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An Association of Serum Prolactin in Diabetic vs Non-Diabetic Chronic Kidney Disease Patients in Tertiary Care Centre in North Eastern UP

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HIGHLIGHTS

- Prolactin increased in diabetic CKD
- Diabetes raised prolactin levels
- Dialysis increased prolactin levels
- Prolactin varied with cardiac abnormalities
- Prolactin reflected CKD severity

Key Words:

Chronic kidney disease
Serum prolactin
Diabetes mellitus
Hemodialysis
Cardiovascular disease
eGFR

ABSTRACT

Introduction: Chronic kidney disease (CKD) is associated with endocrine disturbances, including hyperprolactinemia due to impaired renal clearance and altered neuroendocrine regulation. Diabetes may further influence prolactin levels through metabolic and renal abnormalities. **Aim & Objective:** This study assessed serum prolactin in diabetic and non-diabetic CKD patients and its association with cardiovascular findings. **Material & Methods:** This cross-sectional observational study was conducted at the Department of General Medicine, Moti Lal Nehru Medical College, Prayagraj, from March 2025 to March 2026. One hundred adults with established CKD were consecutively enrolled, comprising 59 diabetic and 41 non-diabetic patients. Patients with conditions or medications known to increase prolactin were excluded. Demographic, clinical and laboratory parameters, including serum prolactin, creatinine, eGFR, urea, glycemic indices, dialysis status and cardiovascular findings, were assessed. Appropriate statistical tests and correlation/association analyses were applied. **Results:** Mean serum prolactin was significantly higher in diabetic than non-diabetic CKD patients (75.84 ± 55.20 vs. 28.08 ± 28.93 ng/mL; $p < 0.001$). Diabetic patients also had higher serum creatinine and urea and lower eGFR (all $p < 0.001$). Hemodialysis was more frequent among diabetic patients (59.32% vs. 36.59%; $p = 0.04$). Across the study population, prolactin was significantly higher among patients receiving hemodialysis than those not receiving dialysis (79.07 ± 52.30 vs. 33.44 ± 40.23 ng/mL; $p < 0.001$). Prolactin differed significantly across cardiovascular categories ($p < 0.01$), with the highest mean level in patients with left ventricular hypertrophy (80.72 ± 52.31 ng/mL). **Conclusion:** Serum prolactin was markedly elevated in diabetic CKD and was associated with greater renal dysfunction, dialysis dependence and cardiovascular abnormalities. These findings suggest that prolactin may reflect the severity of endocrine and systemic disturbances in CKD. Further prospective studies are required to determine its independent cardiovascular significance.



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INTRODUCTION

Chronic kidney disease (CKD) is an increasing global public health concern, affecting an estimated 674 million people worldwide in 2021 and contributing to approximately 1.48 million deaths in 2023, making it one of the leading causes of mortality globally. India carries a substantial proportion of this burden, with an estimated 138 million adults affected by CKD in 2023. The prevalence among Indian adults has increased from approximately 11.12% during 2011–2017 to 16.38% during 2018–2023. CKD frequently remains asymptomatic during its early stages, resulting in delayed diagnosis and presentation with advanced renal dysfunction. The burden is particularly important in resource-limited and rural populations, where limited awareness, inadequate screening and restricted access to nephrology services may contribute to late diagnosis and poorer outcomes. In Uttar Pradesh, increasing rates of diabetes and hypertension, along with possible environmental and socio-economic factors, may further contribute to the CKD burden, highlighting the importance of early detection and preventive strategies [1,2].

Diabetes has become one of the major drivers of CKD worldwide. Individuals with diabetes have a substantially greater prevalence of CKD than those with normal glucose regulation, with diabetic kidney disease representing an important cause of progressive renal dysfunction and end-stage kidney disease. Renal involvement in diabetes may initially be clinically silent, followed by albuminuria and progressive decline in estimated glomerular filtration rate (eGFR). The rate of progression varies between individuals and is influenced by glycemic control, hypertension, albuminuria, dyslipidemia and associated cardiovascular and metabolic abnormalities. Compared with diabetic CKD, non-diabetic CKD demonstrates greater etiological heterogeneity, including glomerular, tubulointerstitial and hypertensive renal diseases, and its progression depends on the underlying renal

pathology and response to treatment. Koye DN, et. al; 2018 and Hauwanga WN, Abdalhamedet. Al; 2024, reported a higher frequency of rapid renal function decline and progression to dialysis among patients with diabetic CKD than among those without diabetes. The increasing prevalence of type 2 diabetes therefore emphasizes the importance of early identification of renal disease and appropriate metabolic and renal-protective interventions [3,4].

CKD is associated with extensive endocrine disturbances because the kidneys participate in the metabolism, degradation and clearance of several hormones. Progressive renal dysfunction can therefore alter multiple endocrine pathways, including those involving pituitary and reproductive hormones. Among these abnormalities, hyperprolactinemia is frequently observed in patients with CKD, and serum prolactin (PRL) concentrations may increase as renal function deteriorates. The elevation of PRL is primarily attributed to reduced renal clearance, together with increased secretion and impaired dopaminergic inhibition in the uremic state. Persistent hyperprolactinemia may have reproductive and metabolic consequences, including hypogonadism, menstrual disturbances, sexual dysfunction and infertility. Kaka N, et. al; 2022, suggested that prolactin may represent an important component of the endocrine alterations accompanying progressive renal dysfunction [5,6].

Prolactin is a polypeptide hormone primarily secreted by lactotroph cells of the anterior pituitary. Although traditionally recognized for its role in mammary gland development and lactation, PRL has multiple physiological actions involving reproductive, metabolic and immune functions. Its secretion is predominantly regulated by hypothalamic dopamine and occurs in a pulsatile and circadian pattern, with physiological increases during sleep and in response to stimuli such as stress and suckling. Prolactin receptors are expressed in several tissues, including adipose tissue, liver, vascular tissues and immune cells,

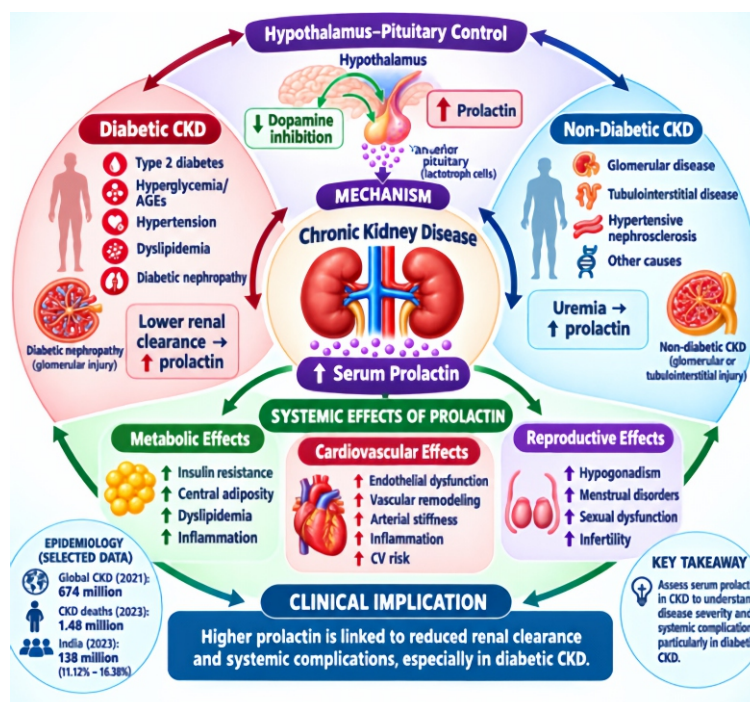


Figure 1: Mechanistic Association of Serum Prolactin in Diabetic vs Non-Diabetic Chronic Kidney Disease

suggesting broader effects on energy metabolism, insulin sensitivity, lipid metabolism and inflammatory responses. Consequently, disturbances in prolactin regulation may have implications extending beyond reproductive physiology and may contribute to systemic metabolic and inflammatory abnormalities [7,8].

Hyperprolactinemia in CKD results from several interacting mechanisms. As glomerular filtration declines, renal metabolism and clearance of circulating prolactin become impaired, resulting in its accumulation within the circulation. In addition, uremia may increase pituitary prolactin secretion and reduce the inhibitory effect of hypothalamic dopamine, thereby further elevating circulating PRL concentrations. Accumulation of uremic toxins, chronic inflammation, metabolic abnormalities and neuroendocrine dysfunction may collectively contribute to this hormonal disturbance. Philipse E, et. al; 2017, suggested that the severity of renal dysfunction is an important determinant of prolactin elevation, irrespective of the underlying cause of CKD. However, comparative evidence regarding differences in prolactin concentrations between diabetic and non-diabetic CKD remains limited, making this an important area for further investigation [9,10].

Elevated prolactin may also have potential cardiometabolic implications in CKD. Prolactin and its biologically active fragments can influence vascular endothelial cells and vascular smooth muscle, potentially affecting endothelial function, vascular remodeling and arterial stiffness. Increased PRL has additionally been associated with insulin resistance, central adiposity, dyslipidemia and low-grade inflammation, all of which are recognized cardiovascular risk factors. In CKD populations, observational studies have reported associations between higher prolactin concentrations, impaired vascular function, increased arterial stiffness and cardiovascular events; one cohort reported a 27% increase in cardiovascular event risk for every 10 ng/mL increase in serum PRL. Nevertheless, evidence remains inconsistent, as some prospective studies have not demonstrated an independent association between modest prolactin elevation and subsequent cardiovascular events after adjustment for confounding factors. Thus, prolactin may function as a marker or potential mediator within the complex interaction of renal dysfunction, inflammation, endocrine abnormalities and cardiovascular risk rather than as an established independent causal factor. Evaluating serum prolactin in diabetic and non-diabetic CKD patients may therefore provide useful information regarding endocrine alterations and their possible relationship with cardiovascular disease [11,12]. Prolactin in Diabetic and Non-Diabetic CKD (**Figure 1**)

The present study aims to assess the association of serum prolactin levels in patients with chronic kidney disease, to compare serum prolactin levels between diabetic and non-diabetic patients with chronic kidney disease, and to evaluate the relationship between serum prolactin levels in chronic kidney disease patients and the presence of cardiovascular disease.

MATERIAL & METHODS

This cross sectional observational study was conducted at the Department of General Medicine, Moti Lal Nehru Medical College, Prayagraj from March 2025 to March 2026. Ethical approval has been obtained from the Ethical Approval Committee of Moti Lal Nehru Medical College, Prayagraj.

Study Population

The study included patients attending the Medicine and Nephrology outpatient departments or admitted to inpatient wards during the study period. Participants were consecutively enrolled after eligibility screening and categorized into CKD patients with diabetes mellitus and CKD patients without diabetes mellitus. Individuals aged >18 years with established CKD of any etiology were included. Patients with hypothyroidism, pregnancy, chest or spinal cord lesions, seizures, prolactinoma, acromegaly, Cushing's disease, or relevant prolactin-elevating medications were excluded.

Data Analysis

Data will be collected, entered, and analyzed using Microsoft Excel and an appropriate statistical software package. Descriptive statistics will be used to summarize the study variables. Appropriate tests of statistical significance will be applied based on the distribution and nature of the data. Continuous and categorical variables will be analyzed using suitable statistical methods. Correlation analysis will be performed to assess the relationship between serum prolactin levels and relevant clinical and laboratory parameters.

RESULTS

The study included 100 participants, comprising 59 diabetic and 41 non-diabetic CKD patients. The mean age was 51.31 ± 14.30 years in the diabetic group and 48.61 ± 13.49 years in the non-diabetic group, with median ages of 52.00 and 54.00 years, respectively; the difference was not statistically significant ($t=0.96$, $p=0.34$). Females constituted 68.89% of the diabetic group and 31.11% of the non-diabetic group, whereas males constituted 50.91% and 49.09%, respectively. However, gender distribution did not differ significantly between groups (Fisher's exact test, $p=0.10$). Fasting blood sugar was significantly higher among diabetic patients (128.95 ± 16.31 mg/dL) than non-diabetic patients (105.51 ± 8.41 mg/dL), with a highly significant difference ($t=9.39$, $p<0.001$).

The mean PPBS was significantly higher in diabetic participants than in non-diabetic participants (193.27 ± 28.41 vs. 164.88 ± 18.62 mg/dL; $t=6.84$, $p<0.001$), while mean HbA1c was also significantly higher in diabetics ($7.32 \pm 1.17\%$ vs. $5.30 \pm 0.57\%$; $t=11.45$, $p<0.001$) (**Table 1**). The mean serum creatinine was significantly higher in diabetic than non-diabetic participants (3.42 ± 1.18 vs. 2.61 ± 0.96 mg/dL; $t=3.65$, $p<0.001$), while serum prolactin was also markedly elevated in diabetics (75.84 ± 55.20 vs. 28.08 ± 28.93 ng/mL; $t=5.63$, $p<0.001$) (**Table 2**). The mean eGFR was significantly lower in diabetic than non-diabetic participants (7.85 ± 1.81 vs. 12.79 ± 2.29 mL/min/1.73 m²;

$t=-12.03$, $p<0.001$), indicating greater impairment of renal function among diabetic CKD patients (**Table 3**). Normal cardiovascular findings were more frequent in non-diabetic participants, whereas LVH and DCMP were more common among diabetic participants; however, the overall difference was not statistically significant (Fisher's exact test, $p=0.10$) (**Figure 2**). The mean serum urea was significantly higher in diabetic than non-diabetic participants (114.67 ± 17.84 vs. 86.62 ± 18.11 mg/dL; $t=7.68$, $p<0.001$), indicating greater accumulation of nitrogenous waste products among diabetic CKD patients (**Table 4**). Hemodialysis was significantly more common among diabetic than non-diabetic participants (59.32% vs. 36.59%), with a statistically significant association between diabetic status and hemodialysis requirement (Fisher's exact test, $p=0.04$)

(**Figure 3**). Being not on dialysis was significantly more common among non-diabetic than diabetic participants (63.41% vs. 40.68%), with a statistically significant association between diabetic status and dialysis status (Fisher's exact test, $p=0.04$) (**Table 5**).

Serum prolactin levels were significantly higher in patients undergoing hemodialysis than in those not on dialysis (79.07 ± 52.30 vs. 33.44 ± 40.23 ng/mL; $t=5.04$, $p<0.001$), with higher median levels also observed in the hemodialysis group (**Table 6**). Serum prolactin levels differed significantly across cardiovascular findings, with the highest mean level in patients with LVH (80.72 ± 52.31 ng/mL), followed by DCMP (54.61 ± 27.99) and normal CVS findings (44.30 ± 50.82 ; ANOVA, $p<0.01$) (**Table 7**).

Table 1: Comparison of Post-prandial Blood Sugar and HbA1c by Enrolment Group

Parameter	Statistic	Case (Diabetic) N = 59	Control (Non-diabetic) N = 41	t-value	p-value
PPBS (mg/dL)	Mean $\pm$ SD	193.27 $\pm$ 28.41	164.88 $\pm$ 18.62	6.84	<0.001
	Median (IQR)	195.00 (178.00 – 208.00)	165.00 (150.00 – 175.00)		
	Min–Max	124–250	140–200		
HbA1c (%)	Mean $\pm$ SD	7.32 $\pm$ 1.17	5.30 $\pm$ 0.57	11.45	<0.001
	Median (IQR)	7.40 (6.50–8.00)	5.30 (4.90–5.60)		
	Min–Max	5.40–11.50	4.20–6.80		

Table 2: Comparison of Serum Creatinine and Serum Prolactin by Enrolment Group

Parameter	Statistic	Case (Diabetic) N = 59	Control (Non-diabetic) N = 41	t-value	p-value
Serum Creatinine (mg/dL)	Mean $\pm$ SD	3.42 $\pm$ 1.18	2.61 $\pm$ 0.96	3.65	<0.001
	Median (IQR)	3.40 (2.60–4.20)	2.50 (1.90–3.10)		
	Min–Max	1.40–6.20	1.20–4.80		
Serum Prolactin (ng/mL)	Mean $\pm$ SD	75.84 $\pm$ 55.20	28.08 $\pm$ 28.93	5.63	<0.001
	Median (IQR)	65.00 (23.10–109.37)	17.14 (14.30–22.60)		
	Min–Max	4.40–200.00	8.18–120.00		

Table 3: eGFR by enrolment group

eGFR	Enrolment group		Unpaired t-test	
	Case (Diabetic) N = 59	Control (Non-diabetic) N = 41	T	p-value
Mean ($\pm$ SD)	7.85 $\pm$ 1.81	12.79 $\pm$ 2.29	-12.03	0.001
Median (IQR)	7.8 (6.6 – 8.9)	12.9 (11.8 – 14.1)		
Min – Max	4.5 – 10.5	10.8 – 15.3		

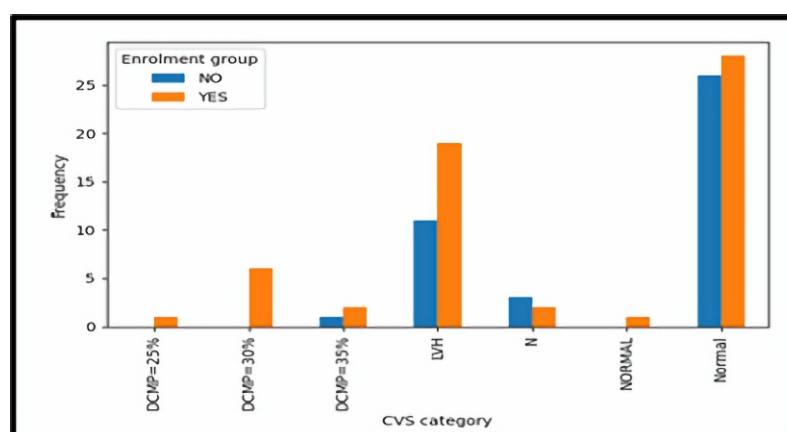
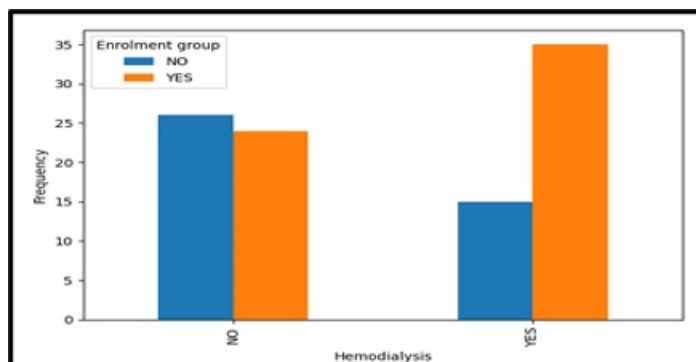


Figure 2: CVS categories by Enrolment Group

Table 4: Serum Urea by enrolment group

S. Urea (mg/dL)	Enrolment group		Unpaired T test	
	Case (Diabetic)	Control (Non- diabetic)	T	p-value
Mean ($\pm$ SD)	114.67 $\pm$ 17.84	86.62 $\pm$ 18.11		
Median (IQR)	115 (103 – 127)	87 (74 – 96)	7.68	< 0.001
Min – Max	90 – 140	65 – 101		

**Figure 3: Hemodialysis status by Enrolment Group****Table 5: Not on Dialysis status by Enrolment Group**

Not on Dialysis	Enrolment group			Fisher's exact test	
	DIABETIC N(%)	NON- DIABETIC N(%)	Total N(%)	X ²	p-value
Yes	24 (40.68)	26 (63.41)	50 (50.00)	Fisher	
No	35 (59.32)	15 (36.59)	50 (50.00)	Fisher	0.04
Total	59 (59.00)	41 (41.00)	100 (100)	–	–

Table 6: Association of Serum Prolactin with Hemodialysis Status

Serum Prolactin	Enrolment group		Unpaired t test	T	p-value
	Case	Control			
	Hemodialysis – Yes N=50	Hemodialysis – No N=50			
Mean ($\pm$ SD)	79.07 $\pm$ 52.30	33.44 $\pm$ 40.23			
Median (IQR)	65.00 (22.60 – 110.00)	17.00 (12.00 – 45.00)			
Min – Max	4.40 – 200.00	4.40 – 178.00		5.04	< 0.001

Table 7: Association of Serum Prolactin with Cardiovascular Findings

Serum Prolactin	Enrolment group			One-way ANOVA	p-value
	Group 1 Normal CVS N=60	Group 2 LVH N=30	Group 3 DCMP N=10		
Mean ($\pm$ SD)	44.30 $\pm$ 50.82	80.72 $\pm$ 52.31	54.61 $\pm$ 27.99		
Median (IQR)	20.30 (13.00 – 65.00)	91.84 (38.20 – 110.00)	50.44 (23.00 – 96.00)		
Min – Max	4.40 – 200.00	8.18 – 200.00	10.00 – 115.00		< 0.01

DISCUSSION

Chronic kidney disease (CKD) is a major and progressively increasing global health problem characterized by persistent impairment of renal function and disruption of metabolic, endocrine, and homeostatic processes [13]. Diabetes mellitus is one of the principal causes of CKD, with chronic hyperglycemia producing microvascular and glomerular injury and accelerating nephron loss. Hypertension and intrinsic renal diseases also contribute substantially. Irrespective of etiology, CKD is associated with considerable morbidity and mortality, with cardiovascular disease (CVD) remaining one of the most important causes of death. In addition to conventional metabolic & hemodynamic

abnormalities, CKD causes significant neuroendocrine disturbances, including alterations in prolactin (PRL) physiology [2,14]. Prolactin is primarily secreted by anterior pituitary lactotrophs and is traditionally associated with lactation, although it also participates in reproductive, metabolic, immune, and vascular processes. Under normal conditions, circulating PRL is maintained at relatively low concentrations through hypothalamic dopaminergic inhibition and adequate renal clearance. In CKD, impaired renal clearance and altered hypothalamic-pituitary regulation associated with uremia contribute to accumulation of circulating PRL and development of hyperprolactinemia [6]. Increased PRL concentrations have been reported with worsening

renal dysfunction and may be associated with reproductive abnormalities, endothelial dysfunction, inflammation, and metabolic disturbances. Experimental and observational evidence has also suggested relationships between PRL and inflammatory mediators such as tumor necrosis factor- α and interleukin-6, although the clinical significance of these associations remains incompletely established [15,16].

The influence of diabetes on prolactin concentrations in patients with CKD remains uncertain. Diabetes may affect the hypothalamic-pituitary-renal axis through chronic hyperglycemia, insulin resistance, inflammation, and associated metabolic abnormalities [12]. Some studies have reported higher PRL concentrations in diabetic nephropathy compared with non-diabetic CKD, whereas others have demonstrated only modest or inconsistent differences. **Lu M, et. al; 2026** and **Soni A, et. al; 2025**, suggested higher median PRL levels among diabetic CKD patients indicate that interactions between metabolic abnormalities and impaired renal hormone clearance may influence prolactin regulation. Clarifying these differences may have relevance for endocrine assessment and understanding cardiovascular risk among patients with CKD [17,18].

In the present study, age was comparable between diabetic and non-diabetic CKD groups, with mean ages of 51.31 ± 14.30 and 48.61 ± 13.49 years, respectively ($p=0.34$). Similarly, no statistically significant difference was observed in sex distribution ($p=0.10$), thereby reducing the likelihood that demographic differences substantially influenced comparisons of prolactin. In contrast, glycemic parameters were significantly higher in diabetic CKD patients. Fasting blood glucose was 128.95 ± 16.31 mg/dL versus 105.51 ± 8.41 mg/dL ($p<0.001$), while HbA1c was $7.32 \pm 1.17\%$ versus $5.30 \pm 0.57\%$ ($p<0.001$). Postprandial blood glucose was also significantly higher in diabetic patients (193.27 ± 28.41 vs. 164.88 ± 18.62 mg/dL; $p<0.001$). **Kirubhakar K, et. al; 2026** and **Jayashankar CA, et. al; 2022**, confirmed substantial metabolic differences between the two groups and are consistent with reports linking prolactin alterations with glycemic status [19,20]. Renal parameters also demonstrated significantly greater impairment among diabetic CKD patients. Serum creatinine was higher in diabetics than non-diabetics (3.42 ± 1.18 vs. 2.61 ± 0.96 mg/dL; $p<0.001$), while eGFR was substantially lower (7.85 ± 1.81 vs. 12.79 ± 2.29 mL/min; $p<0.001$). Serum urea was similarly elevated in diabetic patients (114.67 ± 17.84 vs. 86.62 ± 18.11 mg/dL; $p<0.001$). **Kirubhakar K, et. al; 2026** and **Carrero JJ, et. al; 2012**, indicated more advanced renal dysfunction in the diabetic group. Importantly, serum prolactin was markedly higher among diabetic CKD patients (75.84 ± 55.20 ng/mL) than among non-diabetic patients (28.08 ± 28.93 ng/mL; $p<0.001$). The observed association is consistent with evidence that declining renal function reduces PRL clearance and that diabetic metabolic abnormalities may further influence endocrine regulation [16,19,21]. A significantly greater proportion of diabetic CKD patients were receiving hemodialysis compared with non-diabetic patients (59.32% vs. 36.59%; $p=0.04$), supporting the greater

severity of renal dysfunction in the diabetic group. Across the entire study population, prolactin was significantly higher among patients undergoing hemodialysis than those not receiving dialysis (79.07 ± 52.30 vs. 33.44 ± 40.23 ng/mL; $p<0.001$). Similar findings have been reported in patients with end-stage renal disease, in whom hyperprolactinemia remains common despite dialysis, reflecting impaired renal metabolism and altered neuroendocrine regulation. **Lo JC, et. al; 2017** and **Zhang K, et. al; 2023**, supported renal dysfunction and dialysis dependence as important factors associated with increased serum PRL concentrations [22,23].

Finally, serum prolactin demonstrated a statistically significant variation across cardiovascular categories (ANOVA, $p<0.01$). Patients with left ventricular hypertrophy had the highest mean PRL concentration (80.72 ± 52.31 ng/mL), followed by those with dilated cardiomyopathy (54.61 ± 27.99 ng/mL), whereas patients with normal cardiovascular findings had lower mean levels (44.30 ± 50.82 ng/mL). **Rusu CC, et. al; 2025**, described associations between elevated PRL, vascular dysfunction and cardiovascular risk in CKD, while other studies have reported inconsistent independent associations after adjustment for confounding factors. **Carrero JJ, et. al; 2012**, suggested an association between hyperprolactinemia, severity of renal dysfunction, diabetes, dialysis dependence, and cardiovascular abnormalities, while further prospective studies are required to determine whether prolactin independently contributes to cardiovascular disease or primarily reflects the underlying severity of CKD and its metabolic complications [16,21].

CONCLUSION

In this study, serum prolactin levels were significantly higher among diabetic than non-diabetic CKD patients and were associated with greater renal dysfunction, reflected by higher creatinine and urea, lower eGFR, and increased dialysis requirement. Prolactin concentrations were also significantly elevated among patients receiving hemodialysis and varied across cardiovascular findings, with the highest levels observed in left ventricular hypertrophy. These findings indicate that hyperprolactinemia may accompany advanced renal and metabolic disturbances in CKD. However, its independent role in cardiovascular disease remains uncertain, and larger prospective studies are needed to establish its clinical significance.

LIMITATIONS & FUTURE PERSPECTIVES

The study's limitations include a single-centre setting, a relatively small sample size, and a short study duration, which may limit the broader applicability of the results. Future studies should incorporate multicentre designs with larger populations to enhance validity, assess long-term outcomes, and investigate advanced diagnostic & management approaches. Such efforts will improve overall patient care & help minimize complications.

CLINICAL SIGNIFICANCE

The clinical significance of this study lies in its potential to bridge the gap between research findings and practical health-care, diagnosis, and treatment outcomes. By highlighting real-care applications. It emphasizes the importance of translating scientific observations into meaningful improvements in patient world relevance, the study contributes to evidence based medical practice and supports informed clinical decision making. Ultimately, the findings aim to enhance patient quality of life, optimize therapeutic strategies, and promote better disease management in clinical settings.

ABBREVIATIONS

CKD: Chronic Kidney Disease

eGFR: Estimated Glomerular Filtration Rate

LVH: Left Ventricular Hypertrophy

DM: Diabetes Mellitus

PRL: Prolactin

HD: Hemodialysis

CV: Cardiovascular

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AUTHOR CONTRIBUTIONS

All authors significantly contributed to the study conception and design, data acquisition, or data analysis and interpretation. They participated in drafting the manuscript or critically revising it for important intellectual content, consented to its submission to the current journal, provided final approval for the version to be published, and accepted responsibility for all aspects of the work. Additionally, all authors meet the authorship criteria outlined by the International Committee of Medical Journal Editors (ICMJE) guidelines

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CONFLICT OF INTEREST

Authors declared that there is no conflict of interest.

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ETHICAL APPROVAL & CONSENT TO PARTICIPATE

All necessary consent & approval was obtained by authors.

CONSENT FOR PUBLICATION

All necessary consent for publication was obtained by authors.

DATA AVAILABILITY

All data generated and analyzed are included within this research article. The datasets utilized and/or analyzed in this study can be obtained from the corresponding author upon a reasonable request.

USE OF ARTIFICIAL INTELLIGENCE (AI) & LARGE LANGUAGE MODEL (LLM)

The authors confirm that no AI & LLM tools were used in the writing or editing of the manuscript, and no images were altered or manipulated using AI & LLM.

AUTHOR'S NOTE

This article serves as an important educational tool for the scientific community, offering insights that may inspire future research directions. However, they should not be relied upon independently when making treatment decisions or developing public health policies.

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