiac muscle and nervous system. According to hypothesis, cardiometabolic disorders and diabetes can both result in reversed microvascular injury and related dysfunction in the early stages. These conditions may or may not eventually progress to permanent and structurally identifiable diseases. In addition to the well-known visual loss, decreased kidney function, and peripheral neuropathy, further consequences may include heart failure, sarcopenia, cognitive decline, and worsening metabolic dysfunction. Thanks to new genetic along with imaging technologies, our knowledge of healthy vascular functioning and early impairment is expanding quickly. As a result, new preventive measures are being tested in tissues like the retina at the earliest, reversible phases of microvascular damage. There is a great deal of confidence that several of these techniques could become successful treatments (36).

Liu B et al, in 2019 found that Among the most prevalent types of diabetes mellitus in the world is type 2. Insulin resistance along with hyperglycemia are important factors in type II diabetes. A key component of glycaemic regulation is renal glucose reabsorption. In healthy people with euglycemia, the kidneys filter 160 g of glucose every day. Cardiovascular disease and impaired renal function are two diabetes-related cardiovascular conditions that are becoming more common as a result of the growing diabetes epidemic. One essential mechanism for development, metabolism, and equilibrium is cellular intake of glucose. There are three categories for glucose transporters known to exist in humans: the sodium-glucose cotransporter SGLTs, the glucose facilitators GLUTs, and the newly discovered SWEETs. All three families' main isoforms structures were examined. The majority of the capacity is provided by the sodium-glucose cotransporter (SGLT2). Inhibitors of SGLT2 have been shown to lower mortality from cardiovascular causes in people with type II diabetes in numerous studies

of cardiovascular results trials. These elements are outlined in the current review study, which also explores potential ways that SGLT2 inhibitors may prevent heart failure and renal impairment in diabetic individuals. SGLT2 inhibitors decrease bodily weight and total fat by glucosuria, which also causes a change in substrate usage from carbs to lipids and potentially ketone bodies. The results showed that the SGLT2 inhibitor decreased serious cardiovascular incidents and slowed up the development of chronic renal disease in people at risk with diabetes (37).

Iwase M et al, in 2019 conducted a research in Japan that it has not gathered enough epidemiological data on diabetic kidney disorders. In Japanese individuals with diabetes, we prospectively examined the prevalence of ESRD (end-stage renal disease) and indicators of risk for the development of impaired renal function. Using a multicenter OPD registry, 4904 individuals with type II diabetes were examined for the development of ESRD needing dialysis over a 5-year period. The yearly eGFR drop rates and associated risk factors underlying the evolution of renal impairment were assessed. As chronic renal disease progressed, the incidence percentages of ESRD along with all-cause mortality rose, reaching 4.1/1000 individual years as well as 12.3/1000 individual years, respectively. At five years, the prevalence of annual decrease of at least 30% in eGFR from the starting point was recorded. Older age, poor glycaemic control, high blood pressure, eGFR, prior cardiac disease, lifestyle variables (BMI, reduced dietary fibre intake, increased intake of sodium, lack of regular physical activity), and depressive symptoms were all significantly linked to the progression of kidney problems. The significance of multifactorial approaches on risk variables in order to reduce the elevated rate of end-stage renal disease (ESRD) in Japanese population had been highlighted by this prospective research (38).

Kuo C-C et al, in 2019 found that because kidney ultrasound imaging is safe, convenient, and reasonably priced, it continues to be regarded as ideal in the clinical setting for the prediction of renal function as well as chronic kidneys disease (CKD). To automatically ascertain the approximate glomerular filtration rate (eGFR) and CKD status, we created a deep learning method. Using 4,505 renal ultrasound pictures labelled with eGFRs generated from serum creatinine concentrations, we used the transfer learning approach to anticipate kidney function by integrating the potent ResNet model preconditioned using

an ImageNet dataset into neural network design. Researchers used kidney length markers to exclude the kidneys' periphery and several data augmentation techniques to provide more data with variables in order to expand relevant data from ultrasonography pictures. Additionally, bootstrap aggregation had been employed to enhance the model's generalisation and prevent overfitting. Furthermore, the CKD situation, which is indicated by an eGFR of less than 60 ml/min/1.73 m², was determined using renal function features that deep neural system had acquired. The significant association between GFR calculations generated by artificial intelligence (AI) with creatinine was demonstrated by a Pearson correlation coefficient of 0.741. The model used had a general classification precision of 85.6% for CKD status, which was greater than of skilled nephrologists (60.3%–80.1%). The first essential step in achieving the goal of turning kidney ultrasonography into a useful, real-time, remote screening instrument is model. One of the main objectives of AI-powered functioning automation into clinical practice is the potential for noninvasive renal function testing, which AI-GFR estimation provides (39).

Variables	Patients (N = 1297)	Ultrasound Images (N = 4505)	Ultrasound Images in nontesting dataset (N = 4,010)	Ultrasound Images in testing dataset (N = 495)	p value ^a
Demographics, median (IQR)					
Age (years)	65 (53, 74)	65 (52, 74)	65 (53, 74)	63 (51, 74)	0.269
Male, n (%)	715 (55.1)	2464 (54.7)	2201 (54.9)	263 (53.1)	0.459
Comorbidity, n (%)					
Cardiovascular disease	1001 (77.2)	3504 (77.8)	3117 (77.7)	387 (78.2)	0.820
Hypertension	968 (74.6)	3405 (75.6)	3024 (75.4)	381 (77.0)	0.446
Diabetes	533 (41.1)	1888 (41.9)	1697 (42.3)	191 (38.6)	0.112
Biochemical value, median (IQR)					
Serum creatinine (mg/dL)	2.1 (1.4, 4.4)	2.1 (1.4, 4.3)	2.1 (1.4, 4.4)	2.0 (1.4, 3.6)	0.003
eGFR (mL/min/1.73 m ²)	30.0 (12.3, 48.6)	30.1 (12.6, 48.7)	29.9 (12.3, 48.2)	31.6 (15.7, 55.5)	0.001
Sonographic parameter, median (IQR)					
Kidney length (cm)	9.72 (8.76, 10.53)	9.62 (8.69, 10.51)	9.63 (8.66, 10.51)	9.54 (8.80, 10.48)	0.703

^ap values denotes probability for difference between the nontesting and testing datasets and are calculated by Wilcoxon rank sum test for continuous variables and Chi-square test (or Fisher's exact test as appropriate) for categorical variables

Figure 2. 4

Patyna S et al, in 2019 concluded that the most prevalent organ manifestation of chronic lupus erythematosus (SLE) and lupus nephritis (LN), which is also linked to a bad prognosis. There is yet no proven noninvasive technique for diagnosing LN. Therefore,

we assessed the potential of blood sphingolipids as reliable biomarkers for renal damage. The 82 respondents in the present longitudinal study were split into 3 categories: 29 LN individuals, 17 individuals with SLE minus renal damage, and 36 unaffected controls. Kidneys biopsies and compromised renal function had been employed to identify LN patients. All subjects had venous blood drawn, and LC-MS/MS were employed to measure the amounts of sphingolipids in the plasma or serum. The findings indicate that chain-length particular ceramides in bloodstream, most probably C24:1Cer amounts in serum, may function as powerful biomarkers for impaired renal function among individuals with SLE. Additionally, ROC evaluates of both serum and plasma samples containing the most modified ceramide species across LN subjects and SLE subjects show a high assessment differentiation and elevated AUCs, particularly for C24:1Cer blood plasma levels. Additionally, C24:1Cer serum concentrations were unaffected by glucocorticoid therapy and demonstrated no correlation with other kidney-related markers, including serum creatinine, albumin ration/ creatinine ratio and eGFR (40).

Klinkhammer BM et al, in 2021 investigated the glomerular filtration rate, urine sediment, levels of protein, including tissue from invasive renal biopsies are key components of a multiple diagnosis including the disease's progress evaluation in nephrology. Yet, the majority of serum plus urine biomarkers, as well as currently available non-invasive functional indicators, are unable to precisely identify intrarenal conditions. Furthermore, while both morphological and molecular changes can be seen in kidney biopsy samples by histopathological investigations, these analyses only reveal details about a limited portion of the the kidneys and does not permit ongoing monitoring. These restrictions not only make it more difficult to comprehend the dynamics of particular a kidney disorder processes, but they also restrict the creation of new tailored therapies and the ability to target interventions only during active stages of the illness. By integrating imaging methods with particular molecular instruments, molecular imaging allows for the quantitative along with non-invasive evaluation of physiologic versus pathological conditions. Here, we go over the most recent nephrology preclinical and clinical molecular imaging techniques. The safe and efficient use of medications as well as companion diagnostics to direct clinical trials can be facilitated by

the non-invasive visualisation of the kidneys using molecular imaging, which can be used to identify and track disease activity over time (41).

Argaiz ER et al, in 2021 found Nephrologists' primary focus is on treating complicated fluid and electrolyte imbalances. Accurate clinical judgement is impossible due to the poor sensitivity for bodily examination primarily in determining fluid status. A useful noninvasive bedside screening technique for the objective assessment of physiologic and haemodynamic parameters linked to condition of fluids including susceptibility is point-of-care ultrasound (POCUS). Quantitative information on pulmonary congestion and qualitative information on heart function may both be obtained with a quick bedside sonographic assessment. Goal-directed Doppler echocardiography alongside additional modern POCUS techniques offer more quantitative data, such as pressures along with flow velocities throughout the heart structures. Congestive organ dysfunction has recently been connected to aberrant Doppler circulation patterns across the abdominal cavity caused by elevated right ventricular pressure, which adds another element to the haemodynamic evaluation. Further clarification of a patient's haemodynamic condition can be achieved by combining POCUS results with clinical and laboratory data. This enables customised treatment for each patient and influences choices upon the administration of crystalloids or, on the other hand, diuresis and ultrafiltration. During this study, researchers explore significant study results and give a brief description of the targeted evaluation of cardiac function as well as pulmonary alongside venous congestion by employing POCUS (42).

Deferarri G et al, in 2019 conducted a research that Haemodynamic equilibrium depends on the heart with kidney working together, as is widely recognised. It has been known since the early 1800s that cardiac and renal disorders often coexist. Although it is widely acknowledged in the field of nephrology that cardiovascular illnesses are more likely to arise in people with renal disorders, it is not generally understood that cardiovascular disorders can cause or worsen renal dysfunctions, which further impairs cardiac function and creates a vicious cycle. The part that venous congestion plays in the aetiology of renal failure is sometimes disregarded in a similar clinical domain. Irrespective of the ejection fraction, this research meticulously measures the prevalence of acute and chronic kidney abnormalities in cardiovascular illnesses, primarily heart failure, and the effects of kidney

disorders on primarily heart failure, as well as the effects of renal abnormalities on both organs, establishing cardiovascular disorders as an important risk indicator in kidney diseases. Furthermore, in terms of pathophysiological features, we try to demonstrate how venous congestion, particularly renal venous a high blood pressure and fluid overload play a crucial role in the pathophysiology of both acute as well as chronic kidney disease that occurs in cardiac failure (43).

Chapter 3

METHODOLOGY

3.1. Study Design:

The current study design was cross sectional in nature.

3.2. Study setting:

Study setting was Allied Hospital Faisalabad

3.3. Sample Size:

250 Participants were included in this study.

3.4. Sampling Technique:

Non-probability purposive sampling technique was used.

3.5. Study Duration:

The duration of study was 4 month after approval synopsis.

3.6. Sample Selection Criteria

3.6.1 Inclusion Criteria

All diabetic adult individuals 18 years and above were experienced diagnostic renal ultrasonography from August 2024 to October 2024 after consenting, BMI Participants across all BMI categories (underweight, normal weight, overweight, and obese) as defined by WHO standards, Both male and female participants were included in this study (45).

3.6.2 Exclusion criteria:

(1) Pregnant women due to physiological changes in the kidneys that can affect the study outcomes. (2) Known Subjects with history of acute kidney injury Kidney transplant

patients. (3) Patients on hemodialysis or peritoneal dialysis. The current study design was cross sectional in nature (11).

3.7. Ethical Consideration

- Ethical approval was obtained from research and ethics committee.
- Data was collected by using permission letter.
- Consent was taken from target population.
- Privacy and confidentiality of population was ensured.

3.8. Data Collection Procedure:

Parameters:

Renal ultrasonography: All renal ultrasonography was performed by experienced investigators in ultrasonography unit. Examinations was performed in a supine or prone position using standard gray-scale with 3.5 MHz measurements. The renal length (RL) was measured as the longest pole-to-pole distance in the sagittal plane measured to the nearest millimeter. In the sagittal plane, the renal cortical thickness (CK, from the medullary pyramid to the capsule) and the renal parenchymal thickness (PK, from the cortex to the renal sinus fat) was measured on the upper, middle, and lower thirds of the kidney. To correct the renal size differences based on the patient's height.

Demographic and Anthropometric Data:

Age: Recorded in years.

Sex: Male or Female.

BMI: Calculated as weight (kg) / height (m²)

Renal volume: calculated as: $0.523 \times \text{length (cm)} \times \text{width (cm)} \times \text{depth (cm)}$.

Blood Sample Collection:

Venous blood samples was drawn under sterile conditions. Serum Creatinine was measured using the Jaffe method. Estimated glomerular filtration rate was also measured. Random blood glucose was measured.

Procedure:

250 patients were included in the research. All the patients were asked for their informed consent. After gathering demographic information, medical history, history of medications, drinking, and active smoking will be examined. Every patient received a questionnaire, and a thorough clinical examination was conducted. Ultrasonography were used to evaluate the echogenicity of the both kidneys. A visually measured segment representing the biggest section running longitudinally from one pole to another pole was used for determining the length. A section of the kidney perpendicular to the kidney's longitudinal axis was used to measure the width and thickness. From the renal hilum to the convex boundary of the lateral renal margin, the parenchymal thickness was measured. The cortical thickness was measured perpendicular to the capsule in the sagittal plane of the medullary pyramid. The mean renal longitudinal size, parenchymal thickness, and cortical thickness values for each subject were determined.

Analysis of a Sample

Venous blood was drawn from each participant for biochemical examination. The alkaline picrate technique was used to quantify serum creatinine.

Operational Definition

Early detection of renal dysfunction, or kidney disease, typically involves identifying abnormalities in kidney function tests before significant symptoms appear, often focusing on markers like albuminuria and reduced glomerular filtration rate (GFR). This often means detecting these changes in individuals at higher risk, such as those with diabetes or high blood pressure, before they progress to more severe stages.

3.9. Data analysis

The data was presented as mean and standard deviation (mean $\pm$ SD). One-way analysis of variance (ANOVA) was used to calculate the statistical analysis, and the Pearson correlation coefficient was computed for bivariate associations. The statistical significance was established at the 5% ($p < 0.05$) level using SPSS version 27.

Chapter 4

RESULTS

The study included 250 participants, with 34.4% male and 65.6% female. BMI distribution showed 12.4% underweight ($<18.5 \text{ kg/m}^2$), 42.0% normal weight ($18.5\text{-}24.99 \text{ kg/m}^2$), 23.2% overweight ($25\text{-}29.99 \text{ kg/m}^2$), and 22.4% obese ($>30 \text{ kg/m}^2$), with a mean BMI of $25.640 \pm 6.505 \text{ kg/m}^2$. Normal kidney size was observed in 85.6% of right kidneys and 86.0% of left kidneys, while increases were seen in 10.4% and 10.0%, respectively, and decreases in 4.0% for both. The mean age was 38.388 ± 15.424 years, mean random blood glucose $150.216 \pm 37.150 \text{ mg/dL}$, systolic blood pressure $124.160 \pm 14.349 \text{ mmHg}$, diastolic blood pressure $81.980 \pm 8.972 \text{ mmHg}$, serum creatinine $0.975 \pm 0.710 \text{ mg/dL}$, and eGFR $79.386 \pm 18.527 \text{ mL/min/1.73 m}^2$.

The analysis of serum creatinine and eGFR by gender and BMI showed that male participants had higher serum creatinine and eGFR levels with increasing BMI. Serum creatinine ranged from $0.876 \pm 0.341 \text{ mg/dL}$ in males with BMI $<18.5 \text{ kg/m}^2$ to $1.325 \pm 0.677 \text{ mg/dL}$ in those with BMI $>30 \text{ kg/m}^2$, with an overall mean of $0.997 \pm 0.503 \text{ mg/dL}$ ($p=0.113$). eGFR increased from $77.257 \pm 11.959 \text{ mL/min/1.73 m}^2$ to $101.125 \pm 16.762 \text{ mL/min/1.73 m}^2$, with a significant p-value of 0.001. In females, serum creatinine ranged from $0.790 \pm 0.310 \text{ mg/dL}$ in the <18.5 BMI group to $1.097 \pm 1.004 \text{ mg/dL}$ in the >30 BMI group, with a mean of $0.963 \pm 0.798 \text{ mg/dL}$ ($p=0.014$). eGFR ranged from $62.200 \pm 10.464 \text{ mL/min/1.73 m}^2$ to $84.595 \pm 13.180 \text{ mL/min/1.73 m}^2$, with a significant p-value of <0.001 .

The distribution of kidney size abnormalities varied significantly across BMI categories and genders ($p<0.001$). Most participants had normal kidney size. For the right kidney, normal size was observed in 85.6% of participants, with 10.4% showing an increase and 4.0% a decrease. For the left kidney, 86.0% were normal, 10.0% had an increase, and 4.0% a decrease. Participants with BMI $18.5\text{-}24.99 \text{ kg/m}^2$ had the highest percentage of normal kidneys (38.8%), while those with BMI $>30 \text{ kg/m}^2$ had slightly higher rates of

abnormalities. Gender-wise, females were more likely to have normal kidneys (58.4% right, 58.8% left) compared to males (27.2% for both), while males had slightly higher rates of size increases (5.6% right, 5.6% left).

Table 4. 1: Demographic Information of study Participants

		N	%
Gender	Male	86	34.4%
	Female	164	65.6%
Body Mass Index	<18.5 BMI (kg/m ²)	31	12.4%
	18.5-24.99 BMI (kg/m ²)	105	42.0%
	25-29.99 BMI (kg/m ²)	58	23.2%
	> 30 BMI (kg/m ²)	56	22.4%
Right Kidney	Normal in Size	214	85.6%
	Increase (Cortical echogenicity)	26	10.4%
	Decrease (Parenchymal thickness)	10	4.0%
Left Kidney	Normal in Size	215	86.0%
	Increase (Cortical echogenicity)	25	10.0%
	Decrease (Parenchymal thickness)	10	4.0%
			Mean ±SD
Age in years		250	38.388±15.424
BMI in Kg/m ²		250	25.640±6.505
Random Blood Glucose		250	150.216±37.150
Systolic Blood Pressure		250	124.160±14.349
Diastolic Blood Pressure		250	81.980±8.972
Serum Creatinine		250	.975±.710
Estimated Glomerular Filtration Rate		250	79.386±18.527

The demographic information of the study participants is summarized in Table 1. The total sample size was 250, with 34.4% (n=86) being male and 65.6% (n=164) female. The participants' body mass index (BMI) was distributed as follows: 12.4% (n=31) had a BMI of less than 18.5 kg/m², 42.0% (n=105) fell within the 18.5-24.99 kg/m² range, 23.2% (n=58) were in the 25-29.99 kg/m² category, and 22.4% (n=56) had a BMI greater than 30 kg/m². The mean BMI across all participants was 25.640±6.505 kg/m². Regarding kidney size, 85.6% (n=214) of participants had normal-sized right kidneys, 10.4% (n=26) had increased right kidney size (Cortical echogenicity), and 4.0% (n=10) showed a

decrease in size (Parenchymal thickness). Similarly, for the left kidney, 86.0% (n=215) were normal in size, while 10.0% (n=25) had an increase (Cortical echogenicity) and 4.0% (n=10) showed a decrease (Parenchymal thickness).

The mean age of participants was 38.388 ± 15.424 years. The mean random blood glucose level was 150.216 ± 37.150 mg/DL. The mean systolic blood pressure was 124.160 ± 14.349 mmHg, while the mean diastolic blood pressure was 81.980 ± 8.972 mmHg. The mean serum creatinine level was 0.975 ± 0.710 mg/DL, and the estimated glomerular filtration rate (eGFR) had a mean of 79.386 ± 18.527 mL/min/1.73 m².

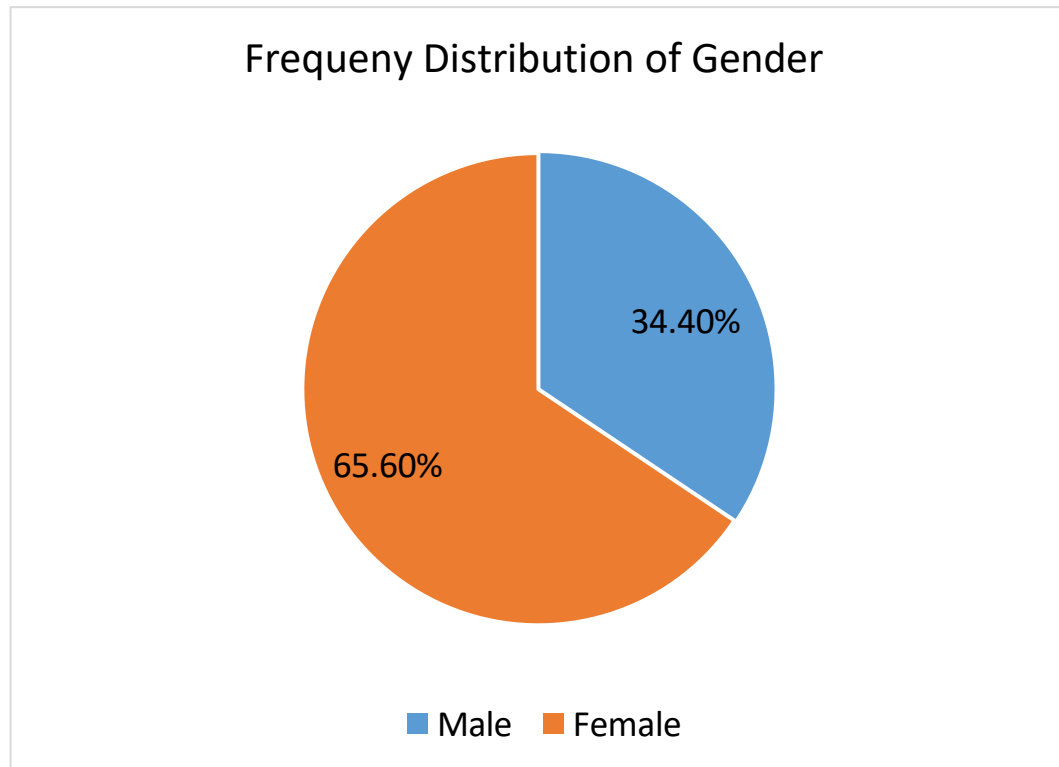


Figure 4. 1: *Frequency Distribution of Gender*

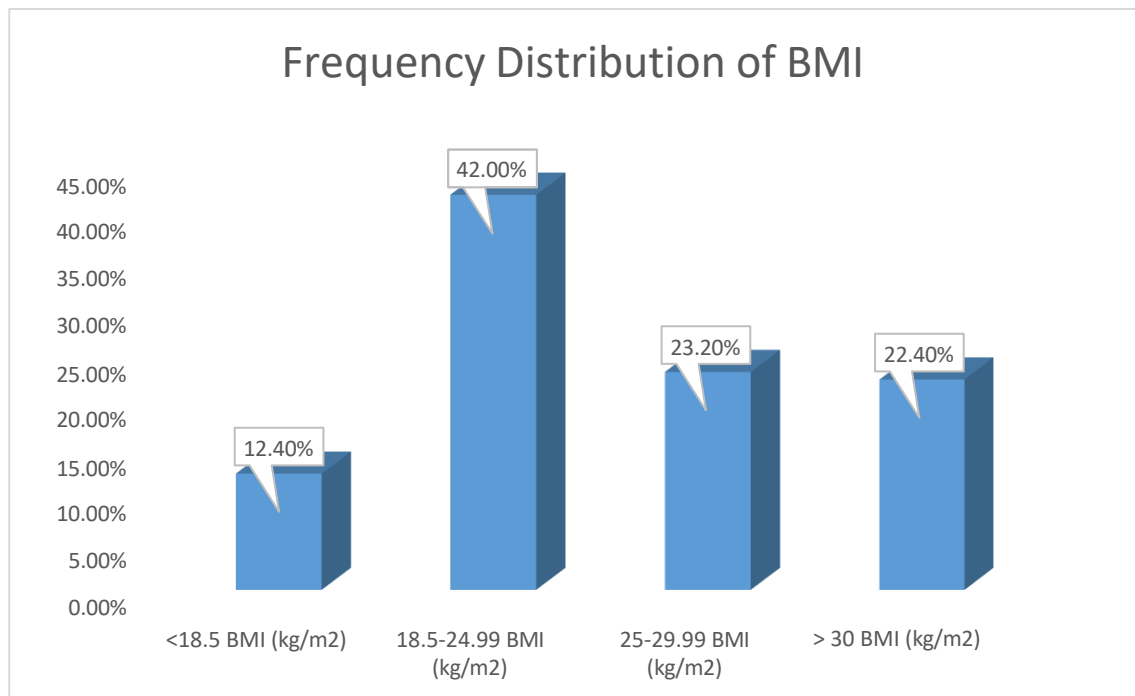


Figure 4. 2: *Frequency Distribution of BMI*

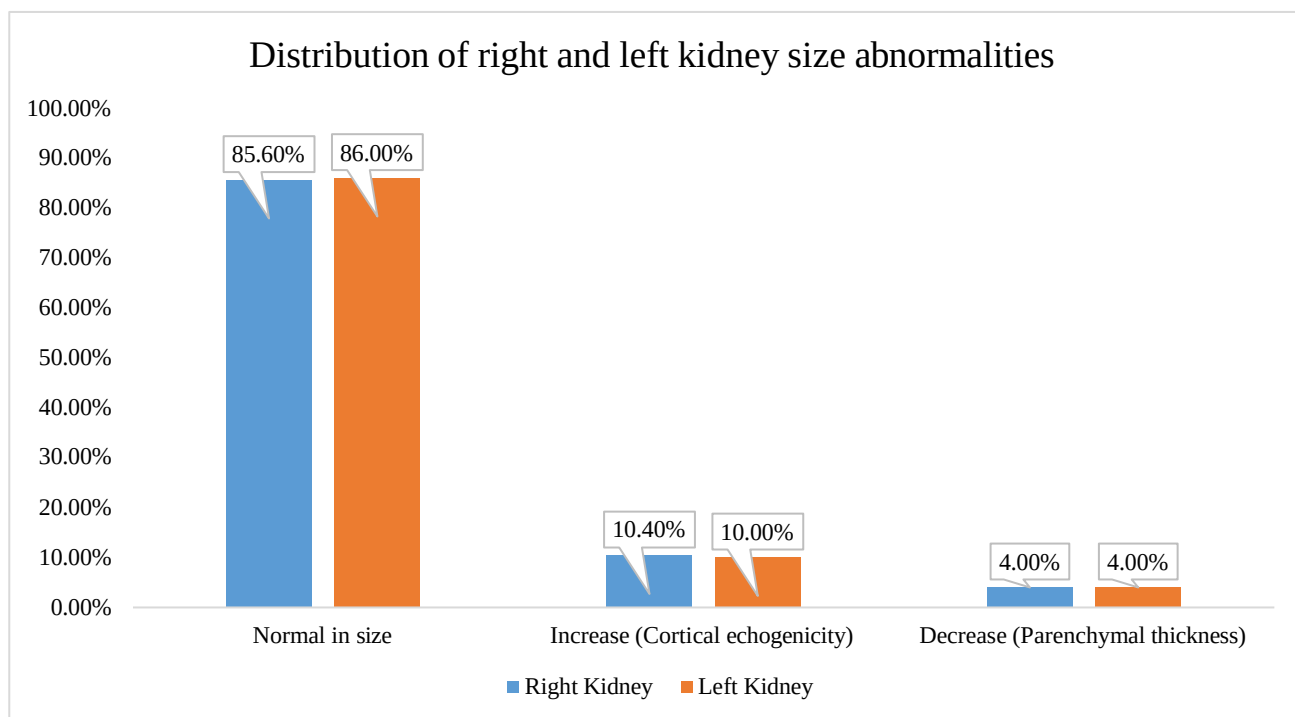


Figure 4. 3: Distribution of right and left kidney size abnormalities

Table 4. 2 Distribution of Serum Creatinine and Estimated Glomerular Filtration Rate across BMI categories and gender

				Creatinine (mg/ dl)	P value	EGFR	P value
Gender	BMI	N	%	Mean $\pm$ SD		Mean $\pm$ SD	
Male	<18.5 BMI (kg/m ²)	21	8.4%	.876 $\pm$.341	0.113	77.257 $\pm$ 11.959	0.001
	18.5-24.99 BMI (kg/m ²)	38	15.2%	.942 $\pm$.471		92.600 $\pm$ 19.721	
	25-29.99 BMI (kg/m ²)	19	7.6%	1.105 $\pm$.589		96.773 $\pm$ 18.562	
	> 30 BMI (kg/m ²)	8	3.2%	1.325 $\pm$.677		101.125 $\pm$ 16.762	
	Total	86	34.4%	.997 $\pm$.503		90.568 $\pm$ 19.073	
Female	<18.5 BMI (kg/m ²)	10	4.0%	.790 $\pm$.310	0.014	62.200 $\pm$ 10.464	<0.001
	18.5-24.99 BMI (kg/m ²)	67	26.8%	.749 $\pm$.164		66.237 $\pm$ 13.822	
	25-29.99 BMI (kg/m ²)	39	15.6%	1.210 $\pm$ 1.116		75.315 $\pm$ 12.089	
	> 30 BMI (kg/m ²)	48	19.2%	1.097 $\pm$ 1.004		84.595 $\pm$ 13.180	
	Total	164	65.6%	.963 $\pm$.798		73.523 $\pm$ 15.301	

The analysis of serum creatinine levels and estimated glomerular filtration rate (eGFR) by gender and BMI categories is summarized as follows. Among male participants, serum creatinine levels varied across BMI categories. Those with a BMI less than 18.5 kg/m² (8.4%, n=21) had a mean serum creatinine of 0.876 $\pm$ 0.341 mg/dL, while those in the 18.5-24.99 kg/m² range (15.2%, n=38) had a mean of 0.942 $\pm$ 0.471 mg/dL. Participants with a BMI of 25-29.99 kg/m² (7.6%, n=19) had higher serum creatinine levels at 1.105 $\pm$ 0.589 mg/dL, and those with a BMI above 30 kg/m² (3.2%, n=8) recorded the highest mean at 1.325 $\pm$ 0.677 mg/dL. The overall mean serum creatinine for male participants was 0.997 $\pm$ 0.503 mg/dL, with a non-significant p-value of 0.113. Male participants' eGFR also increased with BMI, ranging from 77.257 $\pm$ 11.959 mL/min/1.73 m² in the <18.5 BMI category to 101.125 $\pm$ 16.762 mL/min/1.73 m² in the >30 BMI category. The overall mean

eGFR for males was 90.568 ± 19.073 mL/min/1.73 m², showing significant variability with a p-value of 0.001.

In female participants, serum creatinine levels also varied across BMI groups. Those with a BMI below 18.5 kg/m² (4.0%, n=10) had the lowest mean serum creatinine at 0.790 ± 0.310 mg/dL, while participants with a BMI of 18.5-24.99 kg/m² (26.8%, n=67) had a mean of 0.749 ± 0.164 mg/dL. Serum creatinine was higher in the 25-29.99 kg/m² category (15.6%, n=39), with a mean of 1.210 ± 1.116 mg/dL, and slightly lower in the >30 BMI category (19.2%, n=48) at 1.097 ± 1.004 mg/dL. The overall mean serum creatinine for females was 0.963 ± 0.798 mg/dL, with a statistically significant p-value of 0.014. Female participants' eGFR ranged from 62.200 ± 10.464 mL/min/1.73 m² in the <18.5 BMI category to 84.595 ± 13.180 mL/min/1.73 m² in the >30 BMI group. The overall mean eGFR for females was 73.523 ± 15.301 mL/min/1.73 m², with significant variability indicated by a p-value of <0.001.

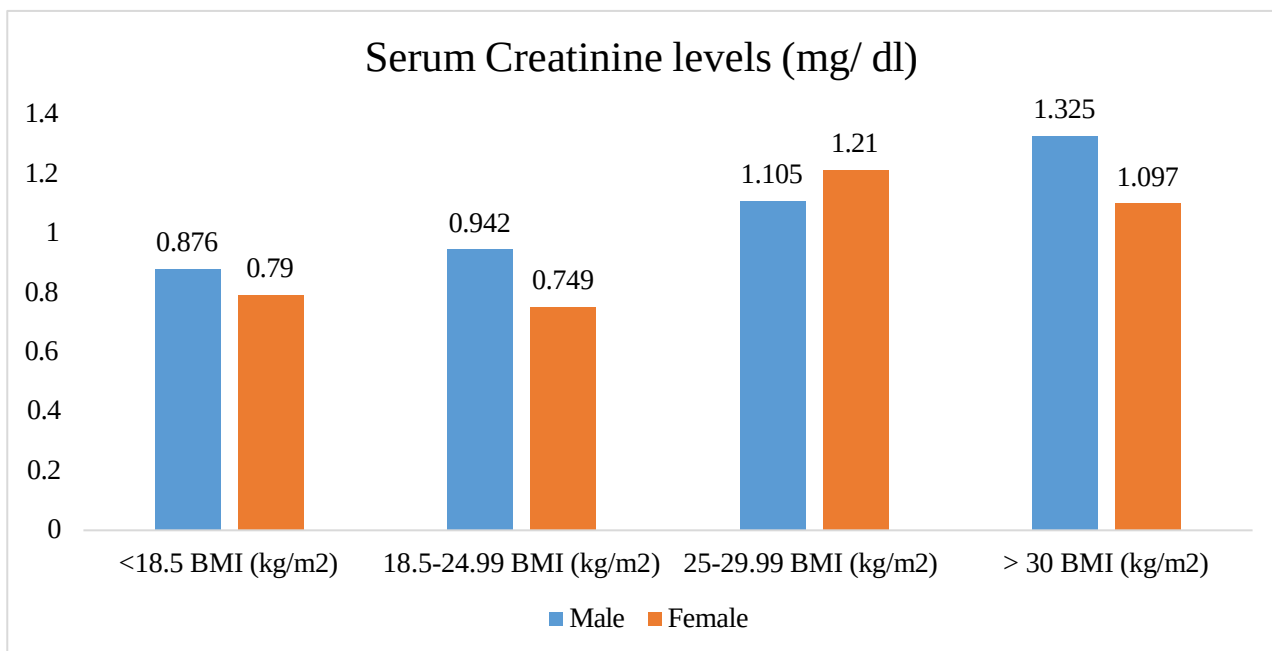


Figure 4. 4: *Distribution of Serum Creatinine level across BMI categories and gender*

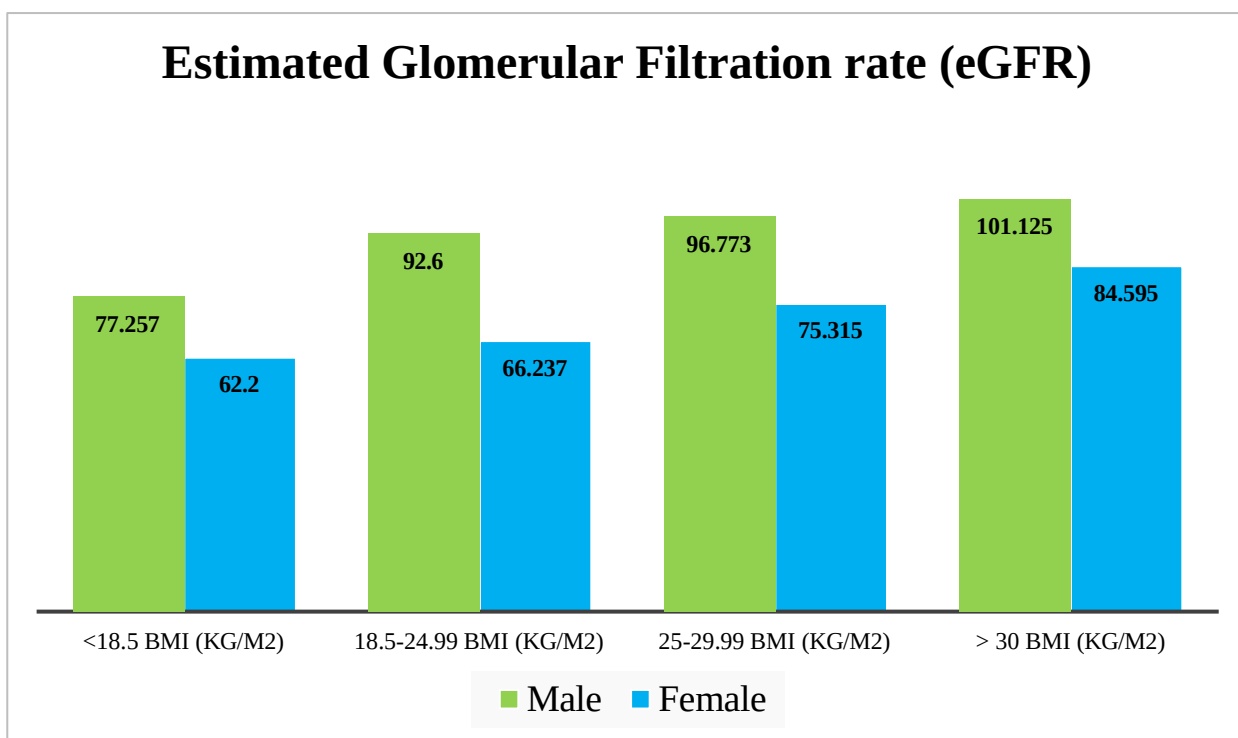


Figure 4. 5: *Distribution of Estimated Glomerular Filtration Rate across BMI categories and gender*

Table 4. 3Distribution of right and left kidney size abnormalities across BMI categories and gender

		BMI								Gender			
		<18.5 BMI (kg/m ²)		18.5-24.99 BMI (kg/m ²)		25-29.99 BMI (kg/m ²)		> 30 BMI (kg/m ²)		Male		Female	
		N	%	N	%	N	%	N	%	N	%	N	%
Right Kidney	Normal	27	10.8%	97	38.8%	45	18.0%	45	18.0%	68	27.2%	146	58.4%
	Increase (Cortical echogenicity)	1	0.4%	6	2.4%	12	4.8%	7	2.8%	14	5.6%	12	4.8%
	Decrease (Parenchymal thickness)	3	1.2%	2	0.8%	1	0.4%	4	1.6%	4	1.6%	6	2.4%
	P value	<0.001		<0.001		<0.001		<0.001		<0.001		<0.001	
Left Kidney	Normal	27	10.8%	97	38.8%	46	18.4%	45	18.0%	68	27.2%	147	58.8%
	Increase (Cortical echogenicity)	1	0.4%	6	2.4%	11	4.4%	7	2.8%	14	5.6%	11	4.4%
	Decrease (Parenchymal thickness)	3	1.2%	2	0.8%	1	0.4%	4	1.6%	4	1.6%	6	2.4%
	P value	<0.001		<0.001		<0.001		<0.001		<0.001		<0.001	

The distribution of right and left kidney size abnormalities across BMI categories and genders is shown in table 3. For the right kidney, the majority of participants had normal kidney size across all BMI categories. Among those with a BMI less than 18.5 kg/m², 10.8% (n=27) had normal-sized kidneys, while 0.4% (n=1) showed an Increase (Cortical echogenicity) and 1.2% (n=3) had a Decrease (Parenchymal thickness) in size. In the 18.5-24.99 kg/m² BMI group, 38.8% (n=97) had normal-sized kidneys, 2.4% (n=6) exhibited an Increase (Cortical echogenicity), and 0.8% (n=2) showed a Decrease (Parenchymal thickness). Among participants with a BMI of 25-29.99 kg/m², 18.0% (n=45) had normal

kidney size, 4.8% (n=12) displayed an Increase (Cortical echogenicity), and 0.4% (n=1) had a Decrease (Parenchymal thickness). For those with a BMI greater than 30 kg/m², 18.0% (n=45) had normal-sized kidneys, 2.8% (n=7) showed an Increase (Cortical echogenicity), and 1.6% (n=4) had a Decrease (Parenchymal thickness). Gender-wise, 27.2% (n=68) of males and 58.4% (n=146) of females had normal kidney size, while 5.6% (n=14) of males and 4.8% (n=12) of females had Increase (Cortical echogenicity)d kidney size. Decrease (Parenchymal thickness)d kidney size was observed in 1.6% (n=4) of males and 2.4% (n=6) of females. Across all groups, the p-value was <0.001, indicating statistically significant differences.

For the left kidney, a similar trend was observed. In the <18.5 kg/m² BMI category, 10.8% (n=27) of participants had normal kidney size, 0.4% (n=1) showed an Increase (Cortical echogenicity), and 1.2% (n=3) had a Decrease (Parenchymal thickness). In the 18.5-24.99 kg/m² group, 38.8% (n=97) had normal kidney size, 2.4% (n=6) exhibited an Increase (Cortical echogenicity), and 0.8% (n=2) showed a Decrease (Parenchymal thickness). Among those with a BMI of 25-29.99 kg/m², 18.4% (n=46) had normal-sized kidneys, 4.4% (n=11) displayed an Increase (Cortical echogenicity), and 0.4% (n=1) had a Decrease (Parenchymal thickness). For participants with a BMI >30 kg/m², 18.0% (n=45) had normal kidneys, 2.8% (n=7) showed an Increase (Cortical echogenicity), and 1.6% (n=4) had a Decrease (Parenchymal thickness). By gender, 27.2% (n=68) of males and 58.8% (n=147) of females had normal kidney size, while 5.6% (n=14) of males and 4.4% (n=11) of females had an Increase (Cortical echogenicity) in kidney size. A Decrease (Parenchymal thickness) in kidney size was seen in 1.6% (n=4) of males and 2.4% (n=6) of females. The p-value for all comparisons was <0.001, indicating significant associations.

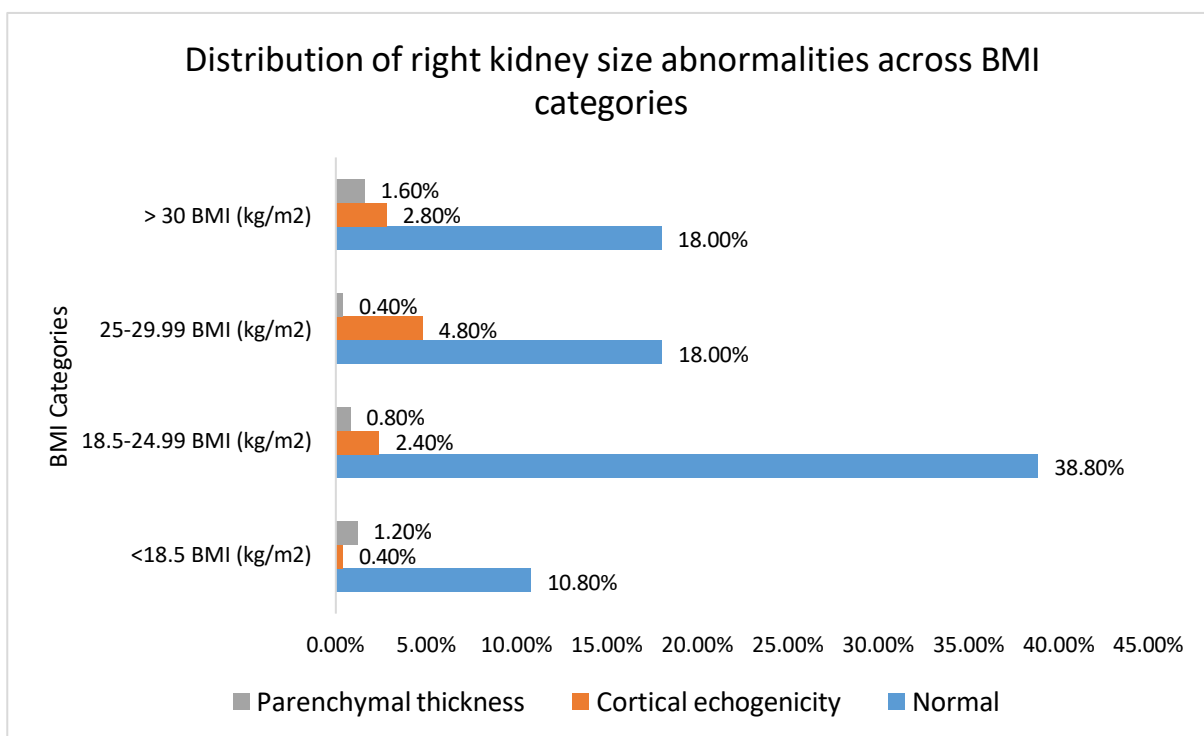


Figure 4. 6: Distribution of right kidney size abnormalities across BMI categories

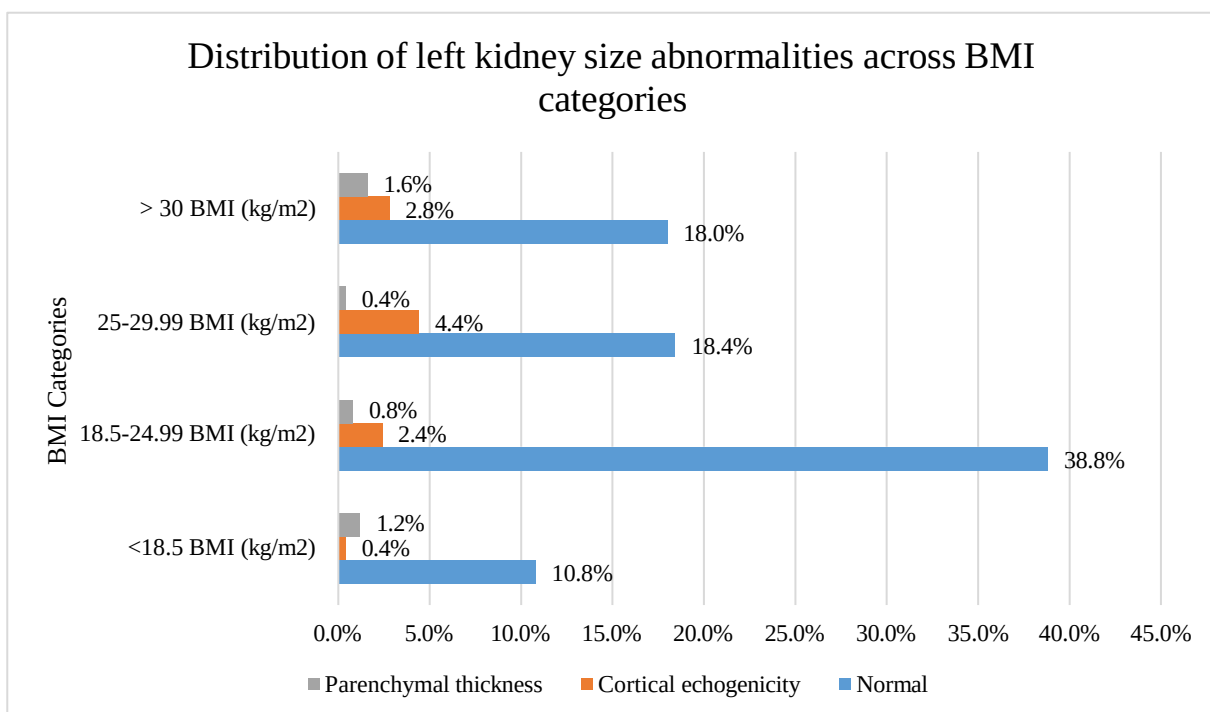


Figure 4. 7: Distribution of left kidney size abnormalities across BMI categories

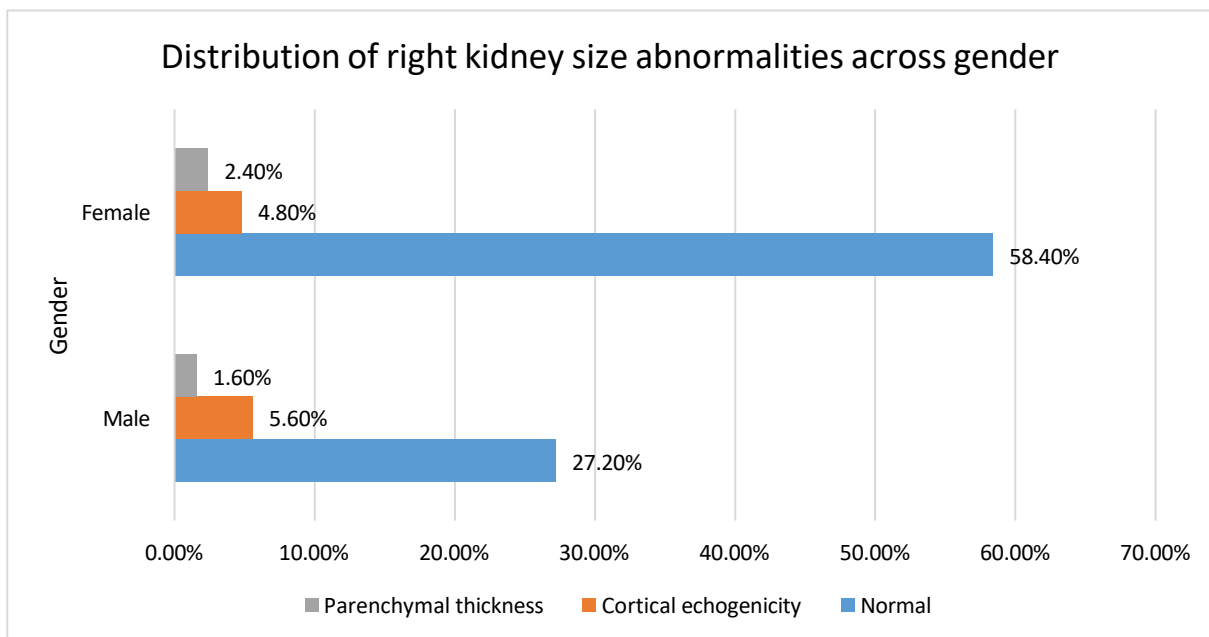


Figure 4. 8: Distribution of Right kidney size abnormalities across gender

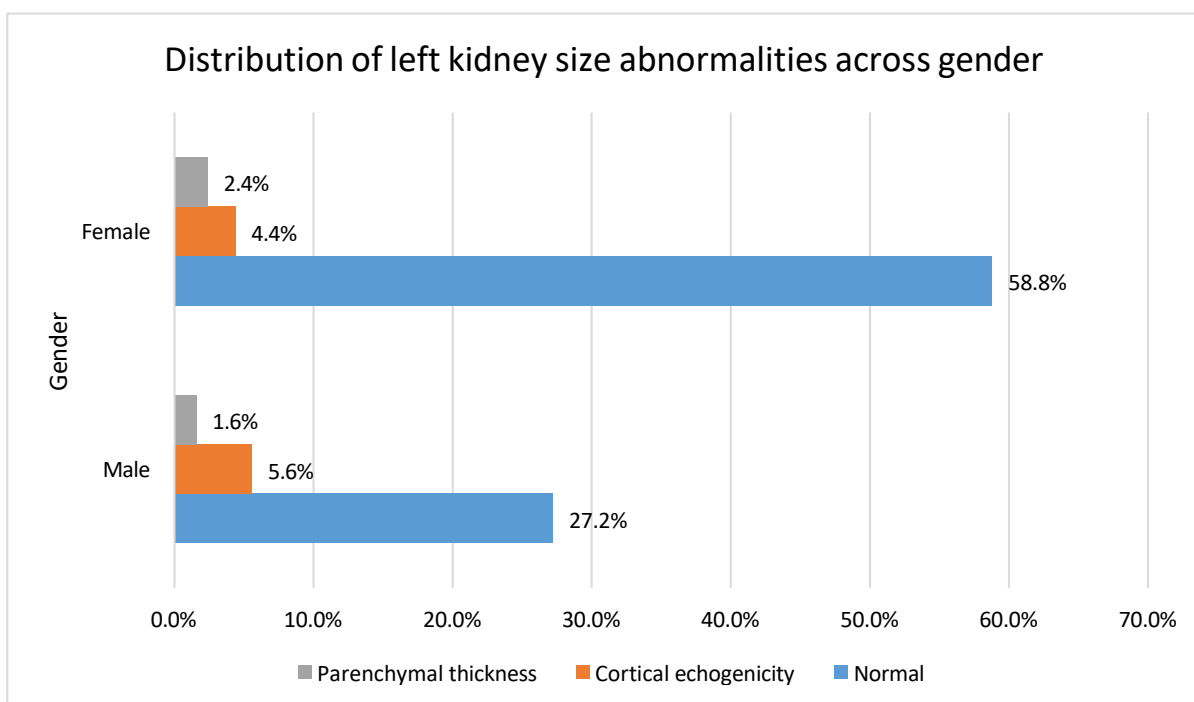


Figure 4. 9: Distribution of Left kidney size abnormalities across gender

DEPARTMENT OF RADIOLOGY & MEDICAL IMAGING
ALLIED HOSPITAL (FAISALABAD MEDICAL UNIVERSITY) FAISALABAD
Ph: 3710005 - Ext: 118

NAME: SULTAN DATE: 18-Oct-24
AGE: 62 yr REG NO: _____

USG ABDOMEN
(Not valid for more than 30 days)

LIVER: 16 cm enlarged. Fatty
is normal in size and showing normal texture parenchyma with smooth and regular margins. No definite focal lesion seen. Portal vein not dilated.

GALLBLADDER: Partially contracted. CBD is not dilated.

PANCREAS: Visualized part of pancreas is normal.

SPLEEN: Not enlarged. Normal in size.

RIGHT KIDNEY: 13 x 6.5 x 3.5 cm. Increased echogenicity
Right kidney is normal in size, shape and echogenicity and preserved CMCA.
No stone/mass/cyst/hydronephrosis seen.

LEFT KIDNEY: 12.8 x 6.3 x 3.5 cm. V: 153.79 cm³
Left kidney is normal in size, shape and echogenicity and preserved CMCA.
No stone/mass/cyst/hydronephrosis seen.

URINARY BLADDER: Empty.
Normal.

GENERAL ABDOMEN: No free fluid collection seen in abdomen and pelvis. No para-aortic lymphadenopathy seen.

IMPRESSION:
Normal abdominal scan.
- Fatty liver
- Right kidney shows increased echogenicity.

Reported By: _____

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Dr. Nadeem Ahmed FRCR
Assistant Professor
Dr. Arshad Nadeem MBBS
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Patient # _____
SS2
Registration # _____
SS20

Patient's Name: SULTAN
Referred by: SELF
Sample: BLOOD
City: Faisalabad

Age/Sex: 62 Years M
Date In: 18-Oct-2024
Date Out: 18-Oct-2024 3:20 pm
Contact # _____

REPORT PROFILE

Test Name	Result	Normal Value
R.F.T. (RENAL FUNCTION TESTS)		
SERUM CREATININE	2.9 mg/dl	0.5 - 1.2 mg/dl
$\frac{140 - 62 \times 68}{72 \times 2.9} = \frac{5304}{69.1}$ $eGFR = 76.7 \text{ mL/min}$		

Dr. Sadia Ijaz Bajwa
M.B.B.S., M.Phil.
(Haematology)

Electronically Generated Report, Does not need Signature

Note: The test performed with clinical care and best method, however overestimation is possible. The test should not be compared with other because different methodologies are used. This sample is taken without CBC confirmation. It has no official importance and there is no legal account.

HAEMATOLOGY CLINICAL PATHOLOGY ENDOCRINOLOGY VIROLOGY & MOLECULAR PATHOLOGY

Figure 4. 14, Figure 4. 15 **Renal Scans**

This patient's serum creatinine is elevated at 2.9 mg/dL with an eGFR of 76.7 mL/min, indicating reduced kidney function. Ultrasound shows a **fatty liver** and **increased echogenicity in the right kidney**, suggesting chronic kidney changes. Kidney sizes are within normal range with no stones or cysts seen. Overall, findings point to **early kidney dysfunction and fatty liver disease**.

Chapter 5

DISCUSSION

The increasing prevalence of obesity and its associated health complications have become a major global concern, with significant implications for renal health. Renal function is influenced by a multitude of factors, including demographic variables, body mass index (BMI), and gender. This study explores the relationship between these variables and sonographic markers of renal health, such as kidney size, serum Creatinine, and estimated glomerular filtration rate.

The analysis encompassed a diverse sample of 250 participants, representing varying BMI categories and gender distributions, to assess how these factors influence renal morphology and function. The study also investigates the prevalence of kidney size abnormalities, including changes in cortical echogenicity and parenchymal thickness, in relation to BMI and gender. By examining these variables, the research aims to elucidate patterns that may help predict and prevent renal dysfunction in at-risk populations.

Findings highlight significant associations between BMI and renal parameters, demonstrating that increased BMI correlates with altered kidney size, higher serum Creatinine levels, and reduced eGFR, particularly among female participants. Additionally, gender disparities in renal markers underscore the need for gender-specific approaches to assessing and managing renal health.

The current study analyzed the distribution of right kidney size abnormalities normal size, increased size due to cortical echogenicity, and decreased size due to parenchymal thickness across different BMI categories and genders. This section compares the findings with previous research and discusses the implications.

Comparison with Previous Studies The majority of participants in this study exhibited normal kidney sizes across BMI categories. However, a trend toward increased cortical echogenicity and decreased parenchymal thickness was noted in higher BMI groups. For

instance, among participants with a BMI >30 kg/m², 2.8% showed increased cortical echogenicity, and 1.6% exhibited decreased parenchymal thickness (44).

In this study, participants with a BMI <18.5 kg/m² had lower rates of kidney size abnormalities, with only 0.4% showing increased cortical echogenicity and 1.2% exhibiting decreased parenchymal thickness. These findings align with research by **Rena et al. (2014)**, which suggested that underweight individuals are less prone to renal structural abnormalities compared to obese populations. However, malnutrition in underweight groups could still lead to reduced parenchymal thickness due to chronic energy deficiency (45).

The study revealed gender differences, with females having a higher prevalence of normal kidney size (58.4%) compared to males (27.2%). Increased cortical echogenicity was more common in males (5.6%) than females (4.8%). Similar findings were reported by **Pinhas et al. (2018)**, who noted that males are more likely to exhibit kidney abnormalities, possibly due to higher rates of hypertension and diabetes. Conversely, females may be more protected from kidney abnormalities due to hormonal factors, particularly estrogen, which may have renoprotective effect (46).

The findings of this study indicate significant associations between BMI categories and kidney size alterations, including normal kidney size, increased cortical echogenicity, and decreased parenchymal thickness. These results corroborate previous literature exploring the relationship between BMI, kidney morphology, and potential renal dysfunction.

Several studies have demonstrated a correlation between elevated BMI and renal structural changes. For instance, **Azizi et al. (2024)** highlighted that obesity-related glomerulopathy is often associated with increased cortical echogenicity, reflecting early nephron damage due to heightened metabolic and hemodynamic stress.(47) Similarly, our results reveal a progressive increase in cortical echogenicity with increasing BMI, consistent with findings by **Poudel et al. (2023)**, who noted glomerular hypertrophy and compromised renal function among overweight and obese individuals (48).

Interestingly, participants with normal BMI (18.5–24.99 kg/m²) exhibited the highest percentage of normal kidney size (38.8%), whereas those in the >30 kg/m² BMI group showed higher proportions of increased cortical echogenicity (2.8%) and decreased parenchymal thickness (1.6%). This is in line with **Branch et al. (2019)**, who suggested that obesity leads to renal structural adaptations, including reduced parenchymal thickness, due to chronic inflammatory and fibrotic processes (49).

Increased cortical echogenicity often signifies early renal injury or fibrosis, as described. In contrast, decreased parenchymal thickness has been associated with chronic kidney disease progression, as noted by **Aziz et al. (2024)**. These structural changes may serve as early indicators of renal impairment, particularly in individuals with elevated BMI (50).

CONCLUSION

Sonographic variables have demonstrated great promise in the early identification of renal impairment in persons with diabetes. Before symptoms manifest or laboratory data change dramatically, early alterations in kidney size, cortical echogenicity, and resistive indices can be identified using imaging techniques such as Doppler ultrasonography and greyscale ultrasound.

The strength of the conclusion regarding the utility of sonographic variables in the early detection of renal impairment in diabetic individuals is considerable. Multiple studies have consistently demonstrated that imaging techniques such as Doppler ultrasonography and greyscale ultrasound can detect subtle anatomical and functional changes—such as alterations in kidney size, increased cortical echogenicity, and abnormal resistive indices—well before clinical symptoms emerge or laboratory values, like serum creatinine, become abnormal. This early detection capacity enhances clinical decision-making by allowing for the timely initiation of therapeutic interventions aimed at slowing disease progression. Furthermore, sonography is non-invasive, cost-effective, and widely accessible, making it a practical tool for large-scale screening and longitudinal monitoring. These attributes collectively reinforce the reliability and clinical relevance of sonographic assessment in the early identification and management of diabetic kidney disease.

Limitations

- The skill and knowledge of the operator greatly influence the ultrasound's outcomes, which can vary.
- Diabetes nephropathy is not the only condition that can exhibit certain sonographic indicators, such as increased echogenicity.
- Early changes may be subtle and difficult to detect particularly in the early stages of renal dysfunction.
- There is no universally accepted protocol for assessing sonographic markers in diabetic patients.
- While ultrasound is generally cost-effective access to advanced sonographic techniques like Doppler imaging may be limited in resource-constrained settings.
- Many studies on the use of sonographic markers for this purpose have small sample sizes, reducing the generalizability and reliability of findings.

Recommendations

- Develop and implement standardized guidelines for the use of sonographic markers in detecting renal dysfunction in diabetic adults to ensure consistency in assessment and interpretation.
- Include regular sonographic evaluations in the routine monitoring of diabetic patients to facilitate early detection and timely interventions.
- Provide specialized training for sonographers and healthcare professionals to improve accuracy and minimize operator dependency in sonographic evaluations.
- Encourage the use of advanced sonographic modalities such as Doppler ultrasonography, to assess renal blood flow and resistive indices for a more detailed evaluation of kidney function.
- Raise awareness among healthcare providers and diabetic patients about the importance of early detection of renal dysfunction using sonographic markers.

Clinical and Public Health Implications

- Enables early therapeutic strategies (glycemic control, ACE inhibitors).
- Sonography is cheaper and more accessible than serum creatinine or biopsy.
- Can be used in mass screening in primary care or community health setups.
- Useful in monitoring disease progression and response to treatment in diabetics.

Policy Implications

- Recommend routine renal ultrasound screening for diabetics >5 years duration or with poor control.
- Incorporate sonography into national diabetes screening programs.
- Train general practitioners and sonographers to identify early markers.
- Equip rural and district hospitals with ultrasound machines.
- Allocate funding for preventive nephrology programs.
- Encourage longitudinal studies on sonographic progression in diabetic nephropathy.
- Create national databases to monitor outcomes and policy efficacy.

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APPENDICES

ENGLISH CONSENT FORM

“Sonographic Markers In The Early Detection Of Renal Dysfunction In Diabetic Adults”

The study has no potential harm to participants. All data collected from you will be coded in order to protect your identity, and should not be disclosed to anyone. Following the study there will be no way to connect your name with your data. Your answers to the questions will not affect the quality of education given to you. Any additional information about the study results will be provided to you at its conclusion, upon your request.

You are free to withdraw from the study at any time. You agree to participate, indicating that you have read and understood the nature of the study, and that all your inquiries concerning the activities have been answered to your satisfaction.

NAME _____ **SIGNATURE** _____

DATE _____

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مینن ائی ب ہ ع م ا ن و ا ئ ل _____ تصدیق کرتا/ کرتی ہوں کہ محترمہ نے اپنی اس تحقیق

"ذیابیطس کے بالغوں میں گردوں کی خرابی کا ابتدائی پتہ لگانے میں سونوگرافک مارکر"

زیرنگرانی **صادیہ صابر** کے متعلق بتا دیا ہے۔ مجھے اس تحقیق کی نوعیت، مقاصد، احداث، توقعات، فوائد اور خطرات کے متعلق ، ساری معلومات فراہم کر دی گئی ہیں اس تحقیق کے دوران ساری معلومات صیغہ راز میں رہیں گی اور مریض کا نام اور دیگر معلومات صرف تحقیق کے لیے استعمال ہوں گی۔ مجھے یہ بھی بتا دیا گیا ہے کہ میں اس تحقیق سے متعلق ہر

قسم کے سوال پوچھنے کا مجاز ہوں اور یہ تحقیق صرف ایک شخص کا مفاد میں نہیں ہے بلکہ بحسب مجموعی انسانیت کا مفاد اس سے واسطہ ہے۔ تمام تفصیلات جاننے کے بعد میں تحقیق میں شامل ہونے یا نہ ہونے پر کسی کا قائل نہیں ہوں۔ اس تحقیق سے کسی بھی وقت علیحدہ ہونے پر مجھ پر کوئی پابندی نہیں ہو گی۔ میں بنا پت خود بقائی حوش و حواس اور رضا مندی سے اس تحقیقاتی عمل میں شامل ہوتی/ ہوتا ہوں

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Date: 29th October 2024

Name: Naiba Emmanuel (MS Allied Health Sciences)

Registration: SU91-MAHSW-S23-070

Subject: Ethical Approval Letter

The Research Ethical Committee convened on Dated: **17th October, 2024** to discuss your protocol titled **"Sonographic markers in the early detection of renal dysfunction in diabetic adults"**

No further corrections and recommendations were suggested. The above-mentioned protocol has been approved after considering various research issues including ethical concerns with condition that the researcher will submit completion report at the end of his/her research.

Prof. Dr. Muhammad Naveed Babur
Dean/Convener REC
Faculty of Allied Health Sciences
Superior University, Lahore

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