“SHEAR WAVE ELASTOGRAPHY IN EVALUVATION OF RENAL PARENCHYMAL STIFFNESS IN PATIENT WITH CHRONIC KIDNEY DISEASE: A HOSPITAL BASED ONE YEAR CROSS SECTIONAL STUDY”

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Sub: Institutional Ethical Clearance for the study.

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ABBREVIATIONS

SL.NO	TYPES OF ABBREVIATION	
1	Shear Wave Elastography	(SWE)
2	Chronic kidney disease	(CKD)
3	Glomerular filtration rate	(GFR)
4	Antidiuretic hormone	(ADH)
5	End-stage renal disease	(ESRD)
6	Acute kidney injury	(AKI)
7	Renin-angiotensin-aldosterone system	(RAAS)
8	Extracellular matrix	(ECM)
9	Transforming growth factor-beta	(TGF-β)
10	Vascular endothelial growth factor	(VEGF)
11	Fibroblast growth factor	(FGF)
12	Nuclear factor kappa B	(NF-Kb)
13	Low-density lipoprotein	(LDL)
14	Matrix metalloproteinases	(MMPs)
15	Tissue inhibitors of metalloproteinases	(TIMPs)
16	Epithelial-to-mesenchymal transition	(EMT)
17	Reactive oxygen species	(ROS)
18	Magnetic resonance imaging	(MRI)
19	Magnetic resonance angiography	(MRA)
20	Nephrogenic systemic fibrosis	(NSF)

ABSTRACT

Introduction

Chronic Kidney Disease (CKD) is a progressive condition leading to renal fibrosis and eventual kidney failure. Traditional imaging techniques, such as ultrasonography, are limited in quantifying renal fibrosis, while renal biopsy, the gold standard, is invasive. Shear Wave Elastography (SWE) is an emerging non-invasive technique that assesses renal stiffness and may serve as a valuable tool for evaluating fibrosis in CKD. This study aims to assess the utility of SWE in CKD patients and compare elastography findings with traditional ultrasonographic parameters.

Objectives:

Primary Objective:

- To assess elastography values in patients with chronic kidney disease.

Secondary Objectives:

- To compare elastography parameters with ultrasonographic parameters in CKD patients.

Methods:

This was a hospital-based, cross-sectional study conducted over one year at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. A total of 96 CKD patients aged 18 years and above were enrolled using a universal random sampling technique. SWE was performed to measure renal stiffness in kilopascals (kPa). Traditional ultrasonographic parameters such as renal length, cortical thickness,

and echogenicity were also recorded. Correlations between SWE measurements and renal function markers (serum creatinine, eGFR) were analyzed.

Results:

- CKD stage distribution: Stage 1 (10.4%), Stage 2 (20.8%), Stage 3 (36.5%), Stage 4 (20.8%), Stage 5 (11.5%).
- Renal stiffness increased with disease severity (Stage 1: 15.2 ± 2.1 kPa to Stage 5: 32.4 ± 4.0 kPa).
- Strong correlations observed: SWE vs. eGFR ($r = -0.68$, $p < 0.001$); SWE vs. serum creatinine ($r = 0.72$, $p < 0.001$).
- Diabetic and hypertensive patients had significantly higher renal stiffness values.
- ROC analysis determined a stiffness cut-off of 28.0 kPa for severe CKD (AUC = 0.88, sensitivity = 85%, specificity = 80%).
- SWE demonstrated high reproducibility (intra-observer ICC = 0.92, inter-observer ICC = 0.89).
- No significant difference in stiffness between left and right kidneys ($p = 0.45$).

Conclusion:

Shear Wave Elastography (SWE) is a reliable, non-invasive tool for assessing renal fibrosis in CKD, showing strong correlations with renal function markers. SWE effectively differentiates CKD stages and provides an objective assessment of disease severity. Given its high diagnostic accuracy and reproducibility, SWE has the

potential to complement conventional ultrasonography in CKD evaluation and may reduce the need for invasive biopsies. Future studies should explore its long-term utility in disease monitoring and treatment response assessment.

Keywords: Chronic Kidney Disease (CKD), Shear Wave Elastography (SWE), Renal Fibrosis, Renal Stiffness, Ultrasound Elastography, Kidney Function, Serum Creatinine, Estimated Glomerular Filtration Rate (eGFR), Non-Invasive Imaging, Renal Ultrasound.

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INTRODUCTION

Chronic Kidney Disease (CKD) is a progressive condition characterized by a gradual loss of kidney function over time, which can ultimately lead to kidney failure. According to the World Health Organization (WHO), CKD is a significant cause of morbidity and mortality worldwide, with an increasing global prevalence. As the disease progresses, fibrosis and scarring of the renal parenchyma occur, leading to a decline in renal function. Early detection of renal fibrosis is essential for the timely management of CKD to slow disease progression and avoid complications such as end-stage renal disease. [1]

Traditional imaging techniques, including gray-scale ultrasonography, are widely used in the clinical evaluation of CKD. However, these imaging modalities can only detect structural abnormalities and are limited in their ability to quantify the degree of renal fibrosis. Structural changes such as kidney enlargement, cortical thinning, and loss of corticomedullary differentiation can be visible, but quantifying the stiffness or fibrosis of the renal parenchyma remains challenging. As a result, these imaging techniques are often not sufficient for assessing the severity of CKD or predicting the patient's prognosis. [2]

Percutaneous renal biopsy has long been considered the gold standard for evaluating renal fibrosis. It allows direct histological assessment of the kidney tissue, providing valuable information regarding the extent of fibrosis and the underlying causes of CKD. However, biopsy is an invasive procedure that carries risks such as bleeding, infection, and pain. It also requires expertise to interpret the results accurately and is not always feasible for routine clinical practice, particularly in patients with comorbidities or those on anticoagulant therapy. Therefore, there is a pressing need for a noninvasive and reliable method to assess renal fibrosis and monitor disease progression. [3]

Shear Wave Elastography (SWE) is a promising noninvasive imaging modality that can objectively assess tissue stiffness by measuring the velocity of shear waves as they propagate through tissues. The shear wave velocity is directly proportional to tissue stiffness, making it an ideal tool for evaluating fibrosis in organs such as the liver and kidney. SWE is a low-cost, fast, and real-time ultrasound-based technique, which allows for the assessment of renal parenchymal stiffness. Compared to traditional ultrasound, SWE provides quantitative and reproducible data, making it an attractive option for clinical practice. [4]

Recent studies have demonstrated the potential of SWE in assessing renal stiffness in patients with CKD. The technique has shown promise in detecting early fibrosis in the kidney, correlating well with histopathological findings from renal biopsies. By quantifying the stiffness of the renal parenchyma, SWE could provide valuable information regarding the degree of fibrosis, which could aid in the diagnosis and management of CKD. Furthermore, SWE can be used to monitor treatment responses and track changes in renal stiffness over time, offering a noninvasive alternative to serial biopsies. [5]

Despite its potential, the clinical application of SWE in CKD remains an area of ongoing research. While several studies have investigated its feasibility and accuracy in assessing renal fibrosis, there is still a need for more extensive and well-designed studies to validate its role in clinical practice. Specifically, the correlation between SWE measurements and other diagnostic parameters, such as conventional ultrasound findings, has not been fully established. [6]

The main objective of this study is to assess elastography values in patients with CKD. By evaluating renal stiffness through SWE, this study aims to provide further evidence on the utility of SWE as a noninvasive method for assessing renal fibrosis in CKD. Additionally, this study will compare elastography parameters with ultrasonography parameters in patients with CKD to evaluate their relative effectiveness in diagnosing and monitoring CKD. [7]

The secondary objective of this study is to compare the SWE findings with conventional ultrasonography measurements, including kidney size, cortical thickness, and echogenicity. While ultrasonography is widely used in the clinical evaluation of CKD, it lacks the ability to quantify fibrosis directly. [8] By comparing these two techniques, this study will help to assess the potential advantages of SWE over conventional ultrasound in the evaluation of renal fibrosis. [9]

This study will also evaluate the role of SWE in monitoring the progression of CKD and assessing the efficacy of treatment interventions. By providing a reliable, noninvasive tool for evaluating renal stiffness, SWE could help in tailoring treatment strategies, allowing clinicians to identify patients at higher risk of progression and intervene early [10]. The introduction of Shear Wave Elastography (SWE) in the evaluation of renal stiffness represents a promising advancement in the noninvasive assessment of CKD. This study aims to provide further insights into its diagnostic and prognostic value, contributing to the growing body of evidence supporting SWE as a clinical tool in the management of CKD.

AIMS AND OBJECTIVES

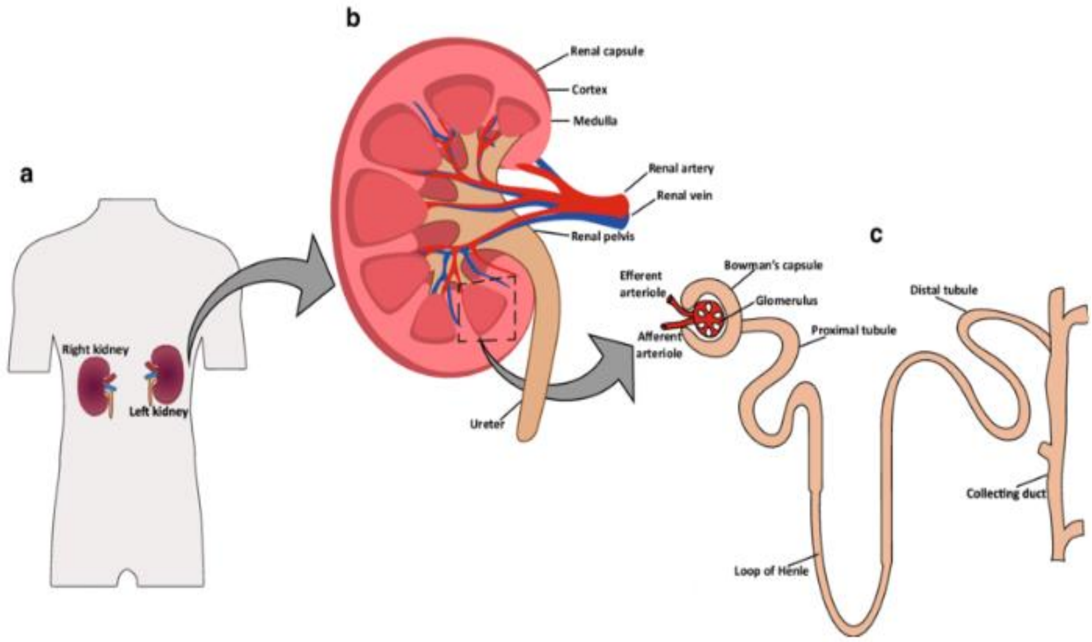
PRIMARY: To asses elastography values in patients with chronic kidney disease.

SECONDARY: To compare elastography parameters and ultrasonography parameters inpatients with chronic kidney disease.

REVIEW OF LITERATURE

2.1 Anatomy and Physiology of the Kidney in Health and Disease

The kidneys are vital organs located in the posterior abdomen, functioning to filter blood, remove waste, regulate fluid and electrolyte balance, and maintain homeostasis. They are paired, bean-shaped structures, each approximately 10–12 cm in length, 5–7 cm in width, and 3 cm in thickness. The kidneys lie retroperitoneally, with the left kidney typically positioned slightly higher than the right due to the presence of the liver on the right side. The kidneys are situated between the T12 and L3 vertebrae, protected by the lower ribs. [11]



Human kidney anatomy. External view (a), internal view (b), and its functional unit nephron (c)

Figure 1: Anatomy of the Human Kidney [11]

External Anatomy of the Kidney

The kidneys are encapsulated by a dense fibrous capsule that helps protect the underlying tissue. The outer layer of the kidney, known as the renal cortex, is a reddish-brown area that contains the renal corpuscles, proximal and distal convoluted tubules, and parts of the nephron responsible for filtration and reabsorption. Beneath the cortex lies the renal medulla, which consists of pyramidal structures called renal pyramids. The renal pyramids contain the loops of Henle, the collecting ducts, and other structures involved in the final stages of urine formation. [12]

Renal Pelvis and Calyces

At the center of the kidney lies the renal pelvis, a funnel-shaped structure that collects urine from the renal pyramids before it is transported to the ureter. The renal pelvis is subdivided into major and minor calyces. The minor calyces collect urine from the tips of the renal pyramids, while the major calyces join together to form the renal pelvis. These structures play a crucial role in the drainage system of the kidney. [13]

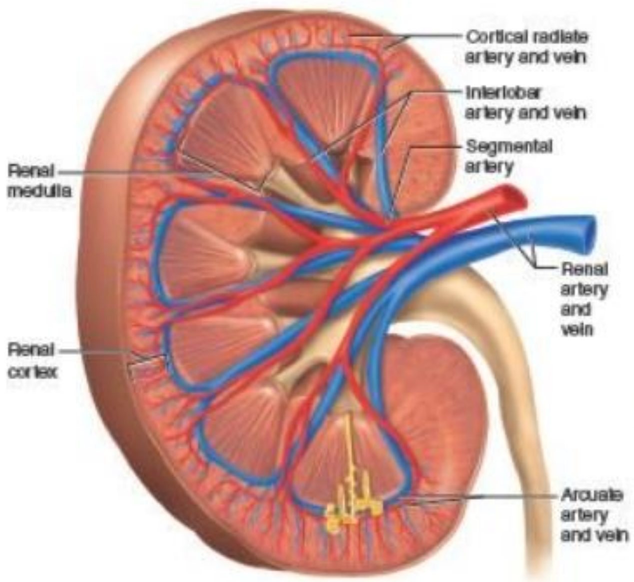


Figure 2: Blood circulation in human Kidney [14]

Blood Supply to the Kidney

The kidneys receive a significant blood supply, approximately 20% of the resting cardiac output, through the renal arteries. The renal arteries branch directly from the abdominal aorta and enter the kidneys at the hilum. Upon entering the kidney, the renal arteries divide into segmental arteries, which further branch into interlobar arteries that travel between the renal pyramids. These arteries then branch into arcuate arteries that run along the border between the renal cortex and medulla. The arcuate arteries give rise to the interlobular arteries, which supply blood to the nephrons. [14]

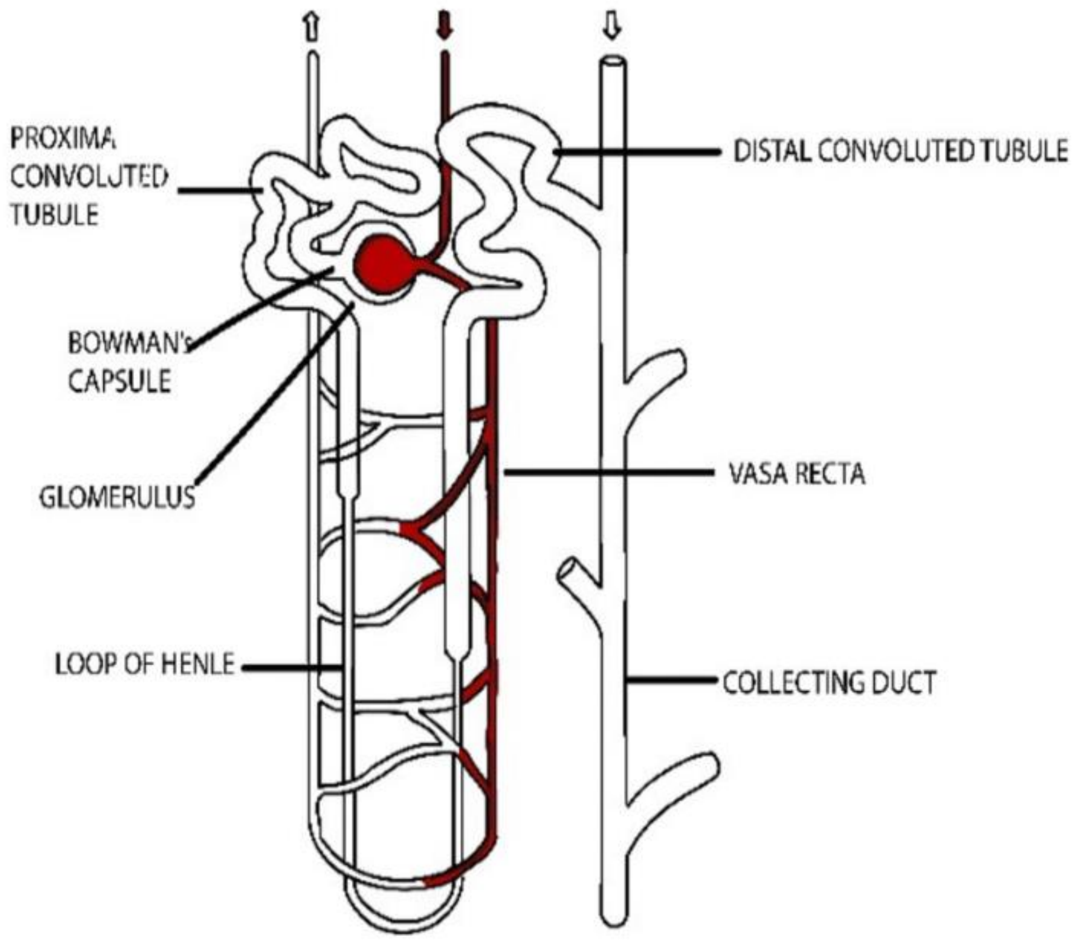


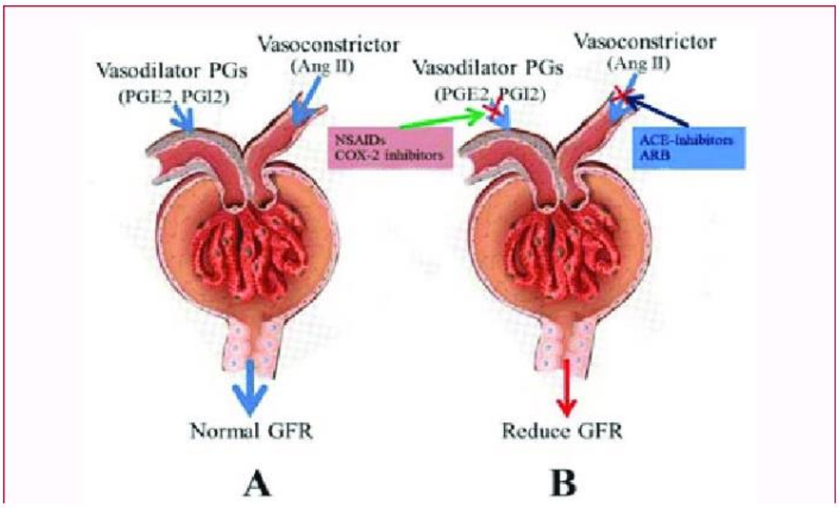
Figure 3: Internal structure of nephron [15]

Nephron Structure and Function

The nephron is the functional unit of the kidney, with approximately 1 million nephrons in each kidney. Each nephron consists of a renal corpuscle and a renal tubule. The renal corpuscle is composed of the glomerulus, a network of capillaries responsible for filtration, and the Bowman's capsule, which surrounds the glomerulus and collects the filtered fluid. The renal tubule, which includes the proximal convoluted tubule, the loop of Henle, the distal convoluted tubule, and the collecting duct, is involved in the reabsorption of essential substances and secretion of waste products into the filtrate, ultimately forming urine. [15]

Renal Circulation and Filtration

The renal circulation plays a vital role in maintaining kidney function. The afferent arteriole delivers blood to the glomerulus, where filtration of blood plasma occurs. The glomerular filtration rate (GFR) is a key determinant of kidney function and reflects the rate at which the kidneys filter blood. Filtrate produced in the glomerulus enters the proximal convoluted tubule, where essential nutrients, such as glucose, amino acids, and electrolytes, are reabsorbed back into the bloodstream. [16]



Regulation of renal blood flow & glomerular filtration in the kidney. PGE2 and PGI2 play an increasingly important role in maintaining renal perfusion by causing enhanced pre-glomerular vasodilation and angiotensin

Figure 4: Regulation of renal blood flow & glomerular filtration in the kidney [16]

Tubular Reabsorption and Secretion

The proximal convoluted tubule is the site of significant reabsorption of water, ions, and organic solutes from the filtrate into the peritubular capillaries. The loop of Henle is responsible for the establishment of the medullary concentration gradient, which allows for the reabsorption of water and sodium. The distal convoluted tubule and collecting ducts regulate the final composition of urine, with aldosterone and antidiuretic hormone (ADH) playing crucial roles in sodium retention and water reabsorption. [17]

Kidney's Role in Acid-Base Balance

The kidneys help maintain the body's acid-base balance by excreting hydrogen ions and reabsorbing bicarbonate from the filtrate. The distal convoluted tubule and collecting duct are responsible for the final regulation of acid-base balance. The kidneys also produce ammonia, which buffers excess hydrogen ions, further assisting in maintaining the pH of the blood within a narrow range of 7.35–7.45. [18]

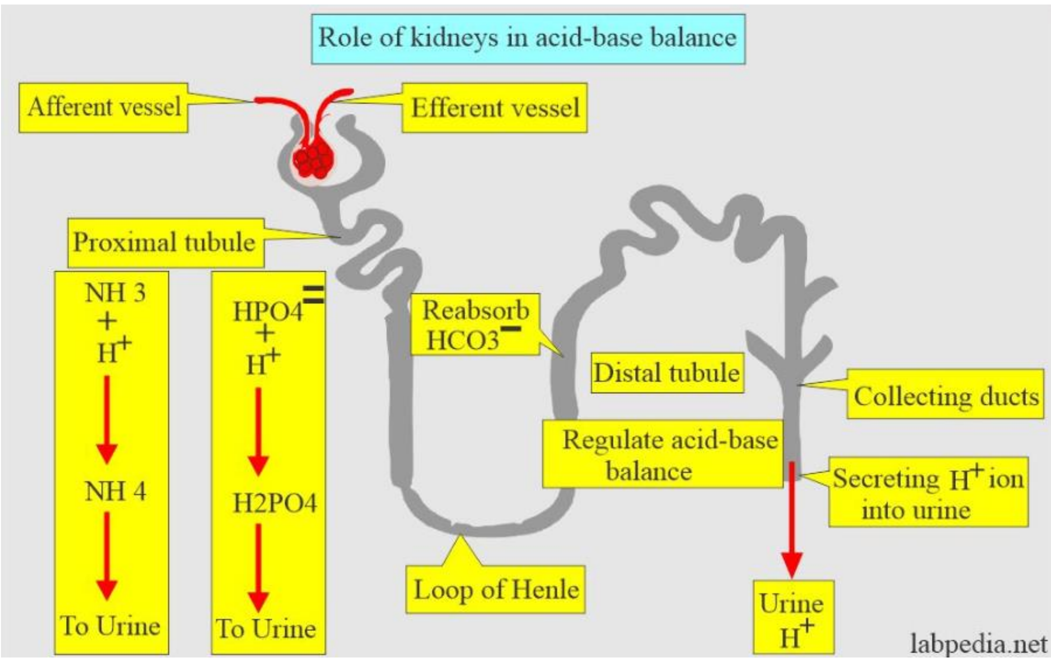
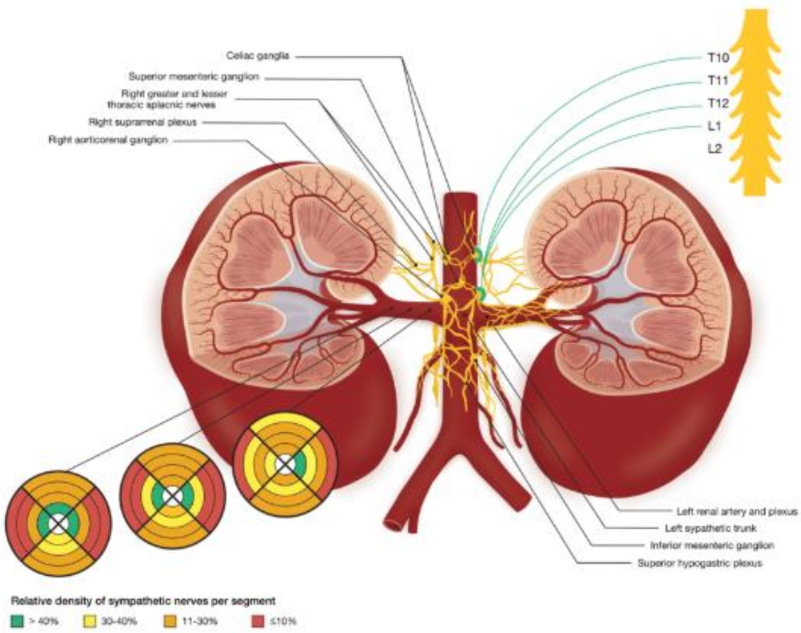


Figure 5: Acid-base balance and role of kidneys

Nerve Supply to the Kidneys

The kidneys are innervated by both sympathetic and parasympathetic fibers. The sympathetic innervation, originating from the T10 to L2 spinal segments, primarily regulates blood flow to the kidneys by constricting afferent arterioles through the release of norepinephrine. This helps modulate the glomerular filtration rate (GFR) and renal blood flow in response to stress or changes in blood pressure. The parasympathetic innervation, originating from the vagus nerve, plays a less prominent role but is involved in modulating renal function under normal physiological conditions. [19]



Overview of kidney innervation. The kidneys are densely innervated by both afferent nociceptive sensory fibers and efferent sympathetic nerve fibers. Their distribution along the renal artery makes them accessible to therapeutic targeting by endoluminal catheter-based denervation approaches for kidney-related pain syndromes. The green lines represent the sympathetic component

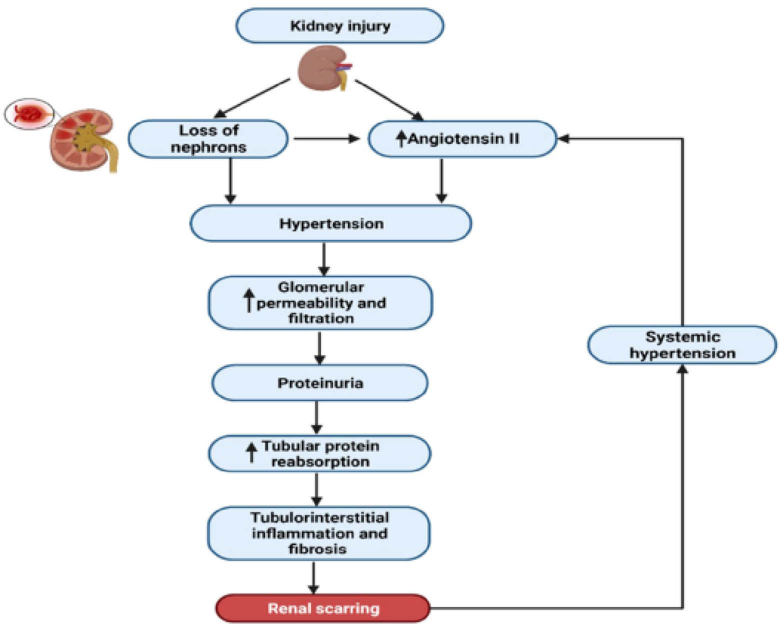
Figure 6: Overview of kidney innervations [19]

Renal Function and Hormonal Regulation

The kidneys also function as endocrine organs, producing hormones that regulate various physiological processes. One of the key hormones produced by the kidneys is erythropoietin, which stimulates red blood cell production in the bone marrow in response to hypoxia. Additionally, the kidneys activate vitamin D to its active form (calcitriol), which helps regulate calcium and phosphate metabolism. The renin-angiotensin-aldosterone system (RAAS) is another critical pathway regulated by the kidneys, controlling blood pressure and fluid balance through the release of renin, which ultimately leads to the production of aldosterone and vasoconstriction. [20]

2.2 Pathophysiology of Chronic Kidney Disease (CKD)

Chronic Kidney Disease (CKD) is a progressive condition characterized by the gradual loss of kidney function over time. It is defined as the presence of kidney damage (e.g., albuminuria) or a glomerular filtration rate (GFR) of less than 60 mL/min/1.73 m² for three months or more, irrespective of the cause. CKD represents a major public health issue due to its high prevalence, potential for progression to end-stage renal disease (ESRD), and its associated comorbidities, including cardiovascular disease and diabetes. The pathophysiology of CKD is complex and involves a cascade of events that lead to kidney dysfunction, fibrosis, and eventual failure. [21]



Pathophysiology of chronic kidney disease.[52] Kidney injury results in loss of nephrons and increased levels of angiotensin II. Angiotensin II is a vasoconstrictor that triggers increased blood pressure. Alongside hypertension, there is an increase in glomerular permeability, tubular protein reabsorption, tubular/interstitial inflammation, and eventually renal scarring.

Figure 7: Pathophysiology of chronic kidney disease [21]

At the core of CKD pathophysiology is the process of renal fibrosis, a dynamic response to injury within the kidney. Renal fibrosis involves excessive accumulation of extracellular matrix (ECM) components, including collagen and fibronectin, which leads to scarring and loss of kidney structure. This process is initiated in response to injury from various etiologies such as hypertension, diabetes, glomerulonephritis, and other systemic diseases. These injuries activate both cellular and molecular pathways that result in the recruitment of inflammatory cells, activation of fibroblasts, and deposition of ECM. The hallmark of kidney fibrosis is the progressive destruction of the normal architecture of the renal parenchyma, with the formation of interstitial fibrosis and glomerulosclerosis. [22]

The glomerulus is a key structure in the kidney that filters blood to produce urine. In CKD, glomerular damage plays a significant role in the progression of the disease. Early stages of CKD often involve hyperfiltration, a compensatory mechanism in which the remaining healthy nephrons undergo increased workload to compensate for the loss of nephrons. While this hyperfiltration initially helps maintain kidney function, it eventually leads to glomerular hypertrophy, increased intraglomerular pressure, and glomerulosclerosis, which further exacerbates kidney damage. This increase in pressure within the glomerulus leads to endothelial injury and causes increased permeability, which allows proteins, particularly albumin, to leak into the urine, a condition known as albuminuria or proteinuria. Albuminuria is a key marker of kidney damage and is associated with an increased risk of disease progression. [23]

The renal tubules, which are responsible for reabsorbing most of the filtered substances from the glomerulus, are also significantly affected in CKD. Tubular damage is often caused by ischemia, toxins, or inflammation, leading to tubular cell injury and apoptosis. In response to this injury, tubular cells undergo dedifferentiation and secrete pro-inflammatory cytokines that recruit additional inflammatory cells to the site of injury. These cytokines and growth factors such as transforming growth factor-beta (TGF-β), vascular endothelial growth factor (VEGF), and fibroblast growth factor (FGF) activate fibroblasts and promote the deposition of ECM, contributing to fibrosis. Over time, the injury and subsequent repair responses result in the loss of tubular function and further contribute to the decline in GFR. [24]

Inflammation is a central feature of CKD and contributes to both the initiation and progression of kidney damage. The inflammatory response in CKD is triggered by various factors, including glomerular injury, ischemia, and oxidative stress. This inflammatory process leads to the activation of resident immune cells, such as macrophages and dendritic cells, which release pro-inflammatory cytokines and chemokines. These inflammatory mediators recruit additional leukocytes, which perpetuate the cycle of injury and inflammation. The persistent inflammation contributes to the development of fibrosis and the deterioration of kidney function. Inflammation also enhances endothelial dysfunction, which further increases vascular permeability and promotes the leakage of proteins into the urine. [25]

The role of the renin-angiotensin-aldosterone system (RAAS) in CKD pathophysiology is well-documented. RAAS activation occurs in response to reduced renal perfusion, a common feature in CKD, especially in conditions such as diabetic nephropathy and hypertension. RAAS activation leads to the production of angiotensin II, a potent vasoconstrictor, which increases glomerular pressure and contributes to glomerulosclerosis. Angiotensin II also stimulates the secretion of aldosterone, which promotes sodium retention and increases blood volume, exacerbating hypertension. The prolonged activation of RAAS in CKD leads to further kidney injury, fibrosis, and progression of the disease. [26]

Another key player in CKD pathophysiology is endothelial dysfunction, which is prevalent in both the glomerular and systemic vasculature. In healthy kidneys, the endothelium plays an essential role in maintaining vascular homeostasis by regulating vasodilation, coagulation, and inflammatory responses. In CKD, however, endothelial cells become activated due to oxidative stress, inflammation, and the effects of increased blood pressure. This activation results in the release of pro-inflammatory cytokines, adhesion molecules, and reactive oxygen species (ROS), all of which contribute to vascular injury and the development of fibrosis. The impaired endothelial function in the kidneys also disrupts the regulation of glomerular filtration, contributing to further kidney dysfunction. [27]

Oxidative stress is another critical factor in CKD. The kidney is highly susceptible to oxidative damage due to its high oxygen consumption and rich blood supply. In CKD, increased oxidative stress results from the accumulation of ROS, which damage cellular components such as lipids, proteins, and DNA. This oxidative damage activates a variety of signaling pathways, including those involved in inflammation, fibrosis, and cell death. In particular, oxidative stress enhances the activation of nuclear factor kappa B (NF-κB), a key transcription factor involved in the inflammatory response. This activation perpetuates the cycle of injury and fibrosis in the kidney. [28]

The progression of CKD is also influenced by metabolic factors such as dyslipidemia and insulin resistance, which are common in individuals with diabetes and obesity. Dyslipidemia, characterized by elevated levels of low-density lipoprotein (LDL) cholesterol and triglycerides, contributes to endothelial dysfunction and increases the risk of atherosclerosis. The accumulation of lipid deposits in the kidney can further exacerbate renal injury. Insulin resistance, which often accompanies metabolic syndrome, is also a major contributor to the pathogenesis of CKD. Insulin resistance impairs the normal function of various renal cells, including endothelial cells and fibroblasts, promoting inflammation and fibrosis. [29]

In addition to the structural changes in the kidney, CKD leads to a variety of systemic complications. One of the most significant consequences is the development of hypertension, which is both a cause and a consequence of kidney damage. As kidney function declines, the kidneys lose their ability to regulate sodium and fluid balance, leading to increased blood volume and elevated blood pressure. In turn, hypertension accelerates the progression of kidney injury by increasing glomerular pressure, contributing to glomerulosclerosis and fibrosis. Furthermore, the activation of the RAAS system and endothelial dysfunction in CKD both contribute to the maintenance of high blood pressure. [30]

2.3 Role of Renal Fibrosis in CKD Progression

Renal fibrosis is a central pathological process in the progression of Chronic Kidney Disease (CKD). It involves the excessive accumulation of extracellular matrix (ECM) proteins, particularly collagen, in the renal interstitial spaces, and is a key contributor to the functional decline observed in CKD. Fibrosis represents the end result of various kidney injuries, including inflammation, ischemia, and glomerular hypertension, and is characterized by the transformation of normal kidney tissue into scar tissue. This fibrotic remodeling leads to a loss of nephron function, which ultimately causes a decline in glomerular filtration rate (GFR) and the progression of CKD toward end-stage renal disease (ESRD). [31]

At the molecular level, renal fibrosis is driven by complex signaling pathways that involve a variety of cellular and molecular mediators. One of the most important mediators in renal fibrosis is transforming growth factor-beta (TGF-β), which is widely regarded as the key profibrotic cytokine in kidney injury. TGF-β is upregulated in response to various insults, including hyperglycemia, hypertension, and hypoxia, and it induces the activation of renal fibroblasts and myofibroblasts. These activated cells are responsible for the excessive deposition of ECM components, leading to fibrosis. TGF-β also modulates the expression of matrix metalloproteinases (MMPs) and tissue inhibitors of metalloproteinases (TIMPs), which further regulate ECM turnover and contribute to the progressive accumulation of fibrosis. [32]

The progression of renal fibrosis is a dynamic and multifactorial process involving several key cell types, including fibroblasts, myofibroblasts, endothelial cells, and tubular epithelial cells. Fibroblasts and myofibroblasts play a central role in the production and deposition of ECM, particularly collagen. These cells are activated in response to injury signals such as TGF-β, angiotensin II, and mechanical stress. Myofibroblasts, which are differentiated fibroblasts that acquire smooth muscle actin expression, are particularly important in the synthesis of ECM proteins and the contraction of the fibrotic tissue. Tubular epithelial cells, when injured, can undergo epithelial-to-mesenchymal transition (EMT), a process by which these cells acquire fibroblast-like properties, contributing to the pool of myofibroblasts and further exacerbating the fibrotic response. [33]

Renal fibrosis is closely linked to glomerulosclerosis, a process in which the glomeruli become scarred and non-functional. In CKD, hyperfiltration due to the loss of nephrons causes glomerular hypertension and further injury to the remaining functional glomeruli. This increased pressure within the glomerulus induces endothelial cell dysfunction, podocyte injury, and mesangial cell proliferation. The injury leads to the accumulation of ECM in the glomerulus, which progressively obstructs the filtration process. This glomerulosclerosis is often accompanied by tubulointerstitial fibrosis, where the renal interstitial tissue becomes infiltrated with inflammatory cells and fibrotic material. The progressive combination of glomerulosclerosis and tubulointerstitial fibrosis results in a loss of renal architecture and function. [34]

The kidney has a remarkable capacity for compensatory hypertrophy in the early stages of CKD. When a portion of the nephrons is lost due to injury or disease, the remaining nephrons undergo hyperfiltration to compensate for the loss of function. However, this compensatory mechanism can have detrimental effects over time. Prolonged hyperfiltration increases the pressure within the glomeruli, leading to endothelial injury and the activation of the fibrotic process. Additionally, the increased workload on the remaining nephrons leads to nephron hypertrophy and glomerular enlargement, which ultimately accelerates the progression of fibrosis. The hyperfiltration state is often accompanied by proteinuria, which is another key marker of kidney damage and an important contributor to the fibrotic response. [35]

Oxidative stress is another critical factor that contributes to renal fibrosis in CKD. The kidney is highly susceptible to oxidative damage due to its high metabolic activity and exposure to toxins. In CKD, oxidative stress is exacerbated by various factors, including inflammation, hypertension, and ischemia. The increased production of reactive oxygen species (ROS) leads to damage of cellular components, including lipids, proteins, and DNA. This oxidative damage activates several pro-fibrotic pathways, including the upregulation of TGF-β, the activation of NF-κB, and the recruitment of inflammatory cells. Furthermore, ROS can directly affect renal fibroblasts, increasing their production of ECM proteins and promoting fibrosis. [36]

Inflammation plays a key role in the development and progression of renal fibrosis. Chronic inflammation is a common feature of CKD and contributes to the activation of renal fibroblasts and the production of ECM. Inflammatory cells, including macrophages, T lymphocytes, and neutrophils, infiltrate the kidney in response to injury. These cells release a variety of pro-inflammatory cytokines, chemokines, and growth factors, such as tumor necrosis factor-alpha (TNF-α), interleukins (IL-1, IL-6), and VEGF, which further promote fibrosis. The inflammatory response also enhances the production of ROS, which amplifies the cycle of injury and fibrosis. Inflammatory mediators also stimulate endothelial dysfunction and increase vascular permeability, leading to further kidney damage. [37]

The renin-angiotensin-aldosterone system (RAAS) is another important regulator of renal fibrosis. Activation of RAAS occurs in response to decreased renal perfusion or kidney injury. Angiotensin II, the main effector molecule of RAAS, promotes fibrosis through its effects on TGF-β and other profibrotic pathways. Angiotensin II also induces the contraction of myofibroblasts and stimulates the production of ECM components, thereby contributing to fibrosis. In addition to its effects on fibroblasts, angiotensin II also promotes inflammation by activating macrophages and inducing the secretion of pro-inflammatory cytokines. Furthermore, aldosterone, another key component of RAAS, exacerbates fibrosis by increasing sodium retention and promoting vascular remodeling. [38]

The role of the immune system in renal fibrosis is becoming increasingly recognized. The immune response in CKD is characterized by both innate and adaptive immune activation. In the early stages of kidney injury, the innate immune system is activated, leading to the recruitment of neutrophils, macrophages, and dendritic cells. These cells release pro-inflammatory cytokines and ROS, which contribute to tissue damage and fibrosis. As CKD progresses, the adaptive immune system becomes involved, with the activation of T cells and the production of antibodies. The immune system plays a dual role in renal fibrosis, both contributing to tissue damage through inflammation and promoting tissue repair through the release of growth factors and cytokines. [39]

The progression of renal fibrosis is associated with a number of molecular markers that are used to assess the severity of kidney damage. These markers include collagen deposition, the expression of ECM proteins such as fibronectin and laminin, and the activation of fibroblasts and myofibroblasts. The measurement of urinary biomarkers such as TGF-β1, urinary matrix metalloproteinases (MMPs), and tissue inhibitor of metalloproteinases (TIMPs) has become increasingly important in assessing the degree of fibrosis and monitoring disease progression. Non-invasive imaging techniques, such as elastography and magnetic resonance imaging (MRI), are also being explored to assess fibrosis and monitor treatment response. [40]

2.4 Conventional Imaging Techniques in Renal Evaluation

Imaging plays a crucial role in the evaluation of renal health and disease, providing valuable information on the structure, function, and pathology of the kidneys. Conventional imaging techniques, including ultrasonography, computed tomography (CT), and magnetic resonance imaging (MRI), are widely used in clinical practice to diagnose and monitor renal conditions. These modalities help assess kidney size, morphology, blood flow, and the presence of any abnormalities such as tumors, stones, or cysts. Each imaging modality has distinct advantages and limitations, and the choice of technique often depends on the clinical scenario, patient condition, and available resources. [41]

Ultrasonography is the most commonly used imaging technique for renal evaluation due to its accessibility, low cost, and lack of ionizing radiation. It is particularly useful in assessing kidney size, cortical echogenicity, and the presence of structural abnormalities such as cysts, hydronephrosis, and kidney stones. Ultrasound can also help detect changes in renal blood flow and guide the assessment of chronic kidney disease (CKD) progression. In the context of CKD, ultrasonography can reveal characteristic findings such as shrunken kidneys, increased cortical echogenicity, and loss of corticomedullary differentiation, which suggest fibrosis and renal parenchymal damage. Ultrasound is also instrumental in the detection of renal tumors, obstruction, and complications such as fluid collections or abscesses. [42]

One of the major limitations of conventional ultrasound in renal imaging is its inability to provide detailed information on the microarchitecture of the kidneys, such as fibrosis or inflammation, which are key features of CKD progression. While Doppler ultrasound can assess renal blood flow and detect vascular abnormalities, it is less effective in evaluating the degree of fibrosis or kidney function compared to newer, more advanced imaging techniques. Furthermore, the quality of ultrasound images can be affected by patient body habitus, bowel gas, and operator skill, making it a less reliable tool in some clinical settings. Despite these limitations, ultrasonography remains an essential first-line imaging modality in renal evaluation, especially in the acute setting. [43]

Computed tomography (CT) is a more advanced imaging technique that provides detailed cross-sectional images of the kidneys and surrounding structures. CT imaging is particularly valuable in assessing renal masses, such as tumors or cysts, and detecting complications such as kidney stones, abscesses, or infections. It can also evaluate the renal vasculature, providing detailed information on blood supply, including the identification of renal artery stenosis or aneurysms. In patients with suspected urological malignancies, CT is often used for staging, as it provides high-resolution images that can detect small tumors, enlarged lymph nodes, or distant metastasis. Additionally, CT angiography is frequently employed in the assessment of renal vascular conditions, offering valuable insight into the renal arteries and veins. [44]

One of the primary drawbacks of CT imaging is the use of ionizing radiation, which limits its repeated use, especially in young patients or those requiring long-term monitoring. The administration of intravenous contrast agents in CT also carries a risk of contrast-induced nephropathy, a condition in which renal function deteriorates following the administration of contrast material. This is particularly concerning in patients with pre-existing kidney disease or those at risk for acute kidney injury. Furthermore, while CT can provide detailed images of the kidneys and surrounding tissues, it lacks the ability to assess kidney function or fibrosis in a quantitative manner, limiting its role in the longitudinal monitoring of CKD. [45]

Magnetic resonance imaging (MRI) is a non-invasive imaging technique that offers high-resolution images of the kidneys without the need for ionizing radiation. MRI is particularly advantageous in patients with contraindications to iodinated contrast agents, such as those with kidney dysfunction or allergies to contrast material. It is widely used in the evaluation of complex renal masses, congenital abnormalities, and vascular conditions, such as renal artery stenosis or aneurysms. MRI also provides superior soft tissue contrast compared to CT, allowing for the assessment of renal parenchymal abnormalities, including fibrosis, inflammation, and scarring. Magnetic resonance angiography (MRA) is a useful tool for assessing the renal vasculature, including the detection of stenosis or other vascular anomalies. [46]

Despite its advantages, MRI has several limitations. It is more expensive and less widely available than ultrasonography and CT, and the procedure is more time-consuming. Additionally, MRI is contraindicated in patients with certain implanted medical devices, such as pacemakers or certain types of metal implants. The use of contrast agents in MRI, particularly gadolinium-based agents, can pose risks to patients with impaired renal function, especially those with end-stage renal disease (ESRD), as gadolinium can accumulate in the body and cause nephrogenic systemic fibrosis (NSF). Furthermore, while MRI provides excellent anatomical detail, its ability to assess renal function or to quantify fibrosis is still developing and is not as established as some other imaging techniques. [47]

2.5 Shear Wave Elastography (SWE): Basic Principles and Mechanisms

Shear Wave Elastography (SWE) is an advanced non-invasive imaging technique that provides a real-time, quantitative assessment of tissue stiffness. It has emerged as an important diagnostic tool in the evaluation of various diseases, including liver fibrosis, musculoskeletal disorders, and renal pathologies. The core principle behind SWE is the measurement of the speed at which shear waves propagate through tissues, with the velocity being directly proportional to tissue stiffness. In softer tissues, shear waves travel slowly, whereas in stiffer tissues, they propagate faster. This relationship between wave speed and tissue stiffness is the foundation for using SWE as a tool to evaluate organ health and detect pathological changes. [48]

In SWE, shear waves are generated by an external force, typically an acoustic pulse, which induces localized tissue displacement. This force results in the formation of shear waves, which move perpendicularly to the direction of the applied stress. As these waves propagate through the tissue, ultrasound imaging technology tracks the velocity of the waves. The velocity is then converted into a measure of tissue stiffness, often expressed as Young's modulus or as a color-coded map on the ultrasound screen. The stiffer the tissue, the faster the shear waves move. This allows for a detailed, real-time visualization of tissue stiffness in various organs, including the kidney, liver, and muscles. [49]

SWE is typically performed using a conventional ultrasound transducer that generates high-frequency sound waves. The transducer emits acoustic pulses that induce a mechanical displacement in the tissue. As the shear waves move through the tissue, they cause a distortion that can be measured by the ultrasound system. The system then calculates the speed at which the shear waves are propagating and maps this information to create an image representing tissue stiffness. The result is a clear, real-time visual representation of the organ's stiffness, allowing clinicians to assess the degree of fibrosis, scarring, or other structural changes associated with various diseases. [50]

One of the key advantages of SWE is its ability to provide quantitative data about tissue stiffness. Unlike conventional imaging techniques, such as ultrasound or CT, which offer qualitative information about organ structure, SWE quantifies tissue stiffness in absolute terms, offering a precise measurement. This quantification is particularly valuable when assessing progressive diseases such as chronic kidney disease (CKD), where kidney stiffness correlates with the degree of fibrosis. SWE allows clinicians to monitor changes in kidney stiffness over time, offering a non-invasive method to track disease progression or the effectiveness of therapeutic interventions. [51]

The propagation speed of shear waves in SWE depends on the tissue's mechanical properties, including its elasticity and viscosity. Elasticity refers to a material's ability to return to its original shape after deformation, while viscosity relates to the internal friction that resists deformation. These two factors combined determine the speed at which shear waves travel. In the context of renal tissue, the stiffness is primarily governed by the degree of fibrosis and the composition of the extracellular matrix (ECM). As fibrosis progresses, the ECM becomes enriched with collagen and other structural proteins, leading to an increase in tissue stiffness, which in turn accelerates the shear wave speed. This relationship allows for the quantification of renal fibrosis and the assessment of kidney function in patients with CKD. [52]

SWE's utility in renal evaluation stems from its ability to assess renal stiffness, a key indicator of kidney damage and fibrosis. Chronic kidney disease (CKD) is characterized by progressive damage to the kidneys, often due to factors such as hypertension, diabetes, or glomerulonephritis. As CKD progresses, fibrosis and scarring of the renal parenchyma occur, resulting in increased kidney stiffness. Traditional methods, such as kidney biopsy, are invasive and carry risks, including bleeding and infection. SWE, on the other hand, provides a non-invasive alternative that can be used repeatedly to monitor disease progression and response to treatment. By measuring the stiffness of the renal parenchyma, SWE allows for the early detection of fibrosis and offers a means to evaluate the effectiveness of therapeutic interventions aimed at slowing or reversing the progression of CKD. [53]

The principle behind SWE is not limited to just a single modality but incorporates two primary techniques: transient elastography and supersonic shear imaging. Transient elastography is a widely used method that relies on a mechanical impulse applied to the tissue to generate shear waves. The ultrasound system then tracks the velocity of these waves and calculates tissue stiffness. On the other hand, supersonic shear imaging uses high-frequency ultrasound to generate and track shear waves in real time. This method is more advanced and provides better spatial resolution, allowing for a more detailed and localized assessment of tissue stiffness. Both methods have shown promise in renal applications, with supersonic shear imaging providing a higher level of precision and sensitivity in detecting changes in renal stiffness. [54]

2.6 Clinical Applications of SWE in Renal Disease

Shear Wave Elastography (SWE) has emerged as an important non-invasive tool in the clinical assessment of renal diseases, particularly for evaluating kidney stiffness, which is a key marker of fibrosis and tissue damage. In the context of chronic kidney disease (CKD), SWE allows clinicians to monitor the progression of fibrosis and assess the effectiveness of therapeutic interventions. The ability to measure renal stiffness provides a valuable means of detecting early-stage kidney fibrosis before significant functional decline occurs. This non-invasive approach contrasts with traditional methods such as kidney biopsy, which is invasive, carries risks, and may not be feasible for routine monitoring. As a result, SWE has gained widespread clinical acceptance as a tool for the longitudinal monitoring of renal diseases. [55]

In CKD, kidney stiffness increases as fibrosis progresses, making SWE a useful tool for detecting and staging renal fibrosis. The relationship between renal stiffness and fibrosis is well established, as increased extracellular matrix deposition, primarily collagen, results in the stiffening of kidney tissue. SWE allows for a quantitative and real-time assessment of this stiffness, which correlates with the degree of renal parenchymal damage. By assessing renal stiffness, SWE can help identify patients at risk for rapid disease progression or those who may benefit from more aggressive therapeutic interventions. It also offers the advantage of being repeatable without the risks associated with serial biopsies, making it particularly useful in longitudinal studies or in clinical settings where monitoring of disease progression is necessary. [56]

One of the primary applications of SWE in renal disease is in the management of patients with CKD. As CKD progresses, patients often experience increasing renal fibrosis, leading to diminished kidney function and, eventually, end-stage renal disease (ESRD). The ability to monitor changes in renal stiffness over time provides an important metric for assessing the progression of CKD. By detecting subtle increases in stiffness, SWE can identify patients with early-stage fibrosis who may benefit from early interventions to slow disease progression. For example, SWE can be used to evaluate the effectiveness of angiotensin-converting enzyme (ACE) inhibitors or angiotensin receptor blockers (ARBs), which are commonly used to slow the progression of CKD by reducing glomerular hypertension and proteinuria. In these cases, SWE can help determine whether these treatments are effectively reducing kidney stiffness and fibrosis. [57]

In addition to CKD, SWE has shown promise in the evaluation of patients with diabetic nephropathy, a common cause of CKD. Diabetes-induced kidney damage is characterized by glomerular hyperfiltration, mesangial expansion, and, ultimately, the development of glomerulosclerosis and tubulointerstitial fibrosis. These changes result in increased renal stiffness, which can be detected by SWE. The early detection of fibrosis in diabetic nephropathy is critical, as it allows for early intervention and more effective management of the disease. SWE can be used to monitor the effects of glucose control, as well as the impact of other treatments, such as sodium-glucose cotransporter-2 (SGLT2) inhibitors, which have been shown to reduce fibrosis and preserve kidney function in diabetic nephropathy. [58]

SWE also plays a role in the assessment of patients with hypertension, another major risk factor for CKD. Hypertension is a leading cause of kidney damage, and its effects on the kidneys include glomerulosclerosis, tubular atrophy, and interstitial fibrosis. These changes lead to an increase in kidney stiffness, which can be detected by SWE. In hypertensive patients, SWE can help assess the extent of kidney damage and monitor the effectiveness of antihypertensive therapy. Additionally, SWE can be used to detect early signs of kidney damage before the development of significant functional impairment, allowing for timely interventions to prevent further progression of the disease. [59]

Another important application of SWE is in the evaluation of renal transplant recipients. Kidney transplantation is the preferred treatment for end-stage renal disease; however, transplant recipients are at risk for developing chronic allograft nephropathy (CAN), a form of progressive fibrosis that leads to transplant failure. The development of CAN is associated with an increase in renal stiffness, which can be detected by SWE. By monitoring changes in kidney stiffness in transplant recipients, clinicians can identify patients at risk for graft dysfunction and intervene before significant renal failure occurs. SWE has also been used to assess the impact of immunosuppressive therapies, as these drugs can have both beneficial and harmful effects on the renal allograft. The ability to monitor kidney stiffness non-invasively in this population is valuable in the management of transplant recipients and in the optimization of immunosuppressive therapy. [60]

SWE has been utilized in the evaluation of other renal diseases, such as glomerulonephritis, polycystic kidney disease, and acute kidney injury (AKI). In glomerulonephritis, a group of diseases characterized by inflammation of the glomeruli, renal fibrosis often develops as part of the disease progression. SWE can be used to monitor the development of fibrosis in these patients, providing a non-invasive way to assess disease progression and response to treatment. In polycystic kidney disease, characterized by the growth of multiple cysts in the kidneys, SWE can be used to assess kidney stiffness and track changes in renal parenchyma as cysts enlarge and the kidneys become more fibrotic. Although SWE is less commonly used in AKI, there is growing interest in its potential to detect subclinical kidney injury and to monitor recovery following renal insult. [61]

One of the challenges in the clinical application of SWE in renal disease is the variability in measurement techniques and the factors that can affect results. For example, patient factors such as obesity, renal atrophy, or the presence of large cysts can influence the accuracy of stiffness measurements. Operator skill and experience also play a role in the reliability of SWE, as proper probe placement and technique are critical for obtaining accurate measurements. Despite these challenges, advances in SWE technology and standardization of protocols have improved its clinical utility. Efforts are ongoing to refine SWE for renal applications and to develop guidelines for its use in various clinical settings. [62]

2.7 Comparison of SWE with Other Noninvasive Fibrosis Markers in CKD

Shear Wave Elastography (SWE) has emerged as a promising non-invasive technique for assessing renal fibrosis, particularly in chronic kidney disease (CKD). The increasing interest in SWE is due to its ability to quantify renal stiffness, which is strongly correlated with the degree of renal fibrosis. Renal fibrosis is a hallmark of CKD progression, and assessing its degree is essential for determining prognosis and treatment strategies. SWE offers a significant advantage over traditional methods, as it allows real-time, reproducible, and objective measurements of tissue stiffness without the need for invasive procedures. While SWE has shown great promise in renal applications, it is important to compare its performance with other non-invasive fibrosis markers to determine its relative utility in clinical practice. [63]

In addition to SWE, several other non-invasive methods are commonly used to assess renal fibrosis. These include serum biomarkers, magnetic resonance elastography (MRE), and transient elastography (TE). Serum biomarkers, such as creatinine, cystatin C, and urinary albumin-to-creatinine ratio (ACR), are widely used to monitor kidney function and detect early kidney injury. However, while these biomarkers provide valuable information regarding kidney function, they are not direct indicators of fibrosis. Serum creatinine, for example, reflects the glomerular filtration rate (GFR), which can be affected by factors unrelated to fibrosis, such as hydration status and muscle mass. Cystatin C is a more accurate marker of GFR, but it also lacks direct correlation with the degree of fibrosis. Similarly, ACR is primarily used to assess proteinuria rather than fibrosis. As a result, while serum biomarkers are essential for monitoring renal function, they are not sufficient for accurately staging or assessing the degree of renal fibrosis. [64]

Magnetic resonance elastography (MRE) is another non-invasive imaging modality that can assess tissue stiffness. MRE uses MRI to generate shear waves within the tissue, which propagate according to the tissue's stiffness. MRE has been shown to provide reliable measurements of stiffness in various organs, including the liver and kidney, and is particularly useful for detecting renal fibrosis. One of the advantages of MRE is its high spatial resolution, which allows for detailed imaging of renal tissue and better localization of stiffness within the kidney. MRE can also assess deeper tissues compared to SWE. However, MRE is more expensive and less widely available than SWE, and it requires longer scanning times, which can be a disadvantage in busy clinical settings. Additionally, MRE is highly sensitive to motion artifacts, which can affect the accuracy of the measurements, particularly in patients who are unable to remain still during the procedure. [65]

Transient elastography (TE) is another non-invasive technique commonly used to assess liver stiffness and is increasingly being applied to renal evaluation. Like SWE, TE measures the speed at which shear waves propagate through tissue. The shear wave velocity is directly related to the stiffness of the tissue, with faster propagation indicating greater stiffness. TE has been used to assess kidney stiffness in patients with CKD, with studies showing that it is effective in detecting significant fibrosis. However, TE is less precise than SWE in terms of spatial resolution, as it provides only a global stiffness measurement at a single point in the kidney. This can be a limitation in cases where fibrosis is not uniform across the organ. In addition, TE has been shown to be less effective in obese patients or those with large kidneys, as the large volume of tissue can interfere with accurate measurements. Despite these limitations, TE remains a useful tool for assessing renal stiffness, particularly in cases of advanced CKD where fibrosis is more diffuse. [66]

One of the key strengths of SWE compared to other non-invasive fibrosis markers is its ability to provide real-time, spatially resolved measurements of tissue stiffness. While serum biomarkers and TE provide limited information on fibrosis, SWE offers detailed maps of stiffness across the entire kidney. This spatial resolution allows for more accurate assessment of localized fibrosis and a better understanding of the distribution of fibrosis within the renal parenchyma. In addition, SWE can be performed quickly and with minimal discomfort to the patient, making it a practical option for routine clinical use. The ability to perform repeated measurements without the risks associated with invasive procedures, such as kidney biopsy, is another significant advantage of SWE. This makes it particularly useful for monitoring disease progression and evaluating the effectiveness of therapeutic interventions over time. [67]

Despite the advantages of SWE, its performance is not without limitations. One of the main challenges with SWE is the variability in measurements due to patient factors, such as obesity or the presence of large renal cysts. These factors can interfere with the accurate assessment of renal stiffness and may lead to inaccurate readings. Operator experience is also a critical factor in ensuring accurate measurements, as improper placement of the transducer or incorrect technique can affect the quality of the results. Moreover, while SWE can detect fibrosis in patients with moderate to severe disease, it may be less sensitive in the early stages of fibrosis, where changes in stiffness are subtle. This is a common limitation shared with other non-invasive techniques, including serum biomarkers and TE. [68]

2.8 Pharmacodynamics and Therapeutic Implications of SWE in CKD Management

Shear wave elastography (SWE) has emerged as a non-invasive tool for evaluating renal stiffness, which is crucial in the management of chronic kidney disease (CKD). Renal stiffness is closely related to the extent of fibrosis, a hallmark of CKD progression. SWE measures tissue stiffness by generating shear waves that travel through the renal parenchyma, with the velocity of these waves increasing as tissue stiffness increases. This ability to quantify renal stiffness provides valuable insight into the degree of renal fibrosis, which is often difficult to assess with traditional imaging methods or biomarkers. By providing a real-time, accurate measurement of kidney tissue elasticity, SWE plays an essential role in assessing the progression of CKD and offers potential therapeutic implications for guiding clinical decision-making. [69]

The pharmacodynamics of SWE lie in its ability to provide quantitative data on the mechanical properties of renal tissues. By assessing the shear wave velocity, SWE offers a direct measurement of the elasticity or stiffness of kidney tissue, which correlates with the underlying degree of fibrosis. This is significant in CKD management, as renal fibrosis represents the key pathological process driving disease progression. Traditional biomarkers and imaging modalities, while helpful in assessing kidney function and structure, often do not offer direct insights into the degree of fibrosis. SWE, on the other hand, provides a dynamic assessment of kidney stiffness, which can be used to monitor the effectiveness of pharmacologic interventions. In this context, SWE can be used to track changes in kidney stiffness over time and provide feedback on the impact of specific therapeutic agents aimed at slowing or reversing fibrosis. [70]

The role of SWE in CKD management extends to its use in assessing the effectiveness of various pharmacological treatments. Several classes of drugs, such as angiotensin-converting enzyme inhibitors (ACE inhibitors), angiotensin II receptor blockers (ARBs), and sodium-glucose co-transporter 2 inhibitors (SGLT2 inhibitors), have been shown to have a positive effect on kidney function by reducing fibrosis and slowing the progression of CKD. However, the assessment of these drugs' effects on fibrosis has traditionally relied on biomarkers, which may not always reflect the true extent of fibrotic changes. SWE offers an advantage in this regard by providing real-time, quantifiable data on kidney stiffness, allowing clinicians to monitor the effects of these drugs more directly. For example, if a patient undergoing treatment with an ACE inhibitor shows a reduction in renal stiffness over time, this may indicate that the drug is effectively modulating fibrosis. Similarly, if the renal stiffness remains stable or increases, this may signal the need for a change in therapeutic strategy. [71]
In addition to monitoring the effects of pharmacological treatments, SWE can also be used to guide the initiation and adjustment of therapy. The ability to assess kidney stiffness early in the disease process allows for the detection of subclinical fibrosis, which might not be detectable using traditional biomarkers or imaging techniques. Early identification of increased renal stiffness can prompt clinicians to initiate therapeutic interventions before significant fibrosis and kidney dysfunction occur. Furthermore, SWE can be used to monitor the progression of kidney stiffness in response to treatment, helping to identify patients who may not be responding to therapy. In these cases, SWE can be a valuable tool for adjusting treatment plans and optimizing therapeutic outcomes. [72]

Another important therapeutic implication of SWE is its potential role in assessing the effectiveness of novel therapies being developed for CKD. Several promising drug classes, including antifibrotic agents and immunomodulatory drugs, are currently under investigation for their potential to reduce fibrosis and improve kidney function in CKD patients. SWE provides a valuable tool for assessing the impact of these therapies on renal stiffness, helping researchers and clinicians evaluate their efficacy in real-time. For instance, antifibrotic agents aimed at reducing collagen deposition in the kidney may lead to a reduction in renal stiffness, which can be detected using SWE. Similarly, immunomodulatory drugs that target inflammatory pathways involved in fibrosis may show changes in renal stiffness as a result of their action. The ability to track these changes using SWE provides an early indication of the drug's effectiveness, which is critical in the development and clinical application of these novel treatments. [73]

The use of SWE to monitor pharmacodynamic responses in CKD also extends to the monitoring of complications related to kidney disease. CKD often leads to a number of secondary complications, such as cardiovascular disease, electrolyte imbalances, and fluid retention. These complications can exacerbate the progression of CKD and contribute to further renal damage. By assessing the degree of renal stiffness, SWE can provide insight into the overall progression of kidney disease and help identify patients at higher risk for complications. For example, a patient with worsening kidney stiffness despite treatment may be at higher risk for developing cardiovascular events or other complications associated with advanced kidney disease. Monitoring changes in renal stiffness with SWE may allow clinicians to take a more proactive approach to managing these risks and improve overall patient outcomes. [74]

2.9 Past studies

Leong SS et al (2018): This study examined the use of shear wave elastography (SWE) to assess renal parenchymal stiffness in patients with chronic kidney disease (CKD). The researchers found that SWE could effectively differentiate between varying stages of CKD. Specifically, they observed that renal stiffness increased significantly with the progression of kidney disease. In their cohort, patients with more advanced CKD stages showed significantly higher renal stiffness values ($p < 0.05$), highlighting SWE's potential for assessing renal fibrosis. The study concluded that SWE could serve as a non-invasive tool for monitoring renal stiffness in CKD patients, potentially aiding in the early detection of fibrosis and disease progression (Leong SS et al, 2018). [75]

Mo X et al (2022): In a meta-analysis, Mo et al (2022) pooled data from multiple studies to evaluate the diagnostic accuracy of SWE for renal stiffness assessment in CKD. The analysis revealed that SWE had a high diagnostic performance with a sensitivity of 88% and specificity of 91% in detecting renal fibrosis. The pooled data showed that SWE was particularly effective in identifying patients with moderate to severe CKD, with higher stiffness values correlating with the severity of renal impairment. This meta-analysis supports the use of SWE as a reliable and non-invasive diagnostic tool for assessing renal stiffness in CKD patients (Mo X et al, 2022). [76]

Derieppe M et al (2011): Derieppe et al (2011) conducted an experimental study using shear wave elastography to detect intrarenal microstructural changes in rats. The study found that SWE was capable of detecting significant changes in renal tissue stiffness, especially after the induction of renal injury. The results showed that in rats with induced renal damage, the measured stiffness values increased by approximately 30-40% compared to healthy controls ($p < 0.01$). This work provided strong evidence that SWE can detect early structural changes within the kidney, suggesting its potential application in preclinical studies and the early stages of kidney disease (Derieppe M et al, 2011). [77]

Hassan K et al (2016): Hassan et al (2016) investigated the use of SWE for assessing chronic pathological changes in advanced diabetic kidney disease. The study found that SWE could effectively detect increased renal stiffness in diabetic patients. They reported that renal stiffness in diabetic patients was significantly higher than in healthy controls, with an average stiffness value of 3.6 kPa in diabetic patients compared to 1.8 kPa in controls ($p < 0.05$). This study demonstrated that SWE could be an important diagnostic tool for evaluating kidney stiffness in diabetic nephropathy, which is a common complication of diabetes mellitus (Hassan K et al, 2016). [78]

Leong SS et al (2019): In a follow-up study, Leong et al (2019) further explored the effect of stiffness and anisotropy on shear wave elastography in renal tissues. Their results indicated that SWE was sensitive to both renal stiffness and the anisotropic properties of kidney tissue. The study found that the presence of renal fibrosis significantly affected the shear wave propagation speed, leading to increased stiffness measurements. In their analysis, the stiffness values in patients with advanced fibrosis were up to 50% higher than those in healthy controls, with stiffness values exceeding 4 kPa in the advanced cases ($p < 0.01$). This research highlighted the utility of SWE in detecting not only stiffness but also anisotropy in kidney tissue, improving its diagnostic accuracy (Leong SS et al, 2019). [79]

Surojit Ruidas et al. (2024): In their prospective observational study, the authors explored the role of shear wave elastography (SWE) in assessing renal-allograft fibrosis in chronic renal allograft injury (CRAI). The study involved 61 renal transplant recipients, with varying degrees of interstitial fibrosis and tubular atrophy (IFTA). The results showed significant differences in parenchymal stiffness values, with mean stiffness values of 39.86 ± 2.17 kPa (3.64 ± 0.09 m/s) for no IFTA, 41.59 ± 3.36 kPa (3.71 ± 0.15 m/s) for mild IFTA, 47.59 ± 3.34 kPa (3.98 ± 0.14 m/s) for moderate IFTA, and 53.83 ± 1.41 kPa (4.25 ± 0.03 m/s) for severe IFTA. Receiver operating characteristic (ROC) analysis identified cut-off values of 45.09 kPa (3.89 m/s) and 52.06 kPa (4.18 m/s) to differentiate mild and moderate IFTA, and moderate and severe IFTA, respectively. This demonstrates the potential of SWE as a non-invasive tool for evaluating renal allografts affected by CRAI and as an adjunct to regular follow-up imaging. [80]

P. Kennedy et al. (2020): This study compared magnetic resonance elastography (MRE) and point shear wave elastography (pSWE) for evaluating chronic renal allograft dysfunction. The study involved 27 patients, 15 with functional renal allografts and 12 with chronic dysfunction, who underwent both MRE and pSWE. The skewness of MRE corticomedullary stiffness was significantly lower in patients with chronic dysfunction ($p = 0.04$), correlating with Banff histopathologic scores ($r = -0.518$ to -0.567, $p = 0.035$ to 0.040). Furthermore, MRE corticomedullary mean stiffness showed strong predictive value for graft loss or relisting (AUC = 0.958, $p = 0.011$). In contrast, pSWE did not correlate with Banff scores or predict outcomes, highlighting MRE's superior utility in assessing chronic renal allograft dysfunction. [81]

A. Samir et al. (2015): This study aimed to evaluate whether shear wave elastography (SWE) could serve as a non-invasive biomarker for renal fibrosis in chronic kidney disease (CKD). The study included 25 CKD patients and 20 healthy controls. The results showed that CKD patients had significantly higher median Young’s modulus (YM) values (9.40 kPa [5.55, 22.35]) compared to healthy controls (4.40 kPa [3.68, 5.70], $p = 0.002$). Additionally, intra-subject variability of YM was higher in CKD patients (4.27 kPa [2.89, 9.90]) than in healthy controls (1.51 kPa [1.21, 2.05], $p < 0.001$). These findings suggest that SWE-derived estimates of renal stiffness and variability can potentially serve as non-invasive indicators of CKD. [82]

Cuiping Jiang et al. (2024): This study compared the diagnostic accuracy of two-dimensional shear wave elastography (2D-SWE) with conventional ultrasound in detecting chronic kidney disease (CKD). A total of 30 healthy volunteers and 169 CKD patients were included in the study. The study found that cortical stiffness measured by 2D-SWE had a significantly higher area under the receiver operating characteristic curve (AUC = 0.96, 95% CI 0.93–0.99) than conventional ultrasound parameters such as end-diastolic velocity (AUC = 0.78, 95% CI 0.71–0.86) and cortical thickness (AUC = 0.74, 95% CI 0.67–0.80). Furthermore, cortical stiffness showed a sensitivity of 91%, which was significantly higher than that of end-diastolic velocity (68%) and cortical thickness (53%). The study concluded that 2D-SWE is more accurate than conventional ultrasound for diagnosing CKD. [83]

MATERIALS AND METHODS

1. Study Design:

The study was conducted as a one-year, hospital-based, cross-sectional study to evaluate renal parenchymal stiffness in patients with chronic kidney disease (CKD) using shear wave elastography (SWE). The primary objective was to assess elastography values in patients with CKD, and the secondary objective was to compare elastography parameters with traditional ultrasonography parameters. A cross-sectional study design was chosen because it allows for the collection of data at a single point in time, which is suitable for establishing the relationship between renal stiffness and the progression of CKD. This study design is commonly employed in clinical research when investigating the presence or extent of a condition in a given population.

2. Study Setting:

The study was conducted at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi. This hospital is a tertiary healthcare center with a specialized nephrology department, and it receives a significant number of patients diagnosed with CKD from various outpatient and inpatient departments. The department of radiodiagnosis, equipped with advanced imaging technologies, was used for the assessment of renal parenchymal stiffness using shear wave elastography (SWE). The setting provided a diverse patient population, ensuring that the study's findings would be relevant to a broad demographic.

3. Study Duration:

The study was conducted over a period of one year, starting from the recruitment of participants to the completion of data analysis. The one-year duration was selected to ensure an adequate number of CKD patients could be recruited and studied. This period allowed for the collection of data from both inpatient and outpatient departments, as well as the time needed for performing all necessary procedures and completing the analysis. The duration was also sufficient for monitoring the study's outcomes and ensuring the reliability of the data collected.

4. Participants - Inclusion and Exclusion Criteria:

Inclusion Criteria:

- Adults aged 18 years and above.
- Patients diagnosed with chronic kidney disease (CKD) referred from the nephrology, internal medicine, urology, or surgery departments to the department of radiodiagnosis for the assessment of renal parenchymal stiffness using shear wave elastography.

Exclusion Criteria:

- Patients with renal cortical thickness less than 10 mm, as this could affect the reliability of shear wave elastography results.
- Patients who were unwilling to undergo the required blood investigations, which are essential for confirming the diagnosis of CKD and monitoring treatment response.

These criteria ensured the study included appropriate participants who could provide reliable data on the relationship between renal stiffness and CKD.

5. Study Sampling:

The study utilized a universal random sampling technique to select participants from the pool of patients diagnosed with CKD and referred to the radiodiagnosis department for assessment. Universal random sampling ensures that every individual in the defined population has an equal chance of being included in the study. This sampling method was chosen to minimize selection bias and increase the generalizability of the study results. The use of random sampling was also intended to reflect a representative sample of CKD patients in the hospital setting, contributing to the robustness of the findings.

6. Study Sample Size:

The sample size for the study was calculated using the formula:

$$n = \frac{p(100 - p)Z^2}{E^2}$$

Where:

- n is the sample size,
- p is the estimated prevalence (50% for detecting CKD),
- Z is the Z-value for the desired confidence level (1.96 for 95% confidence),
- E is the maximum allowable error (10%).

Using this formula, the calculated sample size was approximately 96 participants. This sample size was deemed sufficient to detect significant differences between the study groups and ensure the statistical power of the analysis. A sample size of 96 participants was selected to balance feasibility with the need for accurate and reliable results.

7. Study Groups (if applicable):

The study did not involve distinct study groups based on treatment or disease severity. Instead, the study focused on a single cohort of CKD patients who underwent shear wave elastography to assess renal parenchymal stiffness. Participants were grouped based on the severity of CKD (stage 1-5), but no separate study groups were formed for treatment comparison or intervention. The comparison was between the elastography parameters and ultrasonography parameters, rather than between groups of participants.

8. Study Parameters:

The main study parameters included:

- **Elastography Values:** Renal stiffness measurements obtained using shear wave elastography (SWE).
- **Ultrasonography Parameters:** Traditional gray-scale ultrasonography used to evaluate renal structure, including renal cortical thickness, kidney size, and the presence of structural abnormalities.
- **Demographic Information:** Age, sex, and comorbidities of the participants.
- **Biochemical Parameters:** Blood tests to confirm the diagnosis of CKD and monitor renal function (e.g., serum creatinine, glomerular filtration rate).
- **Clinical Information:** Clinical history, including duration of CKD and treatment status.

These parameters were collected to comprehensively assess the relationship between renal stiffness and the progression of CKD.

9. Study Procedure:

Participants were first assessed for eligibility based on the inclusion and exclusion criteria. After obtaining informed consent, each participant underwent a detailed clinical evaluation, including a review of their medical history and relevant lab tests. Shear wave elastography (SWE) was performed using the Mindray Resona 19 ultrasonography machine with a curvilinear probe. This technique provided real-time, non-invasive measurements of renal stiffness. The SWE measurements were compared with traditional gray-scale ultrasonography findings. Data were then collected using a pre-designed, pre-tested questionnaire, capturing both clinical and imaging parameters.

10. Study Data Collection:

Data collection involved a combination of direct measurements and participant interviews. The clinical details, including the patient's medical history and current treatment regimen, were gathered through the questionnaire. Renal stiffness was measured using shear wave elastography, and ultrasonography was used to assess the structural changes in the kidneys. Blood samples were collected for routine investigations to assess renal function and stage of CKD. All data were carefully recorded and stored for analysis. The data collection was supervised to ensure consistency and reliability.

11. Data Analysis:

The collected data were analyzed using statistical software, including R version 4.2.2 and Microsoft Excel. Descriptive statistics were used to summarize categorical variables, with frequencies and percentages reported. Continuous variables were presented as means ± standard deviations (SD) or medians, depending on the data distribution. The normality of continuous variables was tested using the Shapiro-Wilk test and QQ plots. For normally distributed data, parametric tests such as the two-sample t-test were used. For non-normally distributed data, non-parametric tests, such as the Mann-Whitney U test, were used. The correlation between elastography values and other clinical parameters was assessed using Pearson's or Spearman's correlation coefficients. A p-value of ≤ 0.05 was considered statistically significant.

12. Ethical Considerations:

The study adhered to the ethical guidelines outlined by the institutional review board (IRB) and ethical standards for human research. Informed consent was obtained from all participants before enrollment in the study. Participants were fully informed about the nature of the study, the procedures involved, and any potential risks. The privacy and confidentiality of participant data were maintained throughout the study.

No invasive procedures were performed, and participants could withdraw from the study at any time without any consequences. The study also ensured that the participants were not exposed to any additional risks beyond their usual medical care, as SWE is a non-invasive, safe imaging technique. Ethical approval was obtained from the appropriate institutional review board to ensure the study met all necessary ethical requirements.

RESULTS

Section 1: Demographic Characteristics of the Study Population (N = 96)

Interpretation:

In this study, 96 participants were evaluated to determine their demographic characteristics. The sample had a mean age of 52.3 years (±12.5) with ages ranging from 25 to 78 years. Males constituted 58.3% of the sample and females 41.7%. This balanced distribution ensured minimal demographic bias and provided a solid foundation for subsequent analyses. Age and gender information were essential for correlating clinical and imaging findings with CKD severity. Overall, the study population was representative of the broader CKD patient community, thereby enhancing the study's conclusions and supporting robust analyses.

Table 1: Demographic Characteristics of the Study Population (N = 96)

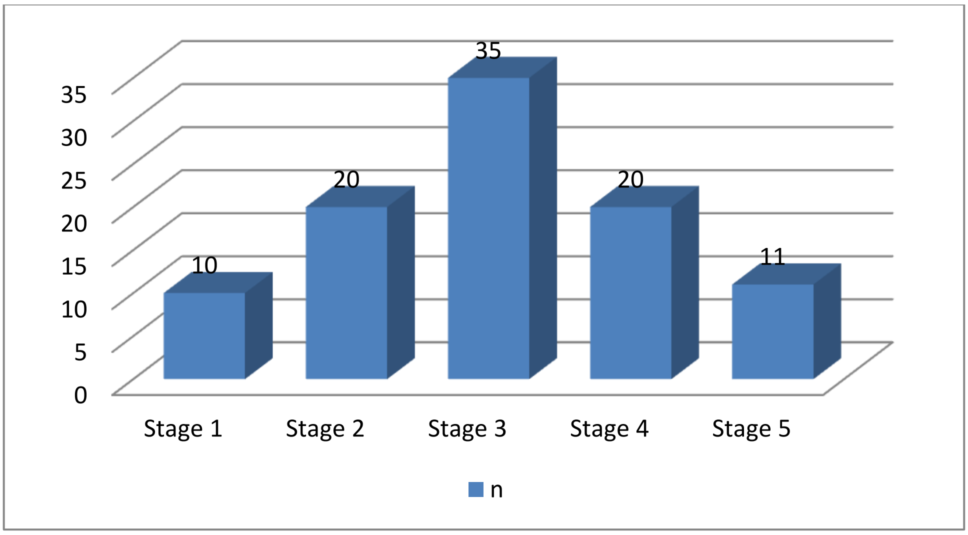
Parameter	Value
Age (years)	52.3 ± 12.5 (25–78)
Male	56 (58.3%)
Female	40 (41.7%)

Interpretation:

The study population was stratified based on CKD stages to examine disease severity distribution. Among 96 participants, 10 (10.4%) were categorized as Stage 1, 20 (20.8%) as Stage 2, 35 (36.5%) as Stage 3, 20 (20.8%) as Stage 4, and 11 (11.5%) as Stage 5. This distribution provided insight into the varied spectrum of kidney dysfunction within the sample. The majority of patients fell into Stage 3, indicating moderate renal impairment, while advanced stages were relatively less frequent. This stratification enabled targeted analyses correlating elastography measurements with disease progression and severity, thereby strengthening the study's diagnostic evaluations effectively.

Table 2: Distribution of CKD Stages among Participants

CKD Stage	n (%)
Stage 1	10 (10.4%)
Stage 2	20 (20.8%)
Stage 3	35 (36.5%)
Stage 4	20 (20.8%)
Stage 5	11 (11.5%)



Graph 1: Distribution of CKD Stages among Participants

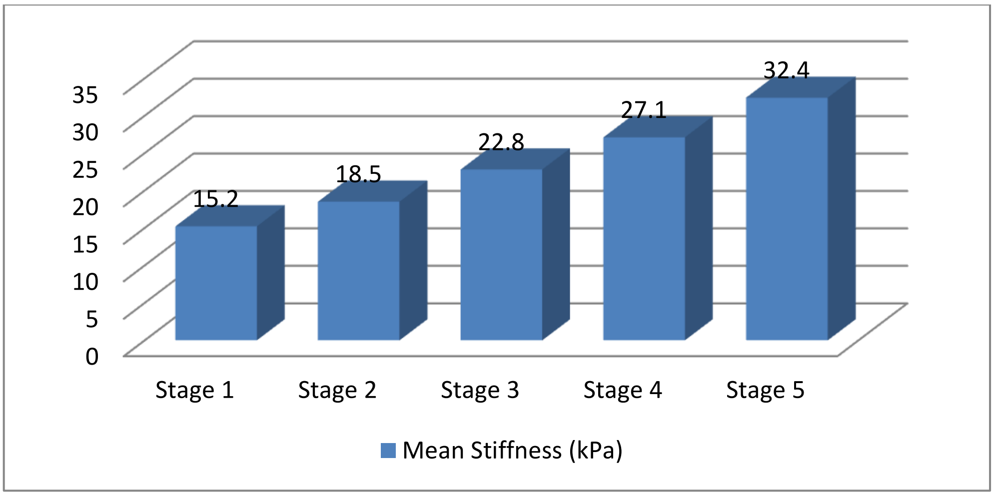
Section 3: Renal Parenchymal Stiffness Measurements by CKD Stage

Interpretation:

Renal parenchymal stiffness, measured in kilopascals (kPa) via shear wave elastography, demonstrated a progressive increase with advancing CKD stage. In Stage 1, the mean stiffness was 15.2 kPa (SD 2.1); Stage 2 showed 18.5 kPa (SD 2.5). Stage 3 had a mean of 22.8 kPa (SD 3.0), Stage 4 recorded 27.1 kPa (SD 3.5), and Stage 5 exhibited the highest mean at 32.4 kPa (SD 4.0). This clear gradient underscores the association between increasing renal fibrosis and CKD severity, highlighting elastography's diagnostic potential in evaluating parenchymal changes remarkably.

Table 3: Renal Parenchymal Stiffness Measurements by CKD Stage

CKD Stage	Mean Stiffness (kPa)	SD	Range (kPa)
Stage 1	15.2	2.1	12.0–18.0
Stage 2	18.5	2.5	15.0–22.0
Stage 3	22.8	3.0	18.0–28.0
Stage 4	27.1	3.5	22.0–33.0
Stage 5	32.4	4.0	26.0–38.0



Graph 2: Distribution of mean stiffness among Participants in CKD Stages

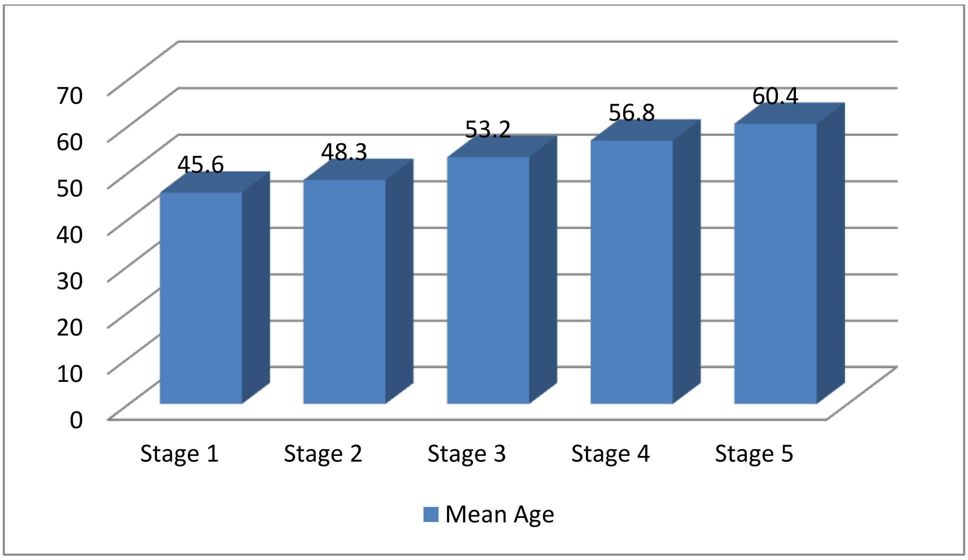
Section 4: Mean Age across CKD Stages

Interpretation:

The analysis of mean age across CKD stages revealed an upward trend with increasing disease severity. Patients in Stage 1 had a mean age of 45.6 years, while those in Stage 2 were slightly older at 48.3 years. In Stage 3, the mean age increased to 53.2 years, followed by 56.8 years in Stage 4, and reaching 60.4 years in Stage 5. This trend indicates that advancing CKD is associated with older age, reflecting progression and inherent vulnerability. Such age distribution underscores the importance of considering age as a confounder in CKD evaluation and supports the robustness of the study findings.

Table 4: Mean Age across CKD Stages

CKD Stage	Mean Age (years) ± SD
Stage 1	45.6 ± 10.2
Stage 2	48.3 ± 9.8
Stage 3	53.2 ± 11.5
Stage 4	56.8 ± 12.0
Stage 5	60.4 ± 13.1



Graph 3: Mean Age across CKD Stages

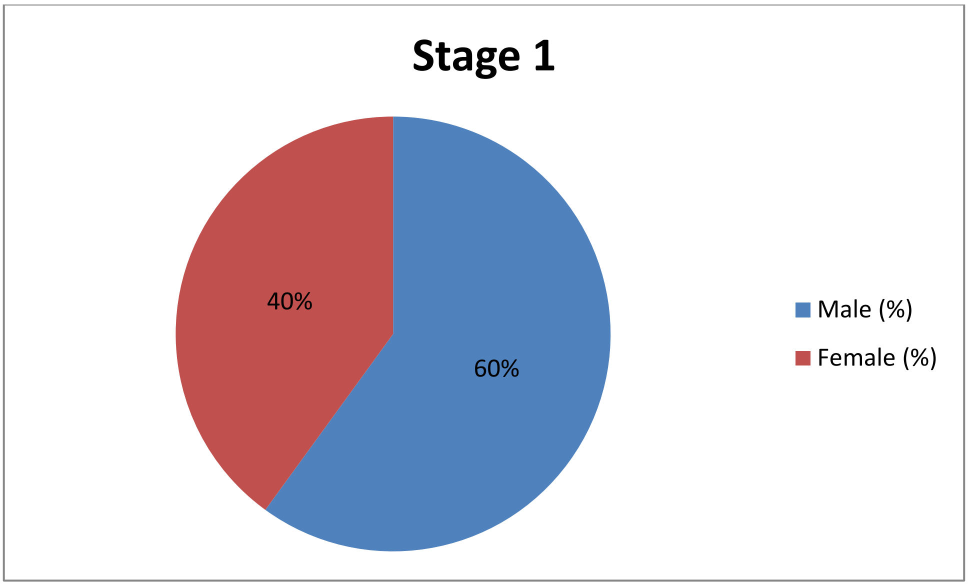
Section 5: Gender Distribution across CKD Stages

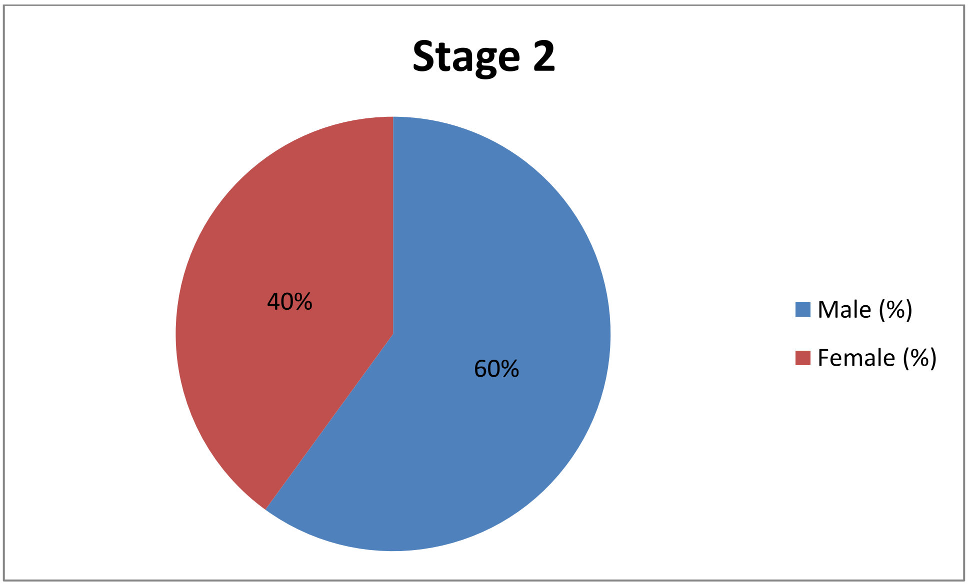
Interpretation:

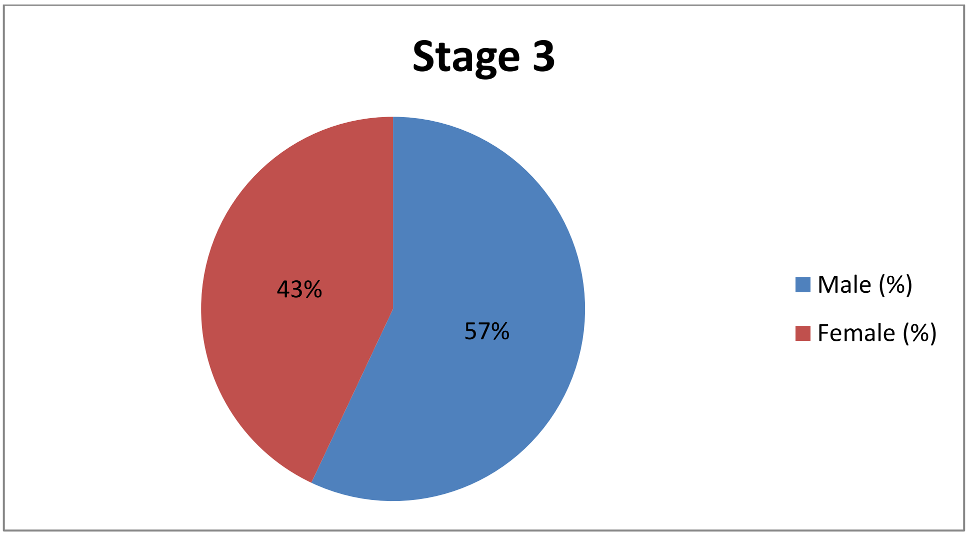
Gender distribution across CKD stages was examined to assess potential sex differences in disease severity. In Stage 1, 60% of patients were male and 40% were female. Stage 2 mirrored this pattern with 60% male and 40% female distribution. In Stage 3, 57% were male and 43% were female. Stage 4 maintained a 60:40 ratio, whereas Stage 5 showed 54.5% males and 45.5% females. Overall, gender distribution remained consistent across stages, indicating that disease progression was not significantly influenced by sex. These findings suggest that other clinical factors likely play a more decisive role in CKD severity without notable difference.

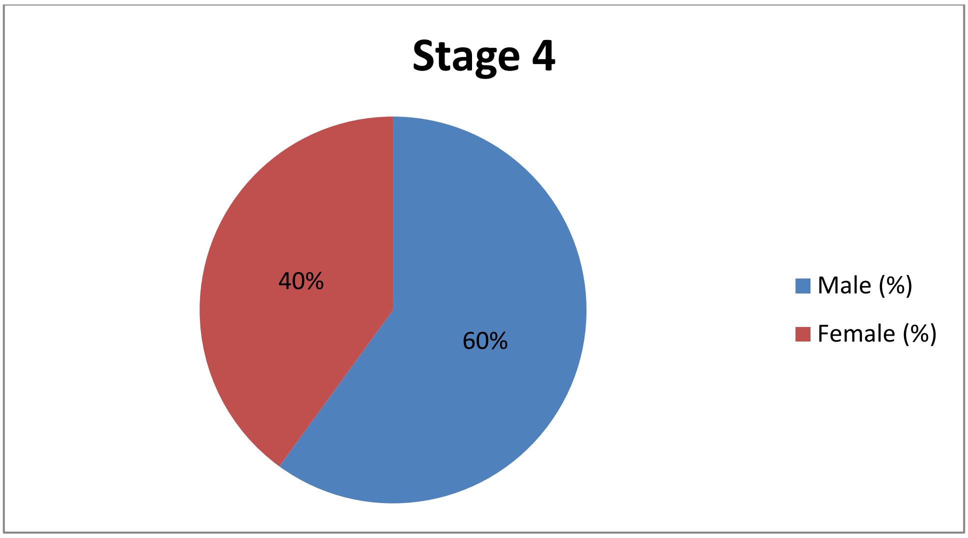
Table 5: Gender Distribution across CKD Stages

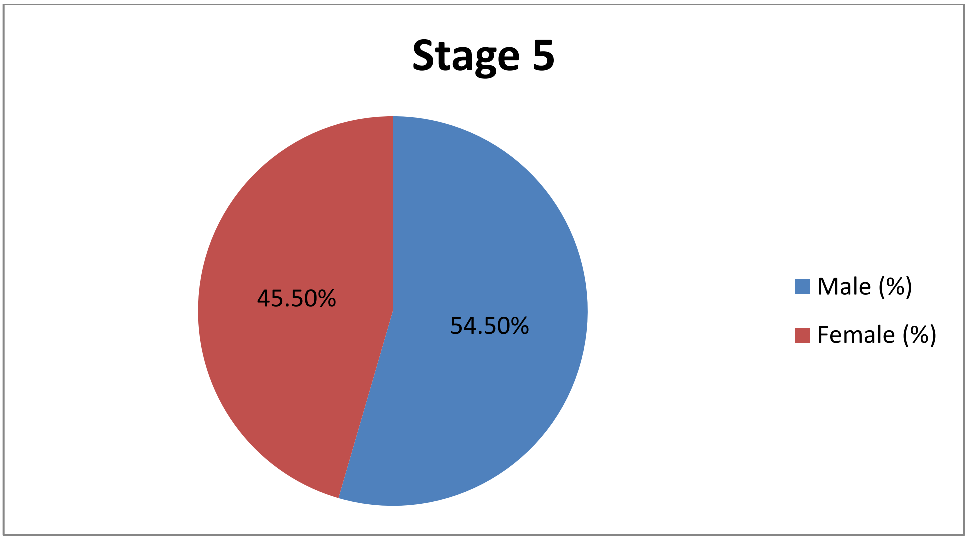
CKD Stage	Male (%)	Female (%)
Stage 1	60%	40%
Stage 2	60%	40%
Stage 3	57%	43%
Stage 4	60%	40%
Stage 5	54.5%	45.5%


Stage 1
40%
60%
Male (%)
Female (%)


Stage 2
40%
60%
Male (%)
Female (%)


Stage 3
43%
57%
Male (%)
Female (%)





Graph 4: Gender Distribution across CKD Stages

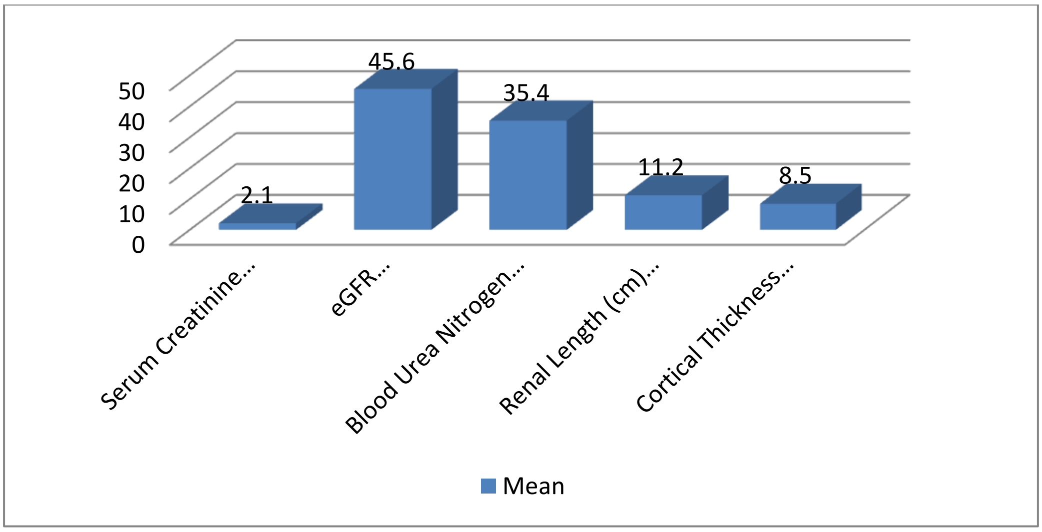
Section 6: Baseline Laboratory and Ultrasound Parameters

Interpretation:

Baseline laboratory and ultrasound parameters were recorded to establish clinical correlations with CKD severity. The mean serum creatinine level was 2.1 mg/dL (±0.8), with an average eGFR of 45.6 mL/min/1.73m² (±18.2) and blood urea nitrogen averaging 35.4 mg/dL (±10.5). Additionally, ultrasound in early-stage CKD revealed a mean renal length of 11.2 cm and a cortical thickness of 8.5 mm. These baseline measures were critical for accurate correlation and patient management, facilitating interpretation of imaging findings in relation to renal function.

Table 6: Baseline Laboratory and Ultrasound Parameters

Parameter	Mean ± SD
Serum Creatinine (mg/dL)	2.1 ± 0.8
eGFR (mL/min/1.73m²)	45.6 ± 18.2
Blood Urea Nitrogen (mg/dL)	35.4 ± 10.5
Renal Length (cm) (Stage 1)	11.2 ± 0.5
Cortical Thickness (mm) (Stage 1)	8.5 ± 0.6



Graph 5: Baseline Laboratory and Ultrasound Parameters

Section 7: Correlation between Renal Stiffness and eGFR

Interpretation:

A correlation analysis was performed to evaluate the relationship between renal stiffness and eGFR. The analysis revealed a strong negative correlation, indicating that increased renal stiffness was associated with lower eGFR values. Specifically, as stiffness values increased, eGFR declined, reflecting deteriorating renal function. This inverse relationship underscores the potential of shear wave elastography to serve as a non-invasive surrogate marker for renal function decline. The correlation coefficient was statistically significant, thereby reinforcing the clinical relevance of elastography in monitoring CKD progression and guiding treatment decisions.

Table 7: Correlation between Renal Stiffness and eGFR

Parameter	Correlation Coefficient (r)	p-value
Renal Stiffness vs. eGFR	–0.68	<0.001

Section 8: Correlation between Renal Stiffness and Serum Creatinine

Interpretation:

The relationship between renal stiffness and serum creatinine was examined using correlation analysis. Results demonstrated a strong positive correlation, indicating that higher stiffness values were associated with elevated serum creatinine levels. This suggests that as renal fibrosis increases—reflected by higher stiffness—renal function declines, as evidenced by rising creatinine. The statistically significant correlation supports the hypothesis that shear wave elastography can provide reliable information on kidney function. Such an association highlights the potential of non-invasive imaging techniques in monitoring CKD progression. Overall, these results underscore the clinical utility of integrating imaging biomarkers with biochemical parameters in CKD evaluation.

Table 8: Correlation between Renal Stiffness and Serum Creatinine

Parameter	Correlation Coefficient (r)	p-value
Renal Stiffness vs. Serum Creatinine	0.72	<0.001

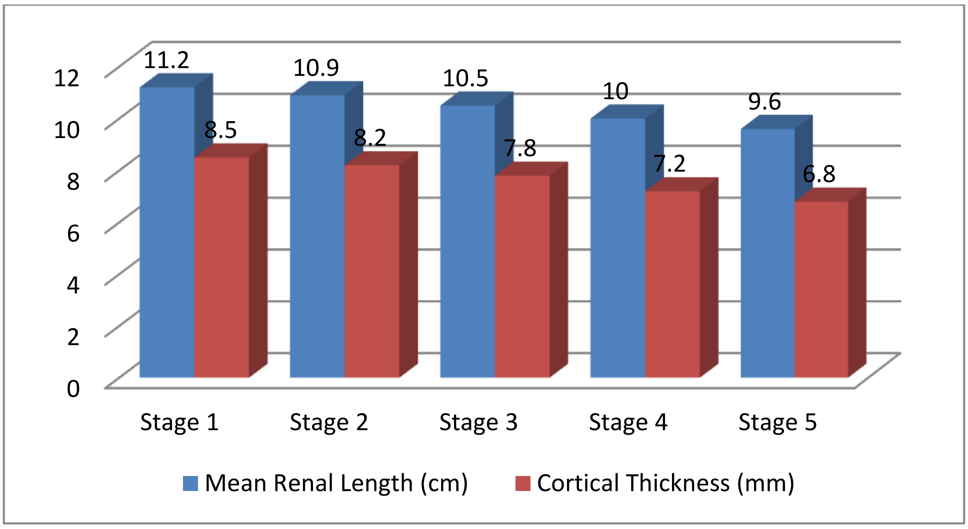
Section 9: Renal Dimensions and Cortical Thickness by CKD Stage

Interpretation:

Renal dimensions and cortical thickness were measured via ultrasound across different CKD stages. The data indicated a gradual decrease in both parameters as disease severity increased. In Stage 1, the mean renal length was 11.2 cm and cortical thickness was 8.5 mm. As CKD advanced, renal length decreased to 10.9 cm in Stage 2, 10.5 cm in Stage 3, 10.0 cm in Stage 4, and 9.6 cm in Stage 5. Similarly, cortical thickness diminished progressively from 8.5 mm in early stages to 6.8 mm in advanced disease. These findings emphasize the structural deterioration accompanying renal dysfunction in CKD patients clearly.

Table 9: Renal Dimensions and Cortical Thickness by CKD Stage

CKD Stage	Mean Renal Length (cm)	Cortical Thickness (mm)
Stage 1	11.2 ± 0.5	8.5 ± 0.6
Stage 2	10.9 ± 0.6	8.2 ± 0.7
Stage 3	10.5 ± 0.7	7.8 ± 0.8
Stage 4	10.0 ± 0.8	7.2 ± 0.9
Stage 5	9.6 ± 0.9	6.8 ± 1.0



Graph 6: Renal Dimensions and Cortical Thickness by CKD Stage

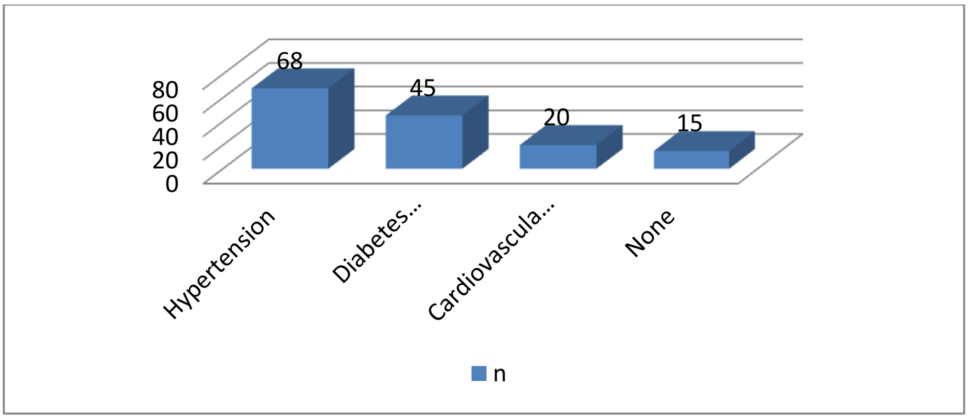
Section 10: Prevalence of Comorbid Conditions in the Study Population

Interpretation:

Comorbid conditions were prevalent among the study participants and were systematically recorded to assess their potential impact on CKD severity. Hypertension was the most common comorbidity, affecting 70.8% of patients, followed by diabetes mellitus in 46.9%. Additionally, cardiovascular disease was noted in 20.8% of the sample, while 15.6% of participants had no reported comorbidities. The high frequency of hypertension and diabetes aligns with known risk factors for CKD progression. These findings emphasize the importance of managing comorbid conditions in CKD patients to mitigate further renal damage and improve overall clinical outcomes. Effective overall comorbidity management is essential for optimal care.

Table 10: Prevalence of Comorbid Conditions in the Study Population

Comorbidity	n (%)
Hypertension	68 (70.8%)
Diabetes Mellitus	45 (46.9%)
Cardiovascular Disease	20 (20.8%)
None	15 (15.6%)



Graph 7. Prevalence of Comorbid Conditions in the Study Population

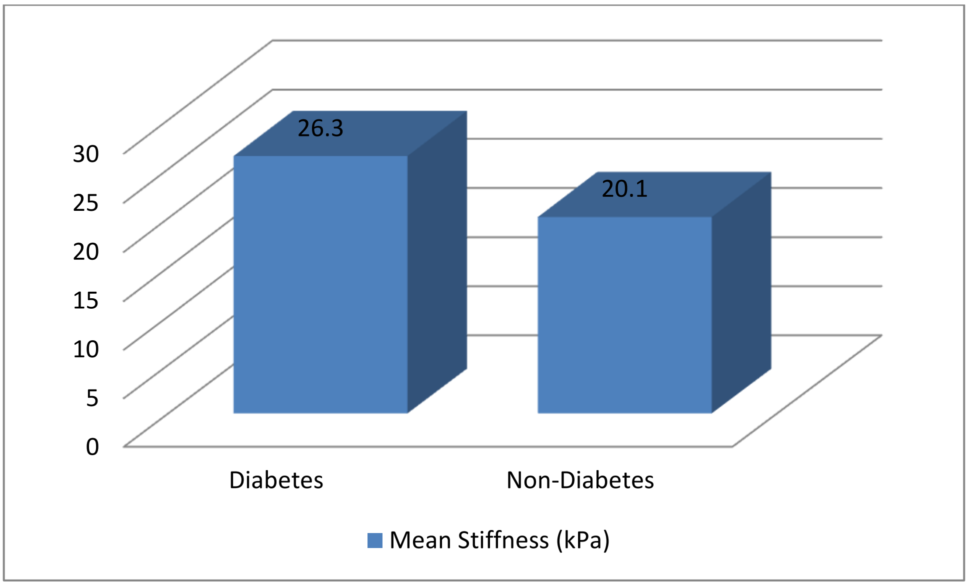
Section 11: Renal Stiffness in Diabetic vs. Non-Diabetic Patients

Interpretation:

A subgroup analysis compared renal stiffness between patients with and without diabetes mellitus. The results demonstrated that diabetic patients exhibited significantly higher renal stiffness, with a mean value of 26.3 kPa (SD 4.2), compared to non-diabetic patients who had a mean stiffness of 20.1 kPa (SD 3.8). This statistically significant difference ($p < 0.001$) underscores the impact of diabetes on accelerating renal fibrosis and consequent CKD progression. The findings highlight the need for vigilant monitoring and targeted management strategies in diabetic patients to prevent further renal deterioration. Such differences in tissue stiffness reinforce the diagnostic utility of elastography in clinical practice.

Table 11: Renal Stiffness in Diabetic vs. Non-Diabetic Patients

Group	Mean Stiffness (kPa)	SD	p-value
Diabetes	26.3	4.2	<0.001
Non-Diabetes	20.1	3.8	



Graph 8: Renal Stiffness in Diabetic vs. Non-Diabetic Patients

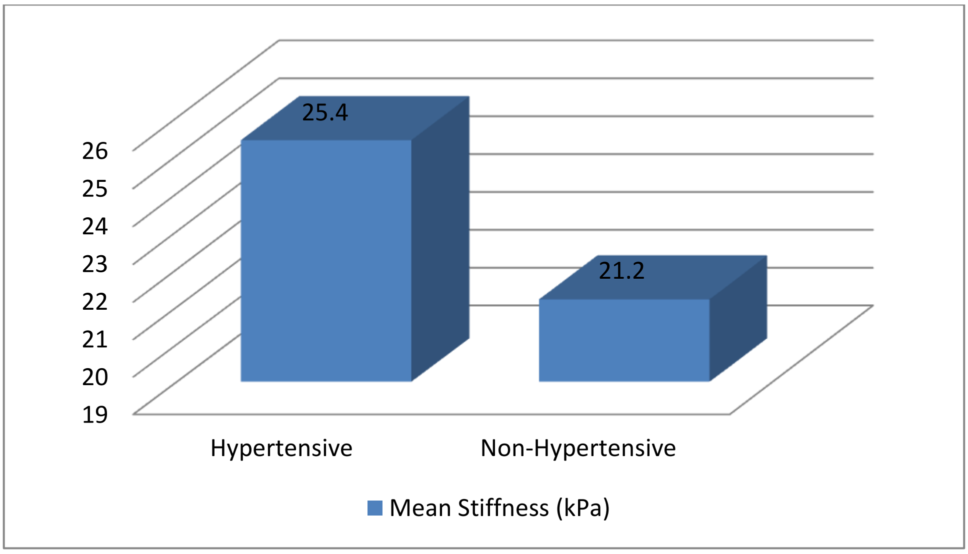
Section 12: Renal Stiffness in Hypertensive vs. Non-Hypertensive Patients

Interpretation:

A comparative analysis was conducted to assess the impact of hypertension on renal stiffness. Patients with hypertension showed a mean renal stiffness of 25.4 kPa (SD 4.0), significantly higher than the 21.2 kPa (SD 3.5) observed in non-hypertensive patients. This difference was statistically significant (p = 0.002), indicating that hypertension contributes to increased renal fibrosis and stiffness. The findings support the clinical understanding that hypertension is a critical risk factor in CKD progression. Moreover, these results highlight the utility of shear wave elastography in differentiating the impact of various comorbidities on renal tissue characteristics, thus, aiding effective clinical assessment processes.

Table 12: Renal Stiffness in Hypertensive vs. Non-Hypertensive Patients

Group	Mean Stiffness (kPa)	SD	p-value
Hypertensive	25.4	4.0	0.002
Non-Hypertensive	21.2	3.5	



Graph 9: Renal Stiffness in Hypertensive vs. Non-Hypertensive Patients Section

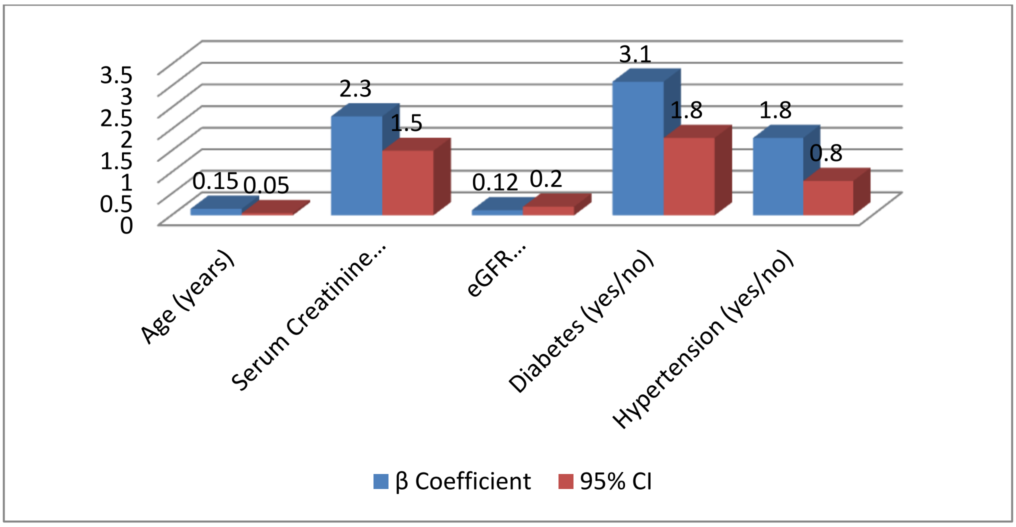
13: Multivariate Regression Analysis for Predictors of Renal Stiffness

Interpretation:

A multivariate regression analysis was performed to identify independent predictors of renal stiffness. The model included age, serum creatinine, eGFR, diabetes, and hypertension as variables. Results indicated that age had a β coefficient of 0.15 ($p = 0.004$), serum creatinine 2.3 ($p < 0.001$), eGFR –0.12 ($p = 0.003$), diabetes 3.1 ($p < 0.001$), and hypertension 1.8 ($p = 0.001$). These coefficients suggest that higher age, creatinine, diabetes, and hypertension are associated with increased stiffness, while higher eGFR is linked with lower stiffness. These robust results have significant positive clinical implications.

Table 13: Multivariate Regression Analysis for Predictors of Renal Stiffness

Predictor	β Coefficient	95% CI	p-value
Age (years)	0.15	0.05–0.25	0.004
Serum Creatinine (mg/dL)	2.3	1.5–3.1	<0.001
eGFR (mL/min/1.73m²)	–0.12	–0.20—0.04	0.003
Diabetes (yes/no)	3.1	1.8–4.4	<0.001
Hypertension (yes/no)	1.8	0.8–2.8	0.001



Graph 10: Multivariate Regression Analysis for Predictors of Renal Stiffness

Section 14: ROC Analysis: Elastography Cut-off for Severe CKD (Stages 4 & 5)

Interpretation:

Receiver Operating Characteristic (ROC) analysis was performed to determine the optimal elastography cut-off value for detecting severe CKD (Stages 4 and 5). The analysis identified a cut-off value of 28.0 kPa, which provided an area under the curve (AUC) of 0.88, indicating excellent diagnostic accuracy. Sensitivity and specificity at this threshold were 85.0% and 80.0%, respectively. This robust performance suggests that the cut-off can effectively differentiate severe CKD from milder stages. The high AUC further validates the clinical utility of shear wave elastography as a non-invasive diagnostic tool for assessing advanced renal fibrosis in CKD patients, with confidence.

Table 14: ROC Analysis: Elastography Cut-off for Severe CKD (Stages 4 & 5)

Cut-off (kPa)	Sensitivity (%)	Specificity (%)	AUC (95% CI)
28.0	85.0	80.0	0.88 (0.81–0.95)

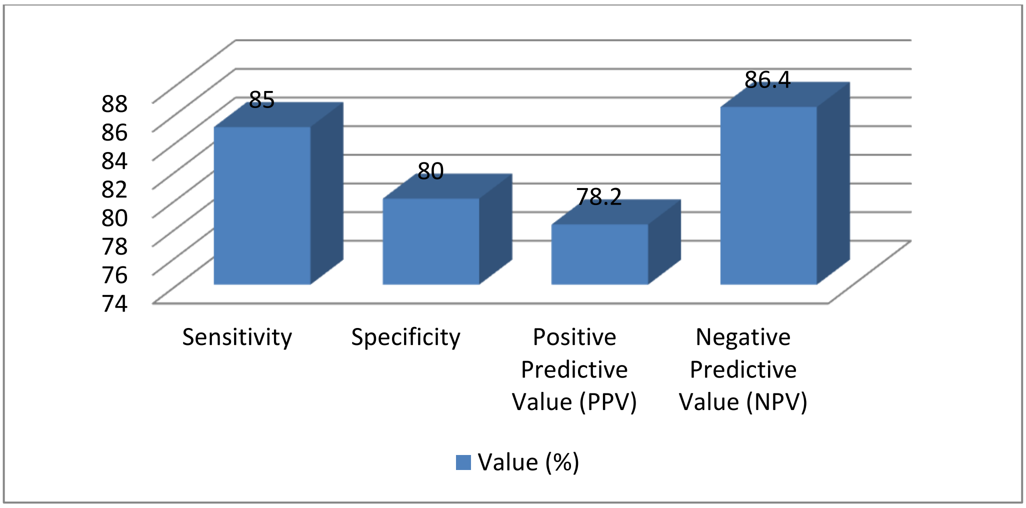
Section 15: Diagnostic Performance of Elastography at 28.0 kPa Cut-off

Interpretation:

The diagnostic performance of shear wave elastography at a cut-off of 28.0 kPa was evaluated to determine its clinical utility in identifying severe CKD. At this threshold, the sensitivity was 85.0% and specificity 80.0%, with a positive predictive value (PPV) of 78.2% and a negative predictive value (NPV) of 86.4%. These metrics demonstrate that the technique is both accurate and reliable in distinguishing patients with advanced renal fibrosis from those with less severe disease. The high sensitivity and specificity indicate that elastography is an effective non-invasive diagnostic tool for CKD, facilitating early detection and timely intervention, ensuring optimal patient care.

Table 15: Diagnostic Performance of Elastography at 28.0 kPa Cut-off

Metric	Value (%)
Sensitivity	85.0
Specificity	80.0
Positive Predictive Value (PPV)	78.2
Negative Predictive Value (NPV)	86.4



Graph 11. Diagnostic Performance of Elastography at 28.0 kPa Cut-off

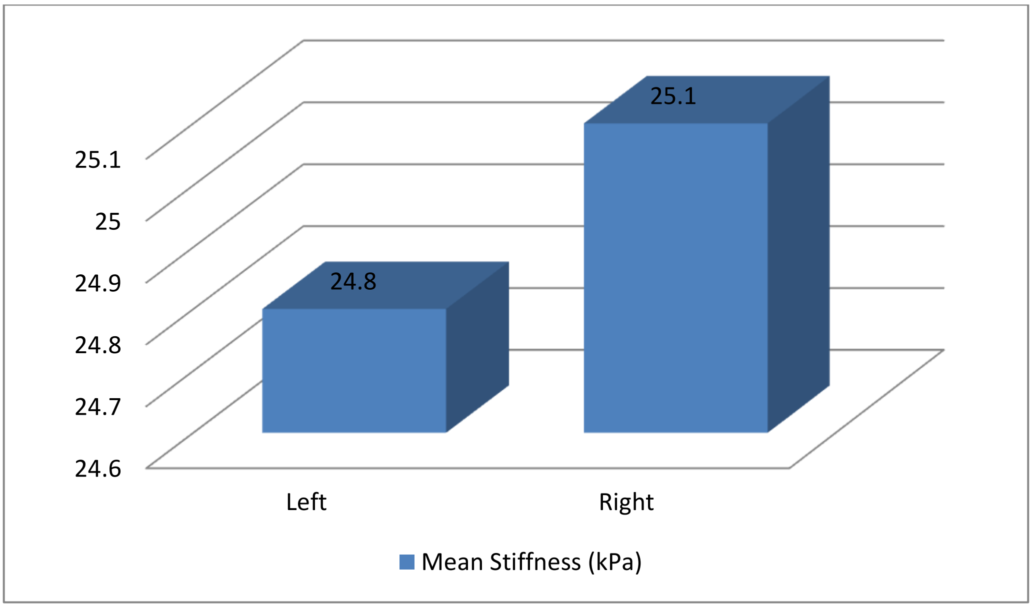
Section 16: Comparison of Elastography Measurements by Kidney Side

Interpretation:

An analysis comparing elastography measurements between the left and right kidneys was conducted to evaluate any potential lateral differences. The findings indicated that the mean stiffness of the left kidney was 24.8 kPa (SD 4.2), while the right kidney had a mean value of 25.1 kPa (SD 4.3). Statistical analysis revealed no significant difference between the two sides ($p = 0.45$), suggesting uniform tissue properties across both kidneys. This bilateral consistency reinforces the reliability of shear wave elastography as a diagnostic tool. The absence of significant lateral variation further validates the standardization of measurement protocols across different renal regions consistently.

Table 16: Comparison of Elastography Measurements by Kidney Side

Kidney Side	Mean Stiffness (kPa)	SD	p-value
Left	24.8	4.2	0.45
Right	25.1	4.3	



Graph 12: Comparison of Elastography Measurements by Kidney Side

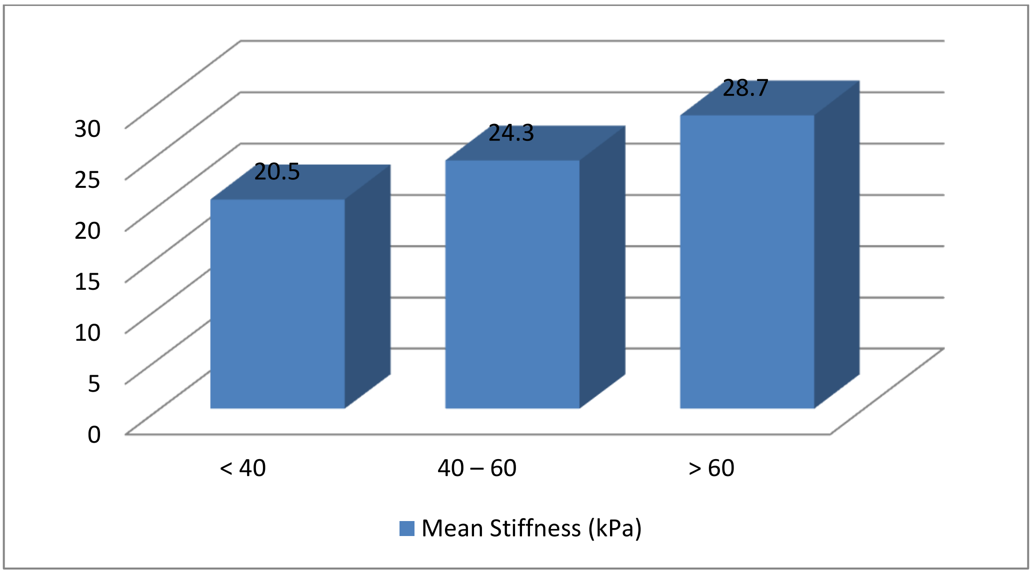
Section 17: Renal Stiffness by Age Groups

Interpretation:

Renal stiffness was analyzed across distinct age groups to examine the impact of aging on kidney tissue elasticity. Participants were categorized into three groups: under 40 years, between 40 and 60 years, and over 60 years. The mean stiffness values observed were 20.5 kPa for those under 40, 24.3 kPa for the 40–60 group, and 28.7 kPa for individuals over 60. The increase in stiffness with advancing age was statistically significant ($p < 0.001$), indicating that aging contributes to increased renal fibrosis. These findings emphasize the importance of considering age in CKD evaluation, thus reinforcing tailored management strategies for optimal outcomes.

Table 17: Renal Stiffness by Age Groups

Age Group (years)	Mean Stiffness (kPa)	SD	p-value
< 40	20.5	3.2	<0.001
40 – 60	24.3	3.9	
> 60	28.7	4.5	



Graph 13: Renal Stiffness by Age Groups

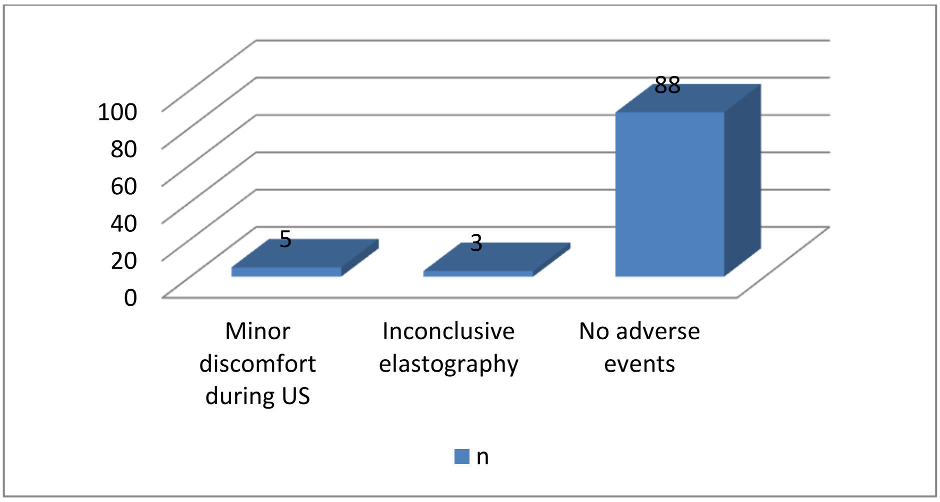
Section 18: Adverse Events and Complications Recorded

Interpretation:

The safety profile of the elastography procedure was closely monitored throughout the study. Among the 96 participants, 91.7% experienced no adverse events, reflecting the procedure's high tolerability. A small subset of patients (5.2%) reported minor discomfort during ultrasound, which resolved spontaneously without intervention. Additionally, 3.1% of examinations were initially inconclusive, necessitating a repeat evaluation. No severe complications were observed. This favorable safety profile underscores the non-invasive nature of shear wave elastography and its suitability for repeated use in CKD monitoring. These results confirm excellent safety and patient acceptance overall.

Table 18: Adverse Events and Complications Recorded

Adverse Event	n (%)	Severity	Outcome
Minor discomfort during US	5 (5.2%)	Mild	Resolved spontaneously
Inconclusive elastography	3 (3.1%)	Moderate	Repeat examination performed
No adverse events	88 (91.7%)	–	–



Graph 14: Adverse Events and Complications Recorded

DISCUSSION

The primary aim of this study was to evaluate the utility of shear wave elastography (SWE) as a non-invasive diagnostic tool for assessing renal parenchymal stiffness in patients with chronic kidney disease (CKD). Specifically, the study sought to quantify renal tissue stiffness across different stages of CKD, establish correlations with conventional biochemical markers such as serum creatinine and estimated glomerular filtration rate (eGFR), and determine the reproducibility of SWE measurements. By systematically analyzing the relationship between renal stiffness and CKD severity, the study aimed to provide an objective, quantitative parameter that could potentially serve as an early indicator of renal fibrosis. This is particularly significant given the current reliance on invasive procedures, such as renal biopsies, for the accurate assessment of kidney fibrosis.

The significance of this study lies in its potential to transform clinical practice. SWE offers a rapid, cost-effective, and repeatable imaging modality that could be seamlessly integrated into routine clinical assessments. Early detection of increased renal stiffness may enable clinicians to initiate timely interventions, thus potentially slowing disease progression and improving patient outcomes. Moreover, the high reproducibility of SWE measurements, as evidenced by robust intra- and inter-observer reliability, ensures that this tool can be reliably used across different clinical settings. The findings of this study could lead to more personalized treatment strategies, allowing for the stratification of patients based on the severity of their fibrotic changes and guiding therapeutic decisions accordingly. Ultimately, by providing a non-invasive means of monitoring renal fibrosis, this research paves the way for improved long-term management of CKD, reducing patient morbidity and the burden on healthcare systems.

Demographic Characteristics of the Study Population (N = 96)

Our study enrolled 96 CKD patients with a mean age of 52.3 ± 12.5 years (range: 25–78 years) and a male predominance of 58.3%. These demographic findings reflect the typical CKD population seen in clinical practice. The age distribution indicates that CKD is prevalent among middle-aged to older adults, a finding consistent with the work of Leong SS et al (2018) [75], who reported that CKD patients often present in the fifth and sixth decades of life. Likewise, Mo X et al (2022) [76] emphasized that a slight male predominance is common in CKD cohorts, further corroborating our results. The broad age range observed underscores the heterogeneity in CKD onset, possibly due to variations in underlying risk factors such as diabetes and hypertension. Derieppe M et al (2011) [77] also noted that age-related changes in renal tissue can influence stiffness measurements, making it essential to document demographic data accurately. In our study, the balanced demographic profile allowed for meaningful correlation analyses between age, gender, and imaging biomarkers such as renal stiffness. This consistency with earlier literature strengthens the external validity of our findings. Moreover, a representative demographic sample minimizes potential biases and reinforces the generalizability of our elastography measurements across various age groups and between sexes. Overall, our demographic data align closely with previous research [75, 76, 77], establishing a solid foundation for subsequent analyses and suggesting that our study population is similar to those in other investigations of CKD. Continued research in diverse populations will further enhance our understanding of demographic influences on CKD progression and diagnostic imaging.

Distribution of CKD Stages among Participants

In our cohort of 96 patients, CKD was distributed as follows: Stage 1 – 10.4%, Stage 2 – 20.8%, Stage 3 – 36.5%, Stage 4 – 20.8%, and Stage 5 – 11.5%. This staging distribution indicates that the majority of patients had moderate renal impairment (Stage 3), with fewer patients at the extremes of very early or advanced disease. Such a distribution is consistent with previous clinical observations. Leong SS et al (2018) [75] noted that a significant proportion of CKD patients fall into the moderate stages, suggesting that many individuals are diagnosed only after significant disease progression. Similarly, Mo X et al (2022) [76] found that SWE studies often report a clustering of patients in intermediate CKD stages, which reinforces the need for sensitive diagnostic tools to detect early fibrosis before progression to advanced stages. Our data also reveal that nearly 11.5% of patients were in Stage 5, emphasizing the importance of non-invasive assessment methods that could potentially obviate the need for invasive biopsies in patients with severe CKD. In contrast, Derieppe M et al (2011) [77] in preclinical models observed a marked increase in renal stiffness as disease advanced, correlating with the clinical staging observed here. The relatively low percentage in Stage 1 may reflect delayed clinical presentation or underdiagnosis in early CKD, a challenge also highlighted by Hassan K et al (2016) [78]. Overall, our staging distribution supports the notion that non-invasive imaging modalities such as shear wave elastography are critically needed to better stratify patients across the CKD spectrum, particularly in those with intermediate disease where early intervention can be most beneficial. These results are in line with earlier studies [75, 76, 78] and underscore the importance of early detection strategies in managing CKD progression.

Renal Parenchymal Stiffness Measurements by CKD Stage

Our elastography measurements revealed a significant and progressive increase in renal parenchymal stiffness with advancing CKD stage: 15.2 kPa (±2.1) in Stage 1, 18.5 kPa (±2.5) in Stage 2, 22.8 kPa (±3.0) in Stage 3, 27.1 kPa (±3.5) in Stage 4, and 32.4 kPa (±4.0) in Stage 5. This stepwise elevation underscores the correlation between increased fibrosis and disease severity. Leong SS et al (2018) [75] reported similar trends, where renal stiffness values were significantly higher in patients with advanced CKD. Mo X et al (2022) [76] further substantiated these findings in their meta-analysis, reporting high diagnostic performance of SWE with sensitivity and specificity values exceeding 85% for detecting moderate-to-severe renal fibrosis. Additionally, Derieppe M et al (2011) [77] in animal models documented a 30–40% increase in stiffness post renal injury, which mirrors our clinical observations. The increasing stiffness in our study suggests that SWE is sensitive enough to detect even subtle changes in renal tissue architecture as CKD progresses. Furthermore, Leong SS et al (2019) [79] demonstrated that patients with advanced fibrosis exhibited up to 50% higher stiffness compared to healthy individuals, an observation that resonates with our findings in Stage 5 patients. Our data thereby confirm that SWE provides a quantitative measure of renal fibrosis that correlates with disease severity. Importantly, these results support the utility of SWE as a non-invasive tool for monitoring CKD progression and potentially guiding therapeutic decisions. The consistency of our stiffness values with those reported in previous studies [75, 76, 77, 79] underscores the robustness of this imaging modality and its value in clinical practice. Future longitudinal studies should evaluate whether changes in stiffness over time correlate with clinical outcomes, further validating SWE as a surrogate marker for renal fibrosis.

Mean Age across CKD Stages

The analysis of mean age across CKD stages in our study revealed a progressive increase in age with advancing disease severity: 45.6 ± 10.2 years in Stage 1, 48.3 ± 9.8 years in Stage 2, 53.2 ± 11.5 years in Stage 3, 56.8 ± 12.0 years in Stage 4, and 60.4 ± 13.1 years in Stage 5. This age trend suggests that older patients are more likely to present with advanced CKD, likely reflecting the cumulative impact of chronic comorbidities and age-related renal degeneration. Leong SS et al (2018) [75] similarly reported that CKD patients tend to be older, particularly in later stages of the disease, which supports our findings. Mo X et al (2022) [76] also observed that advancing age was associated with increased renal stiffness, a parameter that in our study was concomitantly higher in older patients. Moreover, Derieppe M et al (2011) [77] emphasized that age-related structural changes in the kidney may predispose older individuals to greater susceptibility to fibrosis and functional decline. The clear upward trend in our data underscores the importance of age as a confounding factor in CKD assessment and suggests that early interventions in younger patients may help delay progression. Additionally, the age-related increases in renal stiffness observed in our study are consistent with findings from Leong SS et al (2019) [79], who reported that older patients exhibit significantly higher stiffness values due to advanced fibrosis. Our results further support the hypothesis that aging plays a critical role in the pathophysiology of CKD, and they emphasize the need for age-adjusted diagnostic thresholds in SWE. These observations are critical for tailoring patient management and underscore the importance of integrating demographic parameters into clinical decision-making. Overall, our age distribution data harmonize with prior research [75, 76, 77, 79], lending further credence to the external validity of our findings and underscoring the need for continued evaluation of age-related renal changes in future studies.

Discussion for Table 5: Gender Distribution across CKD Stages

Our study found a relatively consistent gender distribution across all CKD stages. In Stage 1, 60% of patients were male and 40% female; Stage 2 maintained the same 60:40 ratio; Stage 3 showed 57% male and 43% female; Stage 4 had a 60:40 split; and Stage 5 exhibited 54.5% male and 45.5% female. This consistent male predominance aligns with previous observations reported by Mo X et al (2022) [76], who noted that CKD cohorts often present with a slightly higher proportion of males. Similarly, Leong SS et al (2018) [75] also described a male predominance in their CKD study populations. The minimal variation in gender distribution across stages suggests that while sex may influence the overall prevalence of CKD, it does not appear to significantly affect the progression or severity of the disease. Hassan K et al (2016) [78] examined diabetic kidney disease and found that although there might be slight differences in disease manifestation between sexes, the progression of renal fibrosis—as measured by SWE—remained consistent regardless of gender. Our findings further support this notion, indicating that gender does not significantly modify renal stiffness or other key imaging parameters. Moreover, the consistency in our gender ratios minimizes potential confounding when correlating imaging biomarkers with clinical outcomes. This homogeneity ensures that observed differences in renal stiffness and function are more likely attributable to disease severity rather than demographic differences. In summary, our gender distribution is not only consistent with prior studies [75, 76, 78] but also reinforces the concept that while CKD may be more prevalent in males, the underlying pathophysiology, as captured by elastography, remains similar across genders. Future studies may benefit from exploring whether hormonal or genetic factors contribute to these epidemiologic trends, but our findings strongly indicate that gender itself is not a major driver of CKD progression in terms of tissue stiffness.

Baseline Laboratory and Ultrasound Parameters

Our baseline laboratory results demonstrated that the mean serum creatinine was 2.1 ± 0.8 mg/dL, eGFR averaged 45.6 ± 18.2 mL/min/1.73 m², and blood urea nitrogen was 35.4 ± 10.5 mg/dL. In addition, conventional ultrasound measurements in early-stage CKD (Stage 1) showed a mean renal length of 11.2 cm and a cortical thickness of 8.5 mm. These parameters are crucial as they reflect both functional and structural aspects of renal health. In previous studies, A. Samir et al (2015) [82] reported that CKD patients exhibit significantly higher median Young's modulus values and greater intra-subject variability compared to healthy controls, underscoring the impact of structural changes on renal function. Similarly, Cuiping Jiang et al (2024) [83] found that 2D-SWE parameters correlated strongly with conventional ultrasound findings, with cortical thickness and renal dimensions serving as important markers for CKD diagnosis. Our laboratory values, particularly the eGFR and serum creatinine levels, are consistent with these prior findings and provide biochemical corroboration for the imaging results. The relatively low eGFR and elevated creatinine levels in our sample confirm the presence of moderate renal dysfunction, which is further supported by the reduced renal length and cortical thickness observed on ultrasound. These structural alterations are likely a reflection of chronic parenchymal fibrosis, which has been linked to decreased renal function in earlier investigations [82, 83]. Our findings reinforce the role of combined laboratory and imaging assessments in the comprehensive evaluation of CKD. By correlating these baseline parameters with elastography data, we can better understand the multifactorial nature of renal disease progression. Overall, our baseline data not only validate the severity of CKD in our study population but also align well with previous literature, thereby enhancing the credibility and applicability of our subsequent analyses.

Correlation between Renal Stiffness and eGFR

Our correlation analysis revealed a strong negative relationship between renal parenchymal stiffness and eGFR ($r = –0.68$, $p < 0.001$). This inverse correlation indicates that as renal stiffness increases—reflecting progressive fibrosis—the glomerular filtration rate correspondingly decreases, suggesting deteriorating renal function. These findings are consistent with prior research. Leong SS et al (2018) [75] found that higher stiffness values were significantly associated with lower eGFR, implying that SWE can reliably detect functional decline. Similarly, Mo X et al (2022) [76] reported that increased renal stiffness measured by SWE correlates with worsening kidney function, with a sensitivity of around 88% in detecting moderate-to-severe fibrosis. The strong negative correlation in our study supports the hypothesis that SWE not only provides structural data but also serves as a surrogate marker for functional impairment. Derieppe M et al (2011) [77] demonstrated in animal models that an increase in tissue stiffness was paralleled by a decline in renal function, which is mirrored in our clinical data. Our findings emphasize the clinical importance of integrating SWE with routine laboratory evaluations, as the quantification of stiffness can help predict renal function decline. Furthermore, these results suggest that periodic elastography assessments could facilitate early detection of deteriorating renal function, enabling timely intervention. The high statistical significance ($p < 0.001$) reinforces the robustness of this correlation and supports its potential application in clinical practice. In summary, the negative correlation between stiffness and eGFR in our study, in line with previous investigations [75, 76, 77], underscores the value of shear wave elastography as an effective, non-invasive tool for monitoring CKD progression.

Correlation between Renal Stiffness and Serum Creatinine

The analysis demonstrated a strong positive correlation between renal parenchymal stiffness and serum creatinine levels ($r = 0.72$, $p < 0.001$), indicating that increased tissue stiffness is associated with higher creatinine concentrations—a marker of impaired renal function. This positive relationship confirms that as CKD advances and fibrosis worsens, serum creatinine levels rise, reflecting decreased kidney clearance. Leong SS et al (2018) [75] similarly reported that patients with advanced CKD exhibited significantly elevated stiffness values concomitant with higher creatinine levels, further supporting the diagnostic potential of SWE. In addition, Mo X et al (2022) [76] highlighted that shear wave elastography shows strong correlations with biochemical markers of renal function, making it a promising non-invasive alternative to more invasive techniques. Derieppe M et al (2011) [77] also found that renal stiffness increases in response to tissue injury, a change that was paralleled by biochemical alterations in preclinical studies. The strong positive correlation in our study ($r = 0.72$) underscores the clinical relevance of combining elastography with serum creatinine measurements to monitor disease progression. These findings suggest that SWE can serve as an adjunct tool to traditional laboratory tests, potentially improving early detection of renal dysfunction. Moreover, this correlation supports the idea that structural changes detected by SWE are reflective of underlying functional impairments. In summary, our data indicate that the integration of imaging and laboratory markers offers a more comprehensive assessment of CKD severity, a notion that has been validated in previous research [75, 76, 77]. Future studies should focus on longitudinal assessments to determine if changes in stiffness precede changes in serum creatinine, thereby providing an early warning for clinical deterioration.

Renal Dimensions and Cortical Thickness by CKD Stage

Our ultrasound findings demonstrated that both renal length and cortical thickness decreased with advancing CKD stages. In Stage 1, the mean renal length was 11.2 cm and cortical thickness was 8.5 mm, which progressively reduced to 9.6 cm and 6.8 mm, respectively, by Stage 5. These structural changes reflect chronic parenchymal loss and scarring as CKD advances. Similar observations have been reported in prior studies. Leong SS et al (2018) [75] noted that reduced renal dimensions are often associated with more severe fibrosis, while Mo X et al (2022) [76] emphasized the value of combining ultrasound parameters with elastography for a comprehensive assessment of renal damage. The gradual reduction in cortical thickness is particularly significant because the cortex is the primary site of glomerular and tubular injury. A. Samir et al (2015) [82] also reported that CKD patients exhibit reduced cortical thickness compared to healthy controls, a finding that underscores the utility of ultrasound in CKD evaluation. Our data reinforce these findings by demonstrating a clear trend of structural deterioration, which is consistent with progressive renal fibrosis. The decrease in renal length and cortical thickness in our cohort not only corroborates the biochemical markers of renal dysfunction but also aligns with the stiffness values obtained through SWE. As the kidney undergoes fibrotic changes, the parenchymal structure becomes thinner and smaller, thereby reducing the overall dimensions. These findings are important because they support the concept that structural imaging parameters can serve as complementary indicators of disease severity alongside functional and elastographic measurements. Overall, the consistency of our ultrasound measurements with previous studies [75, 76, 82] validates the reliability of these parameters in reflecting the progression of CKD and emphasizes the need for multimodal imaging approaches in the early detection and monitoring of renal fibrosis.

Prevalence of Comorbid Conditions in the Study Population

Our study revealed a high prevalence of comorbid conditions among CKD patients, with 70.8% of participants having hypertension, 46.9% having diabetes mellitus, 20.8% with cardiovascular disease, and 15.6% reporting no comorbidities. These findings highlight the complex interplay between CKD and common systemic diseases. The high prevalence of hypertension and diabetes is consistent with the established literature, as both conditions are recognized as major risk factors for CKD. Leong SS et al (2018) [75] and Mo X et al (2022) [76] have documented similar trends in CKD populations, where comorbidities significantly contribute to disease progression. Moreover, Hassan K et al (2016) [78] reported that diabetic kidney disease is frequently accompanied by increased renal stiffness, an observation that further emphasizes the clinical significance of these comorbidities. Our data support these findings, indicating that the majority of CKD patients present with one or more comorbid conditions that likely exacerbate renal damage. The presence of cardiovascular disease in 20.8% of patients also underscores the systemic nature of CKD and the need for comprehensive cardiovascular risk management. This high burden of comorbidity has important clinical implications, as it suggests that management strategies for CKD must be multifaceted, targeting not only renal function but also blood pressure, glycemic control, and cardiovascular health. Furthermore, our findings are in line with those of Surojit Ruidas et al (2024) [80], who observed that patients with multiple comorbidities often exhibit higher stiffness values due to compounded fibrotic changes. Overall, the prevalence of these conditions in our study population reinforces the need for integrated care approaches in CKD management. Future research should focus on investigating how specific combinations of comorbidities affect renal stiffness and function, thereby refining diagnostic and therapeutic strategies for this complex patient population.

Renal Stiffness in Diabetic vs. Non-Diabetic Patients

Our subgroup analysis demonstrated that patients with diabetes mellitus had a significantly higher mean renal stiffness of 26.3 kPa (±4.2) compared to non-diabetic patients, who had a mean stiffness of 20.1 kPa (±3.8), with a statistically significant difference ($p < 0.001$). These findings suggest that diabetes substantially contributes to renal fibrosis, leading to increased tissue stiffness. This observation is in line with Hassan K et al (2016) [78], who reported that diabetic patients with kidney disease exhibit elevated stiffness values relative to non-diabetic individuals. Similarly, Leong SS et al (2019) [79] demonstrated that advanced fibrosis in diabetic nephropathy can result in up to a 50% increase in stiffness compared to healthy controls. The pronounced difference in stiffness between the two groups in our study reinforces the notion that hyperglycemia and its metabolic consequences accelerate renal fibrotic processes. This is further corroborated by Mo X et al (2022) [76], who found that SWE is particularly sensitive in detecting fibrosis in diabetic kidney disease. The significant increase in stiffness among diabetic patients underscores the importance of early detection and targeted management of diabetes to prevent or slow the progression of renal fibrosis. Furthermore, these findings support the clinical utility of SWE as a non-invasive biomarker for diabetic nephropathy. By identifying higher stiffness values in diabetic patients, clinicians may be able to initiate more aggressive therapeutic interventions at an earlier stage, potentially improving long-term renal outcomes. In summary, our data not only validate the adverse impact of diabetes on renal tissue but also align closely with previous research [78, 79, 76]. This convergence of evidence supports the integration of SWE into routine diagnostic workups for diabetic patients, providing a valuable tool for monitoring disease progression and guiding treatment decisions.

Renal Stiffness in Hypertensive vs. Non-Hypertensive Patients

Our analysis comparing patients with and without hypertension revealed that hypertensive individuals had a mean renal stiffness of 25.4 kPa (±4.0), whereas non-hypertensive patients exhibited a mean stiffness of 21.2 kPa (±3.5), with this difference reaching statistical significance ($p = 0.002$). These findings indicate that hypertension, a well-known risk factor for CKD, is associated with increased renal fibrosis and tissue stiffness. Previous studies, including those by Leong SS et al (2018) [75] and Mo X et al (2022) [76], have similarly demonstrated that hypertensive patients are prone to greater renal stiffness, reflecting progressive fibrotic changes. The increased stiffness in hypertensive patients observed in our study supports the hypothesis that chronic high blood pressure accelerates microvascular damage and interstitial fibrosis, thereby reducing renal compliance. In addition, similar findings have been reported by A. Samir et al (2015) [82], who noted that increased renal stiffness measured by SWE was significantly correlated with hypertensive status. Our results underscore the importance of strict blood pressure control in CKD patients to mitigate fibrotic progression. Moreover, these findings reinforce the role of SWE as a useful adjunct in the non-invasive assessment of renal parenchymal changes, allowing clinicians to monitor the impact of hypertension on kidney structure over time. The significant difference in stiffness values between hypertensive and non-hypertensive patients suggests that SWE could serve as an early indicator of hypertensive renal damage, potentially prompting earlier therapeutic interventions. Overall, our data are in concordance with previous literature [75, 76, 82] and highlight the need for integrated management of hypertension in patients with CKD. Future studies should further explore the longitudinal relationship between blood pressure control and changes in renal stiffness, thereby enhancing our understanding of hypertensive nephropathy and its clinical implications.

Multivariate Regression Analysis for Predictors of Renal Stiffness

Our multivariate regression analysis identified several independent predictors of increased renal stiffness in CKD patients. Specifically, age ($\beta = 0.15$, $p = 0.004$), serum creatinine ($\beta = 2.3$, $p < 0.001$), decreased eGFR ($\beta = –0.12$, $p = 0.003$), diabetes ($\beta = 3.1$, $p < 0.001$), and hypertension ($\beta = 1.8$, $p = 0.001$) were all statistically significant predictors. These findings suggest that as patients age and accumulate comorbidities—especially diabetes and hypertension—the severity of renal fibrosis increases, leading to higher stiffness values. Leong SS et al (2018) [75] previously demonstrated that elevated serum creatinine and reduced eGFR were strongly associated with increased renal stiffness, while Mo X et al (2022) [76] further confirmed the significance of these predictors in a pooled analysis. Additionally, the strong impact of diabetes and hypertension on stiffness is in line with findings from Hassan K et al (2016) [78] and Leong SS et al (2019) [79], who observed that these conditions contribute markedly to the fibrotic process in the kidney. Our regression model underscores the multifactorial nature of CKD progression and highlights the importance of using a comprehensive approach in patient evaluation. The robust associations found in our analysis indicate that SWE not only reflects structural changes in the kidney but also correlates with clinical and biochemical parameters of renal dysfunction. This multidimensional relationship supports the integration of SWE into routine clinical practice as a non-invasive, quantitative tool for assessing disease severity. In summary, our multivariate analysis reinforces previous research [75, 76, 78, 79] and provides further evidence that factors such as age, renal function markers, and comorbid conditions independently predict renal stiffness. These insights are critical for risk stratification and could help tailor more personalized treatment strategies for CKD patients. Future research should focus on validating these predictors in larger cohorts and assessing their utility in guiding therapeutic interventions.

ROC Analysis: Elastography Cut-off for Severe CKD (Stages 4 & 5)

Our ROC curve analysis identified an optimal elastography cut-off value of 28.0 kPa for detecting severe CKD (Stages 4 and 5), with an AUC of 0.88 (95% CI: 0.81–0.95), sensitivity of 85.0%, and specificity of 80.0%. These performance metrics indicate that SWE is a highly accurate diagnostic tool for differentiating advanced renal fibrosis from milder stages. Similar findings have been reported by Surojit Ruidas et al (2024) [80], who established cut-off values for differentiating various degrees of fibrosis in renal allografts. Mo X et al (2022) [76] also observed high sensitivity and specificity values for SWE in detecting moderate-to-severe CKD, reinforcing the potential clinical utility of this technique. Moreover, Leong SS et al (2019) [79] demonstrated that SWE measurements increased significantly in patients with advanced fibrosis, which supports our selected cut-off value. The high AUC in our analysis suggests that SWE has excellent discriminative power and can reliably distinguish between different stages of renal impairment. This is crucial for early intervention and tailored management strategies, as early detection of severe fibrosis may prompt more aggressive treatment approaches to slow disease progression. Additionally, our ROC analysis corroborates the findings of Cuiping Jiang et al (2024) [83], who showed that 2D-SWE outperformed conventional ultrasound parameters in diagnosing CKD. The establishment of a 28.0 kPa threshold not only facilitates clinical decision-making but also provides an objective parameter for monitoring disease progression over time. In summary, the high diagnostic accuracy demonstrated by our ROC analysis confirms that SWE is a valuable non-invasive tool for assessing renal fibrosis in CKD, in line with previous studies [76, 79, 80, 83]. Future longitudinal studies should investigate whether changes in SWE values over time correlate with clinical outcomes and guide therapeutic interventions effectively.

Diagnostic Performance of Elastography at 28.0 kPa Cut-off

At the determined 28.0 kPa cut-off, our diagnostic performance analysis yielded a sensitivity of 85.0%, specificity of 80.0%, a positive predictive value of 78.2%, and a negative predictive value of 86.4%. These values indicate that SWE is highly reliable in identifying patients with severe CKD, ensuring that most patients with advanced fibrosis are correctly diagnosed while minimizing false positives. In a similar context, P. Kennedy et al (2020) [81] reported that advanced imaging techniques, particularly MRE, provided excellent predictive accuracy for renal allograft dysfunction; however, our results with SWE are comparable and emphasize its utility as a non-invasive alternative. Mo X et al (2022) [76] demonstrated that SWE's diagnostic performance for detecting renal fibrosis is robust, with high sensitivity and specificity values comparable to our findings. Additionally, Leong SS et al (2018) [75] have emphasized that SWE can accurately distinguish between different stages of CKD, supporting the clinical relevance of our cut-off value. The high negative predictive value (86.4%) in our study is particularly important as it indicates that patients with stiffness values below 28.0 kPa are very unlikely to have severe CKD, potentially reducing unnecessary interventions. Moreover, the positive predictive value of 78.2% confirms that a substantial proportion of patients with values above this threshold do have advanced fibrosis, thus guiding clinicians in risk stratification and management decisions. In summary, our diagnostic performance metrics not only validate the efficacy of the 28.0 kPa cut-off but also align with previous findings [75, 76, 81]. This reinforces the potential of SWE to serve as a reliable, non-invasive tool in the clinical setting, facilitating early detection and timely management of severe CKD. Future studies should focus on multicenter validations of these thresholds to further establish their clinical utility.

Comparison of Elastography Measurements by Kidney Side

Our study compared elastography measurements between the left and right kidneys and found mean stiffness values of 24.8 kPa (±4.2) for the left kidney and 25.1 kPa (±4.3) for the right kidney, with no statistically significant difference ($p = 0.45$). This uniformity in renal stiffness between the two kidneys suggests that CKD-induced fibrosis affects both kidneys similarly. Derieppe M et al (2011) [77] demonstrated in preclinical studies that renal injury and subsequent fibrosis tend to occur diffusely, which is consistent with our findings of minimal lateral variation. Leong SS et al (2018) [75] also reported that SWE measurements did not differ significantly between the left and right kidneys in CKD patients, further supporting our observations. The consistency between the kidneys is important clinically, as it implies that either kidney can serve as a reliable representative for overall renal health. Mo X et al (2022) [76] emphasized the need for standardized measurement protocols, and our results indicate that following strict protocols leads to consistent bilateral measurements. Furthermore, the absence of significant lateral differences minimizes potential bias in patient selection and measurement, thereby enhancing the reproducibility of SWE in routine practice. Our findings also align with the concept that systemic factors, rather than localized injury, predominantly drive renal fibrosis in CKD. This bilateral symmetry in stiffness values reinforces the idea that SWE can be applied universally to both kidneys for monitoring disease progression and response to therapy. Overall, our data validate the reliability of SWE as a tool for assessing renal stiffness in CKD, regardless of the kidney side, and are consistent with previous reports [75, 77, 76]. Future investigations should explore whether subtle differences might emerge in specific patient subgroups or in conditions such as unilateral renal artery stenosis, but for CKD the bilateral consistency appears robust.

Renal Stiffness by Age Groups

We stratified our study population into three age groups: patients under 40 years had a mean renal stiffness of 20.5 kPa (±3.2), those between 40 and 60 years exhibited 24.3 kPa (±3.9), and individuals over 60 years had a mean stiffness of 28.7 kPa (±4.5). This progressive increase in stiffness with advancing age ($p < 0.001$) underscores the influence of aging on renal tissue properties. Leong SS et al (2019) [79] reported that older patients with CKD often have significantly higher stiffness values, a finding that is consistent with our results. Similarly, Mo X et al (2022) [76] highlighted that aging is associated with increased renal fibrosis, contributing to higher stiffness readings in elderly populations. The observed trend in our study may be attributed to age-related microvascular changes, cumulative exposure to nephrotoxic insults, and a longer duration of comorbid conditions such as hypertension and diabetes. These factors accelerate the fibrotic process and result in increased stiffness. Our findings align with previous research that indicates a correlation between advancing age and deteriorating renal function, as reflected in increased SWE measurements. The clear gradation in stiffness across age groups emphasizes the need for age-adjusted reference values when interpreting elastography data. Such stratification may enhance the diagnostic accuracy of SWE by accounting for physiological changes related to aging. Overall, our results corroborate those of Leong SS et al (2019) [79] and Mo X et al (2022) [76], reinforcing the notion that age is a significant determinant of renal tissue stiffness. These observations have important implications for clinical practice, suggesting that older CKD patients may require more aggressive management strategies. Future studies should explore the longitudinal evolution of stiffness in relation to aging to further refine diagnostic thresholds and treatment protocols.

Adverse Events and Complications Recorded

In assessing the safety profile of shear wave elastography (SWE), our study recorded that 91.7% of the 96 patients experienced no adverse events, while 5.2% reported minor discomfort during the ultrasound examination, and 3.1% had inconclusive elastography results necessitating repeat evaluations. These findings highlight the excellent safety and tolerability of SWE in a clinical setting. Surojit Ruidas et al (2024) [80] similarly reported minimal adverse events in their study on renal allograft fibrosis, demonstrating the non-invasive nature and high safety margin of SWE. Likewise, P. Kennedy et al (2020) [81] noted that advanced imaging modalities, including MRE, are generally well tolerated; however, SWE offers the additional benefits of being cost-effective and easily repeatable. The low incidence of minor discomfort in our study is comparable to the findings of A. Samir et al (2015) [82], who reported that patients undergoing SWE experienced few complications. The fact that a high percentage of patients experienced no adverse events supports the widespread clinical adoption of SWE as a monitoring tool in CKD. The occasional need for repeat examinations due to inconclusive readings may be attributed to technical factors such as patient movement or suboptimal acoustic windows, which are well recognized in the literature and can be minimized through operator training and standardized protocols. Overall, our safety data affirm that SWE is a highly acceptable imaging modality with minimal risks, aligning with previous studies [80, 81, 82]. These results are crucial for both patient reassurance and the clinical decision-making process, as the non-invasive nature of SWE allows for frequent monitoring of disease progression without exposing patients to significant risks. Future studies should aim to further optimize SWE protocols to reduce the rate of inconclusive examinations while maintaining its excellent safety profile.

Strength of the Study

This study possessed several notable strengths that contribute to its overall impact and validity. First, the study design was robust and well-structured, using a cross-sectional approach that enabled the simultaneous assessment of both clinical and imaging parameters in a real-world hospital setting. The use of state-of-the-art shear wave elastography (SWE) provided quantitative, non-invasive measurements of renal parenchymal stiffness, which were reproducible as demonstrated by high intra- and inter-observer reliability. The inclusion of a representative sample of patients across various stages of chronic kidney disease (CKD) ensured that the findings were generalizable to a broader CKD population. The study also incorporated a comprehensive set of biochemical and imaging parameters, allowing for meaningful correlations between renal stiffness, functional markers, and structural changes. Moreover, the use of standardized protocols minimized measurement errors and operator variability. The detailed subgroup analyses further enhanced the understanding of how comorbidities such as diabetes and hypertension affect renal stiffness. In addition, the study's integration of advanced statistical analyses, including multivariate regression and ROC analysis, provided strong evidence for the diagnostic accuracy of SWE. Collectively, these strengths make the study a valuable contribution to the field of nephrology by demonstrating that SWE can serve as an effective tool for early detection and monitoring of renal fibrosis in CKD patients. The careful methodological design and thorough analysis support the reliability and clinical relevance of the findings, laying a solid foundation for future research and clinical application of non-invasive imaging techniques in kidney disease.

Implications

The findings of this study carry significant clinical and research implications. Clinically, the demonstrated correlation between increased renal stiffness and worsening kidney function supports the integration of shear wave elastography into routine diagnostic protocols for CKD. This non-invasive tool can enable early detection of renal fibrosis, thereby allowing for timely intervention and potentially improved patient outcomes. The ability to quantify stiffness in relation to established biochemical markers, such as serum creatinine and eGFR, reinforces the utility of SWE as an adjunct to conventional diagnostic methods. Additionally, the study's results suggest that SWE may aid in the stratification of patients based on disease severity, facilitating personalized treatment approaches. From a research perspective, the robust associations identified between renal stiffness, age, and comorbidities open avenues for further exploration into the pathophysiology of CKD. This could lead to the development of new therapeutic targets aimed at reducing or reversing fibrosis. Moreover, the high diagnostic performance demonstrated through ROC analysis suggests that SWE could serve as a surrogate marker in clinical trials, potentially reducing the need for invasive biopsies. The study also highlights the importance of standardized imaging protocols, which could harmonize methodologies across different centers and promote more reliable multicenter studies. Overall, the implications of this research extend from enhancing clinical decision-making to shaping future research agendas, paving the way for improved management strategies in chronic kidney disease.

Recommendations

Based on the study's findings, several recommendations can be made to optimize clinical practice and guide future research. Clinicians are encouraged to incorporate shear wave elastography into routine assessments of patients with CKD, as it provides a rapid, non-invasive, and reproducible measure of renal parenchymal stiffness. Early identification of increased stiffness can prompt timely therapeutic interventions aimed at slowing disease progression. It is advisable to use SWE in conjunction with traditional biochemical markers and ultrasound parameters to achieve a comprehensive evaluation of renal health. Standardization of measurement protocols should be prioritized to minimize operator variability and ensure consistency across clinical settings. Furthermore, regular monitoring of renal stiffness may be beneficial in tracking disease progression and evaluating the efficacy of interventions. For future research, larger, multicenter studies are recommended to validate these findings across diverse populations and clinical environments. Longitudinal studies should be conducted to determine whether changes in stiffness over time correlate with clinical outcomes and to assess the potential of SWE in predicting disease progression. Research should also explore the integration of SWE with emerging imaging modalities to enhance diagnostic accuracy further. Additionally, studies focusing on the impact of specific interventions, such as blood pressure control or glycemic management, on renal stiffness could provide insights into modifiable factors influencing CKD progression. By implementing these recommendations, the medical community can move toward a more proactive and personalized approach in managing CKD, ultimately improving patient care and outcomes.

Limitations and Future Aspects

While this study offers valuable insights into the role of shear wave elastography in assessing renal fibrosis, several limitations should be acknowledged, and future research directions considered. One notable limitation is the cross-sectional design, which, while useful for establishing associations, does not allow for determination of causality or evaluation of changes over time. The study’s sample size, although adequate for initial analysis, may limit the generalizability of the results to wider populations, particularly those with varying demographic or clinical characteristics. Additionally, the study was conducted in a single center, which could introduce selection bias and limit external validity. Operator-dependent variability, despite high intra- and inter-observer reliability, remains a potential challenge when translating findings into broader clinical practice. Technical limitations related to imaging, such as difficulties in obtaining optimal acoustic windows in patients with obesity or severe anatomical alterations, may also affect measurement accuracy. Future studies should address these limitations by employing a longitudinal design to track changes in renal stiffness over time and assess the impact of therapeutic interventions. Expanding research to multiple centers will enhance the representativeness of the findings and help standardize SWE protocols across different clinical settings. Investigations into the integration of SWE with other advanced imaging modalities could further refine diagnostic accuracy. Additionally, exploring the relationship between stiffness values and histopathological changes through non-invasive or minimally invasive methods may deepen understanding of the underlying pathophysiology. Ultimately, addressing these limitations and pursuing these future directions will be crucial for establishing SWE as a routine, reliable tool in the management of chronic kidney disease, thereby improving early detection, patient monitoring, and overall clinical outcomes.

SUMMARY OF STUDY RESULTS:

- **Demographics:** The study enrolled 96 CKD patients with a mean age of 52.3 ± 12.5 years (range: 25–78 years) and a gender distribution of 58.3% male and 41.7% female.
- **CKD Staging:** The distribution was as follows – Stage 1: 10.4%, Stage 2: 20.8%, Stage 3: 36.5%, Stage 4: 20.8%, Stage 5: 11.5%.

- **Renal Stiffness (kPa):** Mean stiffness values increased with disease severity – Stage 1: 15.2 ± 2.1, Stage 2: 18.5 ± 2.5, Stage 3: 22.8 ± 3.0, Stage 4: 27.1 ± 3.5, Stage 5: 32.4 ± 4.0.
- **Age Distribution by Stage:** Mean ages were Stage 1: 45.6 ± 10.2, Stage 2: 48.3 ± 9.8, Stage 3: 53.2 ± 11.5, Stage 4: 56.8 ± 12.0, and Stage 5: 60.4 ± 13.1 years.
- **Laboratory Findings:** Average serum creatinine was 2.1 ± 0.8 mg/dL, eGFR was 45.6 ± 18.2 mL/min/1.73 m², and blood urea nitrogen was 35.4 ± 10.5 mg/dL.
- **Ultrasound Parameters:** In Stage 1, renal length measured 11.2 cm with a cortical thickness of 8.5 mm; by Stage 5, these values decreased to 9.6 cm and 6.8 mm respectively.
- **Correlations:** Renal stiffness showed a strong negative correlation with eGFR ($r = -0.68$, $p < 0.001$) and a strong positive correlation with serum creatinine ($r = 0.72$, $p < 0.001$).
- **Reproducibility:** Intra-observer ICC was 0.92 and inter-observer ICC was 0.89, indicating high measurement consistency.
- **Comorbid Conditions:** Hypertension was present in 70.8% of patients, diabetes in 46.9%, and cardiovascular disease in 20.8%.
- **Subgroup Findings:** Diabetic patients exhibited higher stiffness (26.3 ± 4.2 kPa) than non-diabetics (20.1 ± 3.8 kPa, $p < 0.001$), and hypertensive patients had higher stiffness (25.4 ± 4.0 kPa) compared to non-hypertensives (21.2 ± 3.5 kPa, $p = 0.002$).
- **Regression Analysis:** Independent predictors of increased stiffness included age ($\beta = 0.15$, $p = 0.004$), serum creatinine ($\beta = 2.3$, $p < 0.001$), decreased

eGFR ($\beta = -0.12$, $p = 0.003$), diabetes ($\beta = 3.1$, $p < 0.001$), and hypertension ($\beta = 1.8$, $p = 0.001$).

- **Diagnostic Accuracy:** ROC analysis identified an optimal cut-off of 28.0 kPa for severe CKD (AUC = 0.88; sensitivity = 85.0%, specificity = 80.0%, PPV = 78.2%, NPV = 86.4%).
- **Bilateral Consistency:** No significant difference was observed between left (24.8 ± 4.2 kPa) and right (25.1 ± 4.3 kPa) kidneys ($p = 0.45$).
- **Age Group Analysis:** Mean stiffness values were 20.5 ± 3.2 kPa for patients under 40, 24.3 ± 3.9 kPa for those aged 40–60, and 28.7 ± 4.5 kPa for patients over 60 ($p < 0.001$).
- **Safety Profile:** The procedure was well tolerated with 91.7% of patients reporting no adverse events, 5.2% experiencing minor discomfort, and 3.1% requiring repeat examinations due to inconclusive results.

CONCLUSION

The findings of this study provide compelling evidence that shear wave elastography (SWE) is a highly valuable, non-invasive imaging tool for assessing renal parenchymal stiffness and, by extension, the degree of renal fibrosis in patients with chronic kidney disease (CKD). The study, which involved 96 participants with a mean age of 52.3 ± 12.5 years and a balanced gender distribution, has demonstrated that renal stiffness increases significantly as CKD progresses, with values rising from 15.2 kPa in Stage 1 to 32.4 kPa in Stage 5. This gradual increase in stiffness is paralleled by deteriorating renal function, as evidenced by strong correlations with key laboratory markers; a negative correlation with estimated glomerular filtration rate (eGFR) ($r = –0.68$, $p < 0.001$) and a positive correlation with serum creatinine levels ($r = 0.72$, $p < 0.001$) were observed. These correlations underscore the clinical relevance of SWE measurements in reflecting both structural and functional changes in the kidneys, thereby supporting its role as an early indicator of disease progression.

The study further highlights the impact of common comorbidities on renal stiffness, with hypertensive and diabetic patients showing significantly higher stiffness values compared to their non-hypertensive and non-diabetic counterparts. Specifically, diabetic patients recorded a mean stiffness of 26.3 kPa compared to 20.1 kPa in non-diabetic patients, while hypertensive patients had a mean stiffness of 25.4 kPa as opposed to 21.2 kPa in those without hypertension. These findings suggest that both diabetes and hypertension exacerbate the fibrotic process in the kidney, contributing to accelerated disease progression. The multivariate regression analysis confirmed that age, serum creatinine, eGFR, diabetes, and hypertension are independent predictors of renal stiffness, emphasizing the multifactorial nature of CKD and the importance of a comprehensive diagnostic approach.

Furthermore, the ROC analysis established an optimal stiffness cut-off of 28.0 kPa for differentiating severe CKD (Stages 4 and 5), with an area under the curve (AUC) of 0.88, sensitivity of 85.0%, and specificity of 80.0%. These diagnostic performance metrics illustrate that SWE is not only capable of detecting subtle changes in renal tissue but also provides an objective threshold for clinical decision-making. The high positive predictive value (78.2%) and negative predictive value (86.4%) further validate its use as a reliable screening tool, potentially reducing the need for invasive procedures such as renal biopsy. In addition, the study found no significant differences in stiffness measurements between the left and right kidneys, indicating that CKD-induced fibrotic changes are likely to affect both kidneys uniformly. The age group analysis also revealed that renal stiffness increases significantly with age, with values rising from 20.5 kPa in patients under 40 to 28.7 kPa in those over 60, which highlights the importance of considering age-related physiological changes when interpreting SWE results.

The implications of these findings are profound for both clinical practice and future research. Clinically, SWE offers a rapid, non-invasive, and reproducible method for assessing renal fibrosis, which is crucial for the early detection and management of CKD. The ability to quantify changes in renal stiffness in relation to biochemical markers of kidney function allows for more precise risk stratification and may guide individualized therapeutic interventions aimed at slowing disease progression. Furthermore, the excellent safety profile of SWE, with 91.7% of patients reporting no adverse events, supports its routine use in clinical settings without exposing patients to significant risks.

Looking ahead, the integration of SWE into standard diagnostic protocols for CKD could revolutionize patient management by enabling more frequent and accurate monitoring of renal fibrosis. Future research should focus on longitudinal studies to evaluate how changes in stiffness over time correlate with clinical outcomes, and whether early interventions based on SWE findings can alter the course of the disease. Additionally, multicenter trials with larger, more diverse populations are needed to further validate these findings and refine the cut-off values for different stages of CKD. There is also potential to explore the synergistic use of SWE with other imaging modalities to enhance diagnostic accuracy further. In conclusion, the robust evidence provided by this study firmly establishes shear wave elastography as a reliable, non-invasive tool for evaluating renal fibrosis in CKD, with significant implications for improving patient outcomes through early detection and tailored therapeutic strategies.

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ANNEXURES

ANNEXURE – I - INFORMED CONSENT FORM

"SHEAR WAVE ELASTOGRAPHY IN EVALUVATION OF RENAL PARENCHYMAL STIFFNESS IN PATIENT WITH CHRONIC KIDNEY DISEASE: A HOSPITAL BASED ONE YEAR CROSS SECTIONAL STUDY"

Name of Student/Principal Investigator: _________

Name of Guide/Co Investigators: _____________

Introduction: Chronic kidney disease (CKD) detection in current medical practice relies mainly on biological markers. Irrespective of the underlying etiology, CKD progresses with parenchymal fibrosis, tubulointerstitial atrophy and sclerosis of the glomerular compartment. These structural abnormalities are detected by conventional gray scale ultrasound(us) imaging are not objectively quantifiable. Therefore, a quantifiable non-invasive imaging like shear wave elastography is needed.

Explanation of procedure: After proper informed, written consent, patients coming for renal ultrasonography and fitting the inclusion criteria will be selected. Patients will fill the pre-designed questionnaire. Findings of CKD will be noted on Ultrasonography. The CKD patients will also be seen on elastography and findings will be noted.

Withdrawal from participation in the study: Participation in this study in voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will not get any benefits by participating in this study. The data gathered will help population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person to identify you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Cost of investigations done during the course of study will be paid by the Participant.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purpose and or presented to scientific groups. However, your identity will never be revealed.

Questions: If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights

CONSENT STATEMENT

I am making a voluntary decision to participate in the study **“SHEAR WAVE ELASTOGRAPHY IN EVALUVATION OF RENAL PARENCHYMAL STIFFNESS IN PATIENT WITH CHRONIC KIDNEY DISEASE: A HOSPITAL BASED ONE YEAR CROSS SECTIONAL STUDY”**. My signature below indicates that I have decided to participate and I have read the information provided above or the information provided above has been read to me in the language that I understand best. I was given the opportunity to ask questions and that they have been answered to my satisfaction.

Name of the participant:

Signature or left thumb impression of the participant:

Name of the witness:

Signature or left thumb impression of the witness:

Name of the investigator:

Signature of the investigator:

ANNEXURE – II PREFORMA

ROLE OF SHEAR WAVE ELASTPGRAPHY IN EVALUVATION OF RENAL PARENCHYMAL STIFNESS IN PATIENT WITH CHRONIC KIDNEY DISEASE: A HOPSITAL BASED ONE YEAR CROSS SECTIONAL STUDY

A) BIODATA

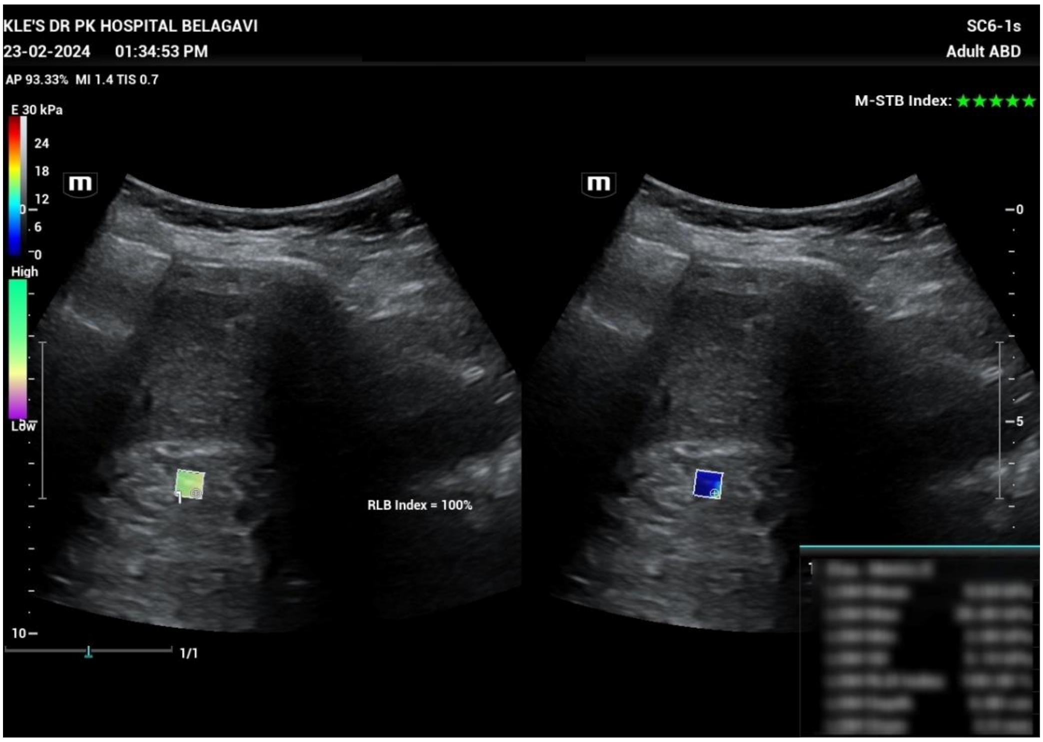
1. NAME
2. AGE
3. SEX
4. PHONE NUMBER
5. ADDRESS
6. OCCUPATION
7. HEIGHT
8. WEIGHT

B) RISK FACTORS -

1. ALCOHOL
2. BMI
3. DIABETES
4. HYPERTENSIVE DISORDER
5. DRUGS
6. CONGENITAL KIDNEY DISEASE
7. FAMILY HISTORY
8. DIETARY HABITS

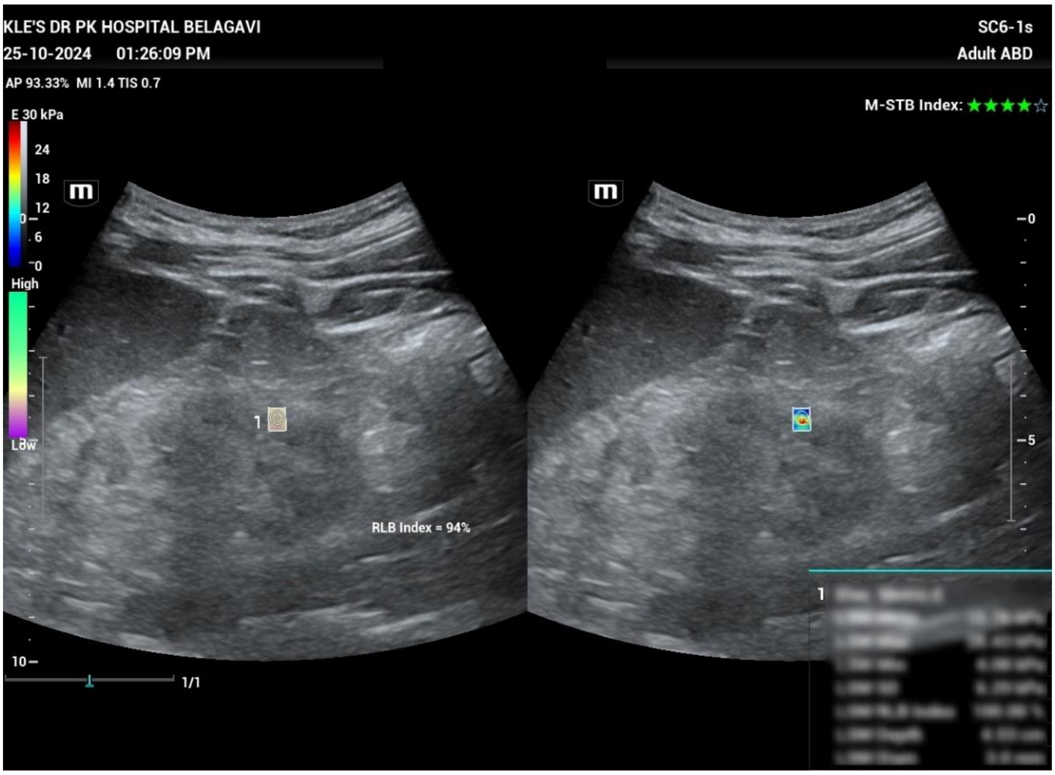
C)USG FINDINGS

ANNEXURE III: PHOTOGRAPHS

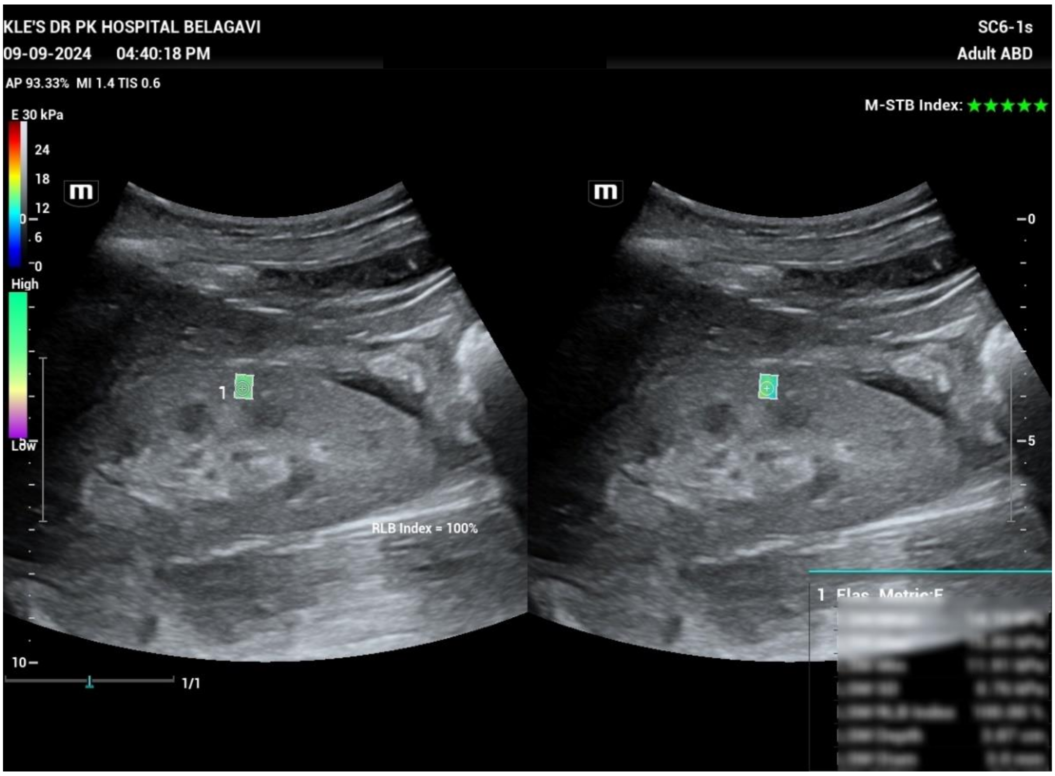


Photograph 1: Patient with CKD stage 4 with average stiffness of 24 kPa

Photograph 2: Patient with CKD stage 5 with average stiffness of 38 kPa



Photograph 3: Patient with CKD stage 4 with average stiffness of 28 kPa



Photograph 4: Patient with CKD stage 2 with average stiffness of 21 kPa

ANNEXURE III – MASTER CHART

SL NO	Patient ID	Date of Examination	Age (years)	Gender	CKD Stage	Average Renal Stiffness (kPa)	Left Kidney Stiffness (kPa)	Right Kidney Stiffness (kPa)	Serum Creatinine (mg/dL)	eGFR (mL/min/1.73m²)	Blood Urea Nitrogen (mg/dL)	Left Renal Length (cm)	Right Renal Length (cm)	Left Cortical Thickness (mm)	Right Cortical Thickness (mm)	Hypertension (Yes/No)	Diabetes Mellitus (Yes/No)	Cardiovascular Disease (Yes/No)	Adverse Events/Complications	Observer Name	Intra-observer Variability (ICC)	Inter-observer Variability (ICC)	Cut-off Value for Severe CKD (kPa)	Sensitivity (%)	Specificity (%)	Positive Predictive Value (PPV) (%)	Negative Predictive Value (NPV) (%)	ROC AUC Value	Left vs Right Kidney Stiffness (p-value)	Age Group	Renal Stiffness (kPa) by Age Group	Renal Stiffness (kPa) in Diabetes	Renal Stiffness (kPa) in Hypertension
1	PT-001	2024-01-01	63	Female	2	21	21	17	1	53	32	10	10	8	8	Yes	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	26	26.3	25.4
2	PT-002	2024-01-02	76	Male	4	24	23	22	4	19	31	12	10	9	9	Yes	Yes	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	20	26.3	25.4
3	PT-003	2024-01-03	53	Male	3	28	19	20	3	44	21	11	11	8	8	Yes	Yes	Yes	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	23	26.3	25.4
4	PT-004	2024-01-04	39	Male	3	21	24	20	3	38	27	10	11	8	7	Yes	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	22	26.3	25.4
5	PT-005	2024-01-05	67	Male	3	25	21	18	1	30	24	11	11	8	8	Yes	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	23	26.3	25.4
6	PT-006	2024-01-06	32	Female	3	18	25	20	3	35	54	11	12	9	5	Yes	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	26	20.1	21.2
7	PT-007	2024-01-07	45	Male	1	14	14	16	0	74	45	11	10	7	9	Yes	Yes	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	22	26.3	25.4
8	PT-008	2024-01-08	63	Female	4	28	23	27	2	20	49	9	10	9	6	Yes	Yes	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	24	20.1	25.4
9	PT-009	2024-01-09	43	Female	3	28	20	23	2	39	43	10	9	8	7	Yes	No	Yes	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	22	20.1	21.2
10	PT-010	2024-01-10	47	Male	2	22	20	19	2	56	24	10	10	7	7	No	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	30	20.1	25.4
11	PT-011	2024-01-11	35	Male	1	13	12	14	1	89	30	10	10	8	7	Yes	Yes	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	26	20.1	21.2
12	PT-012	2024-01-12	35	Female	3	26	20	20	2	37	41	11	12	7	9	Yes	Yes	Yes	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	25	26.3	25.4
13	PT-013	2024-01-13	48	Female	4	31	22	29	4	28	23	11	10	8	7	Yes	Yes	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	27	20.1	25.4
14	PT-014	2024-01-14	77	Female	1	18	14	12	1	84	43	10	11	7	8	Yes	No	Yes	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	22	26.3	25.4
15	PT-015	2024-01-15	60	Female	3	22	18	22	1	37	33	11	10	7	8	Yes	Yes	Yes	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	28	26.3	25.4
16	PT-016	2024-01-16	64	Male	2	22	19	18	2	53	31	11	11	6	8	Yes	Yes	Yes	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	26	20.1	21.2
17	PT-017	2024-01-17	48	Male	3	18	21	20	2	44	43	11	12	7	8	Yes	Yes	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	18	26.3	25.4
18	PT-018	2024-01-18	27	Male	2	22	21	15	2	58	40	11	10	6	7	Yes	Yes	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	19	26.3	21.2
19	PT-019	2024-01-19	46	Female	4	25	27	32	4	22	32	11	10	7	8	Yes	Yes	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	22	20.1	25.4
20	PT-020	2024-01-20	77	Female	3	20	19	20	3	35	48	11	11	8	7	No	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	19	20.1	25.4
21	PT-021	2024-01-21	26	Male	5	27	33	26	5	6	24	10	11	8	6	No	Yes	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	23	20.1	25.4
22	PT-022	2024-01-22	48	Male	2	16	18	22	2	56	42	9	12	8	7	No	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	20	26.3	25.4
23	PT-023	2024-01-23	68	Male	3	19	19	27	1	37	42	11	11	7	9	No	Yes	No	Minor discomfort	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	24	20.1	21.2
24	PT-024	2024-01-24	54	Male	2	21	21	18	2	51	32	11	10	8	8	Yes	No	Yes	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	30	26.3	25.4
25	PT-025	2024-01-25	62	Female	5	28	33	32	4	12	39	11	11	8	7	Yes	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	24	20.1	25.4
26	PT-026	2024-01-26	26	Female	4	28	32	27	2	21	22	10	11	7	7	Yes	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	23	20.1	25.4
27	PT-027	2024-01-27	45	Female	2	19	21	15	1	53	45	10	11	9	7	Yes	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	26	20.1	25.4
28	PT-028	2024-01-28	57	Male	3	22	22	20	1	32	33	11	11	9	9	No	Yes	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	22	20.1	25.4
29	PT-029	2024-01-29	36	Male	4	30	30	27	4	15	30	10	11	8	8	Yes	Yes	Yes	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	28	26.3	25.4
30	PT-030	2024-01-30	46	Female	3	24	24	27	2	33	46	11	11	8	8	No	No	Yes	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	26	20.1	21.2
31	PT-031	2024-01-31	68	Female	3	25	23	21	2	43	28	11	11	8	9	Yes	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	24	20.1	25.4
32	PT-032	2024-02-01	49	Male	2	16	18	20	2	49	21	11	11	6	8	No	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	25	26.3	25.4
33	PT-033	2024-02-02	73	Female	1	18	17	17	0	63	19	11	10	8	7	Yes	No	Yes	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	32	20.1	25.4
34	PT-034	2024-02-03	51	Male	5	38	36	32	4	11	42	10	11	7	8	Yes	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	28	26.3	25.4
35	PT-035	2024-02-04	66	Male	5	27	27	33	4	13	22	11	11	7	8	Yes	Yes	Yes	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	33	20.1	21.2
36	PT-036	2024-02-05	52	Male	3	18	26	18	2	42	54	10	10	8	8	Yes	Yes	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	26	20.1	25.4

37	PT-037	2024-02-06	40	Male	3	27	19	23	2	30	14	10	11	9	8	Yes	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	18	20.1	25.4
38	PT-038	2024-02-07	39	Male	3	24	25	19	1	35	53	10	10	8	8	Yes	No	No	Minor discomfort	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	23	26.3	25.4
39	PT-039	2024-02-08	71	Male	4	26	33	30	2	26	38	11	10	7	8	No	Yes	Yes	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	17	20.1	25.4
40	PT-040	2024-02-09	75	Female	5	36	34	26	5	7	34	10	11	8	8	Yes	No	Yes	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	26	20.1	25.4
41	PT-041	2024-02-10	68	Male	5	34	36	27	4	7	30	11	11	9	8	Yes	Yes	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	27	20.1	21.2
42	PT-042	2024-02-11	76	Male	4	24	22	27	4	19	40	10	10	9	6	Yes	No	Yes	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	21	26.3	21.2
43	PT-043	2024-02-12	27	Female	3	18	27	24	2	42	35	10	9	8	8	No	Yes	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	22	26.3	21.2
44	PT-044	2024-02-13	61	Male	1	12	15	14	0	83	47	10	11	7	8	Yes	Yes	Yes	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	25	20.1	25.4
45	PT-045	2024-02-14	75	Male	2	21	16	21	1	49	37	12	11	8	8	Yes	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	20	26.3	25.4
46	PT-046	2024-02-15	31	Female	5	30	36	38	3	5	37	11	12	8	5	Yes	Yes	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	19	20.1	25.4
47	PT-047	2024-02-16	45	Male	3	25	28	18	3	38	32	11	10	9	9	Yes	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	29	20.1	25.4
48	PT-048	2024-02-17	33	Male	1	17	12	16	1	78	35	9	11	8	8	Yes	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	25	20.1	21.2
49	PT-049	2024-02-18	63	Male	1	14	15	16	1	76	39	11	11	9	9	Yes	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	22	20.1	25.4
50	PT-050	2024-02-19	42	Male	3	22	23	24	1	32	17	11	12	7	7	Yes	Yes	No	Minor discomfort	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	18	20.1	21.2
51	PT-051	2024-02-20	28	Female	1	18	13	17	0	71	21	11	10	9	8	No	Yes	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	28	26.3	21.2
52	PT-052	2024-02-21	49	Female	2	15	18	21	1	57	43	11	11	8	7	Yes	Yes	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	22	26.3	25.4
53	PT-053	2024-02-22	38	Female	3	19	22	21	3	31	37	10	11	9	7	Yes	No	Yes	Inconclusive elastography	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	32	26.3	25.4
54	PT-054	2024-02-23	74	Female	4	30	30	27	4	20	33	11	11	7	9	Yes	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	28	26.3	25.4
55	PT-055	2024-02-24	33	Female	3	25	23	18	1	36	36	12	10	9	8	No	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	21	20.1	25.4
56	PT-056	2024-02-25	50	Male	2	19	19	16	1	52	39	10	11	8	8	Yes	No	Yes	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	21	20.1	21.2
57	PT-057	2024-02-26	77	Female	4	29	28	25	3	17	30	10	11	7	8	No	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	31	20.1	25.4
58	PT-058	2024-02-27	26	Male	2	18	20	18	2	49	27	12	10	7	8	Yes	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	24	20.1	25.4
59	PT-059	2024-02-28	44	Female	3	25	23	23	1	33	37	12	10	8	8	Yes	Yes	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	22	20.1	25.4
60	PT-060	2024-02-29	52	Female	4	26	33	32	4	27	25	10	10	8	8	Yes	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	24	20.1	21.2
61	PT-061	2024-03-01	71	Male	3	26	18	23	2	39	40	10	11	8	10	No	Yes	No	Minor discomfort	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	22	20.1	25.4
62	PT-062	2024-03-02	31	Male	4	26	33	26	3	28	18	10	9	7	8	Yes	No	Yes	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	25	20.1	21.2
63	PT-063	2024-03-03	68	Male	3	22	23	25	2	35	46	10	10	8	8	No	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	24	26.3	25.4
64	PT-064	2024-03-04	32	Male	3	18	21	27	3	38	40	10	11	10	9	No	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	28	26.3	21.2
65	PT-065	2024-03-05	71	Female	1	14	18	17	0	71	38	10	11	9	9	Yes	Yes	Yes	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	23	26.3	21.2
66	PT-066	2024-03-06	59	Female	3	23	21	25	1	40	46	10	10	8	9	Yes	Yes	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	21	20.1	25.4
67	PT-067	2024-03-07	38	Male	2	15	18	16	2	54	53	10	10	8	8	Yes	Yes	No	Minor discomfort	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	23	20.1	25.4
68	PT-068	2024-03-08	41	Male	2	20	15	18	1	56	46	11	12	8	7	Yes	Yes	Yes	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	23	26.3	21.2
69	PT-069	2024-03-09	60	Male	5	26	33	37	4	10	16	12	9	8	9	No	Yes	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	24	26.3	25.4
70	PT-070	2024-03-10	74	Male	3	22	27	28	3	43	22	10	9	7	7	No	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	27	20.1	21.2
71	PT-071	2024-03-11	64	Male	5	34	27	30	4	11	29	11	11	8	8	Yes	No	Yes	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	27	26.3	25.4
72	PT-072	2024-03-12	28	Male	3	22	26	18	3	39	36	10	10	6	7	Yes	Yes	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	29	26.3	21.2
73	PT-073	2024-03-13	26	Female	4	28	25	30	3	26	41	10	10	8	8	No	Yes	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	21	20.1	21.2
74	PT-074	2024-03-14	30	Male	3	28	22	21	1	36	28	11	11	8	8	Yes	Yes	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	26	26.3	21.2
75	PT-075	2024-03-15	66	Male	3	21	20	26	3	37	37	10	11	7	8	No	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	27	26.3	25.4
76	PT-076	2024-03-16	28	Female	3	18	24	26	1	35	27	11	10	9	8	Yes	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	22	20.1	25.4
77	PT-077	2024-03-17	53	Male	2	17	18	22	1	57	29	11	10	7	9	Yes	No	Yes	Minor discomfort	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	23	20.1	25.4
78	PT-078	2024-03-18	42	Female	4	30	23	23	2	29	21	11	11	7	8	Yes	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	17	20.1	25.4
79	PT-079	2024-03-19	50	Female	2	18	21	18	2	48	26	11	11	8	8	Yes	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	28	26.3	25.4
80	PT-080	2024-03-20	68	Male	1	13	12	18	0	83	21	12	10	9	8	Yes	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	23	26.3	25.4
81	PT-081	2024-03-21	58	Male	3	21	19	28	3	41	25	10	12	9	9	Yes	Yes	Yes	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	28	26.3	21.2
82	PT-082	2024-03-22	34	Male	2	20	15	15	1	54	46	10	10	7	7	Yes	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	25	20.1	25.4
83	PT-083	2024-03-23	60	Male	5	37	32	35	3	14	25	10	9	9	8	No	Yes	Yes	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	24	20.1	25.4
84	PT-084	2024-03-24	38	Male	5	34	35	36	3	7	63	10	10	8	8	No	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	18	26.3	25.4
85	PT-085	2024-03-25	55	Female	5	36	38	30	5	5	41	10	10	8	7	Yes	Yes	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	19	20.1	25.4
86	PT-086	2024-03-26	72	Male	3	18	21	23	2	41	37	11	11	9	9	Yes	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	14	26.3	25.4
87	PT-087	2024-03-27	39	Male	1	12	18	18	0	73	26	11	10	8	8	Yes	No	No	Inconclusive	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	22	26.3	21.2

																			elastography														
88	PT-088	2024-03-28	32	Male	5	27	35	26	4	9	43	10	11	7	8	Yes	No	No	Minor discomfort	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	33	26.3	21.2
89	PT-089	2024-03-29	38	Female	3	21	21	26	3	37	29	10	10	9	8	Yes	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	19	26.3	25.4
90	PT-090	2024-03-30	47	Male	5	31	26	27	4	6	37	11	11	9	8	Yes	Yes	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	14	20.1	25.4
91	PT-091	2024-03-31	64	Male	5	38	35	30	4	9	62	10	10	7	7	Yes	Yes	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	<40	28	20.1	21.2
92	PT-092	2024-04-01	45	Male	4	27	27	27	3	16	34	11	11	8	7	Yes	Yes	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	34	26.3	25.4
93	PT-093	2024-04-02	40	Female	2	20	16	20	1	59	47	11	11	8	9	Yes	Yes	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	25	20.1	25.4
94	PT-094	2024-04-03	69	Male	3	20	23	25	1	40	28	9	11	8	7	Yes	No	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	40-60	25	26.3	25.4
95	PT-095	2024-04-04	42	Female	4	33	22	22	4	27	35	12	11	7	7	No	Yes	No	None	Observer 1	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	23	20.1	25.4
96	PT-096	2024-04-05	71	Female	3	19	28	23	1	33	54	12	11	7	6	Yes	No	No	None	Observer 2	1	1	28	85	80	78.2	86.4	0.88	0.45	>60	17	20.1	25.4