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Radiological and Biochemical Evidence in Patients with Chronic Renal Failure: An Institutional Study

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ABSTRACT

Chronic kidney disease (CKD) is a progressive condition characterized by a gradual loss of kidney function over time. This study aimed to evaluate the extent of kidney damage and dysfunction in patients with CKD through detailed radiological imaging and biochemical markers. This cross-sectional study included 35 patients diagnosed with CKD. Radiological imaging involving abdominal ultrasound, CT scans, MRI and renal scintigraphy were used to assess kidney structure and function. Biochemical parameters including serum creatinine, blood urea nitrogen (BUN), estimated glomerular filtration rate (eGFR) and additional metabolic markers were measured to evaluate renal function. Radiological imaging showed normal kidney dimensions with small renal masses and benign cysts. Mild renal artery stenosis was noted via MRI and renal scintigraphy reflected normal perfusion and GFR. Biochemical analysis indicated renal impairment with elevated serum creatinine, increased BUN and reduced eGFR. Patients also presented with anemia, mineral metabolism abnormalities, hypoalbuminemia, hyperkalemia, dyslipidemia and significant proteinuria. The integration of radiological imaging with biochemical markers provides a robust framework for assessing the extent and impact of CKD. The findings align with the structural and functional changes observed in CKD, necessitating an interdisciplinary approach to management and intervention.

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Key Words

Chronic kidney disease, biochemical markers, kidney function, glomerular filtration rate, anemia, CT scan, MRI, renal scintigraphy.

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INTRODUCTION

Chronic renal failure (CRF) also known as chronic kidney disease (CKD) is a complex and multifaceted condition characterized by the gradual loss of renal function over an extended period. It is a global health concern affecting millions of people worldwide, with significant implications for morbidity and mortality. In the context of CRF, radiological and biochemical evidence plays a pivotal role in both the diagnosis and management of this condition. Radiological imaging techniques, such as ultrasound, computed tomography (CT) magnetic resonance imaging (MRI) and renal scintigraphy, provide valuable insights into the structural and functional aspects of the kidneys, helping clinicians assess the severity of renal damage and guide treatment decisions. On the other hand, biochemical markers, including serum creatinine, blood urea nitrogen (BUN) and glomerular filtration rate (GFR), offer crucial information about renal function and disease progression.

Chronic renal failure is a condition that encompasses a wide spectrum of renal dysfunction, ranging from mild impairment to end-stage renal disease (ESRD). It is associated with various etiologies, including diabetes mellitus, hypertension, glomerulonephritis and polycystic kidney disease, among others. Given its insidious nature, CRF often remains asymptomatic in its early stages, making early detection and intervention crucial for preventing further progression and complications[1]. Radiological imaging plays a fundamental role in this regard. Techniques such as ultrasound are frequently employed as initial screening tools, offering a non-invasive and cost-effective means to assess kidney size, shape and structural abnormalities[2]. CT and MRI, with their superior anatomical detail, are valuable for identifying renal masses, cysts and vascular abnormalities, while renal scintigraphy provides insights into renal perfusion and function[3]. These imaging modalities aid in the accurate staging of CRF and enable clinicians to differentiate between reversible and irreversible renal damage.

In parallel with radiological assessments, biochemical markers are indispensable for evaluating renal function and monitoring disease progression. Serum creatinine, a waste product of muscle metabolism excreted by the kidneys is one of the most commonly used bio markers in CRF diagnosis[4]. Elevated serum creatinine levels are indicative of impaired glomerular filtration and reduced renal function. However, it is important to note that creatinine levels may not rise significantly until a substantial portion of renal function has already been lost, limiting its sensitivity as an early diagnostic marker[5]. Blood urea nitrogen (BUN) levels, which reflect the metabolism of nitrogenous waste products, are another valuable biochemical parameter in CRF assessment. This study aims to explore the utility of radiological and biochemical evidence in patients with CRF, shedding light on their diagnostic accuracy, prognostic value and their role in the overall management of this challenging condition. Present study also shows the knowledge regarding these diagnostic modalities, drawing on relevant references and studies to provide a comprehensive understanding of their significance in the clinical assessment of CRF.

MATERIALS AND METHODS

A total of 35 adult patients diagnosed with chronic renal failure were enrolled in this study. Participants were recruited from the Department of General Medicine, Mamata Medical College, Khammam in collaboration with Radiodiagnosis Department. Inclusion criteria consisted of patients aged 18 years or older, with a confirmed diagnosis of chronic renal failure based on clinical and laboratory criteria and who provided informed consent to participate in the study. Exclusion criteria included patients with contraindications to radiological imaging procedures or those unwilling to participate.

Data collection

Radiological imaging: All participants underwent a comprehensive radiological assessment, which included:

- Abdominal ultrasound to assess kidney size, shape and the presence of structural abnormalities
- Computed tomography (CT) scans of the abdomen and pelvis with contrast enhancement to evaluate renal masses, cysts and vascular abnormalities
- Magnetic resonance imaging (MRI) for detailed anatomical assessment, if indicated
- Renal scintigraphy to assess renal perfusion and function

Biochemical evaluation: Blood samples were collected from each participant to assess various biochemical markers of renal function, including:

- Serum creatinine levels, measured using an enzymatic assay
- Blood urea nitrogen (BUN) levels, measured using the urease enzymatic method
- Estimation of glomerular filtration rate (GFR) using the Modification of Diet in Renal Disease (MDRD) equation and the Chronic Kidney Disease Epidemiology Collaboration (CKD-EPI) equation

Statistical analysis: The data obtained were compiled and analyzed using the statistical package for social sciences (SPSS) software version 21. A probability $p>0.05$ was considered statistically significant.

RESULTS

The data in Table 1 indicates that the average kidney size and volume are normal, while CT scans show a small number of renal masses and cysts, likely benign. MRI results suggest mild renal artery stenosis without significant tissue damage and renal scintigraphy reveals normal kidney function across the patient cohort, as evidenced by standard GFR levels and good perfusion. Collectively, these results suggest a generally healthy group with respect to kidney function and structure, barring individual anomalies.

Table 2 shows, Serum Creatinine, Elevated to a mean of 2.0 mg dL^{-1} , which is higher than the normal range, indicating reduced kidney function as is typically seen in CKD patients. Blood Urea Nitrogen (BUN): The increased mean level of 29 mg dL^{-1} suggests a decline in the kidneys' ability to filter urea from the blood, common in CKD. Estimated GFR (MDRD and CKD-EPI) The GFR has decreased to a mean of around 45 Ml min 1.73 m^2, reflecting moderate to severe reduction in kidney function, which is a hallmark of CKD.

Table 3 shows, the mean hemoglobin level is found to be 10.5 g dL^{-1} (SD±1.8). Serum calcium and phosphorus levels average at 8.9 mg dL^{-1} (SD±0.6) and 4.8 mg dL^{-1} (SD±1.1) respectively. The intact Parathyroid Hormone (iPTH) levels are elevated with a mean of 150 pg mL^{-1} (SD±75) which may reflect secondary hyperparathyroidism common in CKD. Albumin levels are slightly reduced at 3.5 g dL^{-1} (SD±0.4) potentially indicating malnutrition or chronic protein loss. Potassium levels are marginally high with a mean of 5.2 mEq L^{-1} (SD±0.8) which could pose a risk of hyperkalemia. Bicarbonate levels average at 22 mEq L^{-1} (SD±3) potentially showing compensated metabolic acidosis. The total cholesterol mean is at the higher end of the spectrum at 200 mg dL^{-1} (SD±40) suggesting dyslipidemia. Iron studies reveal a serum ferritin mean of 300 ng mL^{-1} (SD±150) and transferrin saturation at 20% (SD±10) indicating possible iron deficiency or chronic inflammation. Lastly, the urine protein creatinine ratio is significantly increased with a mean of 1500 mg g^{-1} (SD±500) pointing towards considerable proteinuria that is often seen in CKD patients.

DISCUSSION

The above study encompass radiological and biochemical parameters of 35 patients, potentially indicative of Chronic Kidney Disease (CKD). The radiological assessments, including abdominal ultrasound, computed tomography (CT) magnetic resonance imaging (MRI) and renal scintigraphy, provide a comprehensive evaluation of renal structure and function. The ultrasound findings with a mean kidney size of 12 cm and volumes of 145 cm^3 for the right kidney and 140 cm^3 for the left kidney fall with in

Table 1: Summary of radiological imaging parameters for renal assessment in 35 patients

Ameter	Measurement	M (Mean)	SD (Standard Deviation)
Abdominal ultrasound			
Kidney size (length in cm)	Right kidney	12	1.5
	Left kidney	12	1.4
Kidney volume (volume in cm^3)	Right kidney	145	30
	Left kidney	140	25
CT Scan abdomen and pelvis			
Renal Mass Size (diameter in cm)	Average of observed masses	3	1.7
Number of cysts	Per kidney	4	3
MRI (if indicated)			
Renal artery stenosis (%)	Severity of narrowing	30	20
Renal tissue integrity	Score (1-5 scale)	2.5	1.0
Renal scintigraphy			
GFR (mL^{-1} min 1.73 m^2)	Right kidney	60	20
	Left kidney	58	22
Renal perfusion (units)	Relative uptake	0.7	0.2

Table 2: Biochemical marker data for renal function of chronic kidney disease (CKD) in 35 patients

Parameter	Mean (M)	Standard Deviation (SD)
Serum creatinine (mg dL^{-1})	2.0	0.5
Blood urea nitrogen (BUN) (mg dL^{-1})	29	8
Estimated GFR (MDRD) (mL^{-1} min 1.73 m^2)	45	12
Estimated GFR (CKD-EPI) (mL^{-1} min 1.73 m^2)	44	10

Table 3: Biochemical and hematological parameters in chronic kidney disease patients

Parameter	Mean (M)	Standard Deviation (SD)
Hemoglobin (g dL^{-1})	10.5	1.8
Serum calcium (mg dL^{-1})	8.9	0.6
Serum phosphorus (mg dL^{-1})	4.8	1.1
Intact parathyroid Hormone (iPTH) (pg mL^{-1})	150	75
Albumin (g dL^{-1})	3.5	0.4
Potassium (mEq L^{-1})	5.2	0.8
Bicarbonate (mEq L^{-1})	22	3
Total cholesterol (mg dL^{-1})	200	40
Serumferritin (ng mL^{-1})	300	150
Transferrin saturation (%)	20	10
Urine protein/creatinine ratio (mg g^{-1})	1500	500

the normal range, suggesting that, in terms of size and volume the kidneys are not significantly affected. These measurements are crucial, as kidney size can diminish in chronic kidney disease, a fact established by Emamian group[6] which stated that kidney size decreases progressively with advancing stages of CKD.

CT scan results showing an average mass size of 3 cm with a mean of 4 cysts per kidney could be representative of simple cysts that are often considered benign. However, their presence correlates with a study by Al-Said and O'Neill[7] that established a link between the number of renal cysts and CKD progression. The MRI findings with a 30% mean severity of renal artery stenosis are relatively mild and often do not necessitate immediate intervention, which aligns with the Cooper group study results from[8]. The renal tissue integrity score indicates minimal to mild tissue damage, suggesting that, from a morphological standpoint, the kidneys have preserved their structure, as shown in a study by Remuzzi *et al.*[9] which associated higher structural damage scores with decreased renal function.

In renal scintigraphy, the mean GFR of 60 Ml min 1.73 m^2 for the right kidney and 58 mL min 1.73 m^2 for the left kidney are at the lower limit of the normal range, reflecting a possible early-stage CKD as per the MDRD study equation referenced by Levey *et al.*[10] which described a GFR of <60 mL/min/1.73 m^2 as a marker for CKD. Additionally, relative perfusion rates are within normal ranges, providing evidence against significant ischemic damage or scarring that could affect the kidney function, similar to findings reported in the research by Toto[11].

The biochemical data reveals elevated serum creatinine and BUN levels with means of 2.0 mg dL^{-1} and 29 mg dL^{-1}, respectively, indicating renal function compromise. This observation is consistent with Go *et al.*[12] findings that these levels are predictive markers of kidney disease progression and mortality. The estimated GFR values obtained from MDRD and CKD-EPI equations further substantiate the presence of moderate to severe kidney function impairment, as these values corroborate the CKD classification by KDIGO guidelines, which define CKD Stage 3 as a GFR of 30-59 mL min 1.73 m^2.

The hematological parameters mirror the systemic impact of CKD on the body. The mean hemoglobin of 10.5 g dL suggests anemia, which is a common complication of CKD due to erythropoietin deficiency, as discussed in Babitt *et al.* work[13]. The altered calcium and phosphorus homeostasis, with mean levels of 8.9 and 4.8 mg dL^{-1}, respectively, as well as the elevated iPTH mean level of 150 pg mL^{-1}, signify secondary hyperparathyroidism, aligning with Block group findings[14] on bone mineral disorders in CKD. Albumin levels with a mean of 3.5 g dL^{-1} reflect the nutritional and inflammatory challenges faced in CKD, potentially due to proteinuria, as evidenced by the substantially elevated urine protein/creatinine ratio with a mean of 1500 mg g, which is a hallmark of kidney damage as described by Ruggenenti group[15]. The mean potassium level of 5.2 mEq L raises concerns for hyperkalemia, a life-threatening condition well-documented in the renal literature[16]. Bicarbonate levels indicate a compensated metabolic acidosis state, common in CKD and may contribute to the progression of kidney disease as elucidated by Kraut *et al.*[17].

Lastly, the lipid profile represented by the total cholesterol mean of 200 mg dL^{-1} suggests dyslipidemia, a common cardiovascular risk factor in CKD patients, which has been extensively studied and reported, including in the 4D Study (18). Iron studies revealed mean serum ferritin levels of 300 ng mL and transferrin saturation of 20%, suggesting that the inflammation or iron storage disorders often seen in CKD are present, a phenomenon explored in depth by Kalantar-Zadeh *et al*.[19].

CONCLUSION

The combination of radiological and biochemical findings provides a multifaceted picture of the typical alterations seen in CKD. While kidney size and perfusion remain relatively intact, functional measurements indicate a moderate decline in renal function. The comprehensive assessment underscores the intricate balance between preserving kidney structure and the systemic manifestations of reduced renal function, paralleling the trends seen in current literature. This study shows the importance of early detection and management to slow the progression of CKD, as well as the need for regular monitoring of renal function and associated metabolic complications.

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