

Original Research Article

CLINICAL UTILITY OF SERUM GALECTIN-3 AS AN EARLY BIOMARKER OF CHRONIC KIDNEY DISEASE

S. Rajakumari¹, R. Shobana², S. Syed Ali Fathima³

¹Senior Assistant Professor, Department of Biochemistry, Thanjavur Medical College, Thanjavur, India.

²Assistant Professor, Department of Biochemistry, Government Ariyalur Medical College Ariyalur, India.

³Associate professor Department of Biochemistry, Thanjavur Medical College, Thanjavur, India.

Received : 14/08/2026
Received in revised form : 16/09/2026
Accepted : 29/09/2026

Corresponding Author:

Dr. S. Rajakumari,
Senior Assistant Professor, Department
of biochemistry. Thanjavur Medical
College, Thanjavur, India.
Email: dr.s.rajakumari@gmail.com

DOI: 10.70034/ijmedph.2026.4.98

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (4); 648-654

ABSTRACT

Background: Chronic kidney disease (CKD) is a progressive disorder characterized by irreversible deterioration of renal function and is associated with increased cardiovascular morbidity and mortality. Early diagnosis remains challenging because conventional biomarkers, such as serum creatinine and urinary protein, become abnormal only after significant nephron loss. Galectin-3, a β -galactoside-binding lectin involved in inflammation, fibrosis, and extracellular matrix remodeling, has emerged as a promising biomarker for detecting early renal injury and predicting disease progression. The present study evaluated the clinical utility of serum Galectin-3 as an early biomarker of chronic kidney disease. The aim is to estimate serum Galectin-3 levels in patients with chronic kidney disease and compare them with healthy individuals in order to evaluate its clinical utility as an early biomarker of chronic kidney disease.

Materials and Methods: This hospital-based analytical case-control study was conducted in the Departments of Biochemistry and Nephrology at Thanjavur Medical College Hospital after obtaining Institutional Ethics Committee approval. The study included 100 participants comprising 50 clinically diagnosed CKD patients and 50 age-matched healthy controls. After obtaining written informed consent, fasting venous blood samples were collected for estimation of serum Galectin-3 by sandwich enzyme-linked immunosorbent assay (ELISA). Routine biochemical investigations including blood glucose, blood urea, serum creatinine, and estimated glomerular filtration rate (eGFR) using the MDRD equation were performed. Student's t-test was used for comparison between groups, and Pearson's correlation coefficient was used to determine the association between serum Galectin-3 and renal function parameters. A p-value <0.05 was considered statistically significant.

Results: The study demonstrated significantly elevated serum Galectin-3 levels in CKD patients compared with healthy controls (28.88 ± 9.27 ng/mL vs. 9.02 ± 1.82 ng/mL; $p < 0.001$). CKD patients also had significantly higher serum creatinine (2.37 ± 0.79 mg/dL vs. 0.83 ± 0.18 mg/dL), blood urea (80.56 ± 42.27 mg/dL vs. 36.46 ± 9.48 mg/dL), and markedly reduced creatinine clearance (31.01 ± 10.00 mL/min vs. 111.47 ± 9.90 mL/min) compared with controls (all $p < 0.001$). Serum Galectin-3 levels progressively increased with declining creatinine clearance and showed a strong positive correlation with serum creatinine ($r = 0.649$), blood urea ($r = 0.429$), systolic blood pressure ($r = 0.359$), diastolic blood pressure ($r = 0.194$), and random blood sugar ($r = 0.222$), while demonstrating a significant negative correlation with creatinine clearance ($r = -0.541$).

Conclusion: Serum Galectin-3 is significantly elevated in patients with chronic kidney disease and correlates closely with the severity of renal dysfunction. Its positive association with conventional renal biomarkers and inverse relationship with creatinine clearance suggest that Galectin-3 reflects the inflammatory and

fibrotic processes underlying CKD progression. Serum Galectin-3 may serve as a valuable adjunctive biomarker for the early diagnosis, risk stratification, and monitoring of chronic kidney disease, potentially enabling earlier therapeutic intervention and improved clinical outcomes. Larger prospective multicenter studies are warranted to validate its routine clinical application.

Keywords: Chronic kidney disease, Galectin-3, Biomarker, Renal fibrosis, Serum creatinine, Estimated glomerular filtration rate, ELISA.

INTRODUCTION

CKD is a major worldwide public health problem that results in progressive deterioration in kidney function, substantial morbidity and increased mortality from both cardiovascular⁴ and noncardiovascular causes.^[1-5] It encompasses a spectrum of different pathophysiological processes associated with abnormal kidney function and a progressive decline in Glomerular Filtration Rate (GFR).^[6]

It usually has a gradual onset and in many cases progresses inexorably to a critical state – end stage renal disease (ESRD), in which the patient's survival requires the initiation of renal replacement treatment, either by dialysis or renal transplantation.^[7]

Chronic kidney disease (CKD) refers to an irreversible progressive deterioration in renal function CKD is defined as either kidney damage or GFR < 60 ml/min/1.73 m² for 3 months or more, irrespective of the cause. In CKD, there is a progressive and unrelenting loss of renal function which ultimately leads to end stage renal disease.^[8,9] It is a worldwide, chronic, non-communicable disease, epidemic with adverse outcomes of renal failure, cardiovascular disease and premature death. In developed countries, it affects 10-15% of adult general population.

Prevalence of CKD in India is 0.79%. The domiciliary screening programme for CKD in South India has reported the prevalence of CKD (Stage 5) as 870 per million population (pmp).^[10,11]

In South India, the main causes of CKD are diabetic nephropathy (29.6%), chronic interstitial nephritis (20.4%), chronic glomerulonephritis (17.4%) and hypertensive nephropathy (11%).

CKD with its multitude of complications and devastating outcomes leads to a significantly higher risk for Cardiovascular _Disease (CVD), cerebrovascular disease, peripheral vascular disease and heart failure. Kidney failure is both a cause and consequence of Cardiovascular disease.^[12]

Kidney disease is also associated with chronic inflammation, which promotes endothelial dysfunction, vascular remodelling and progression of atherosclerosis. In CKD, progressive deterioration of renal function may also lead to accumulation of uremic toxins and dyslipidemia, which in turn stimulate inflammation and result in atherosclerosis.^[13]

Chronic kidney disease is typically detected at an advanced stage because of the lack of early clinical signs or symptoms and the poor sensitivity of

currently available biomarkers (serum creatinine and urinary protein). However, when detected early, decline of kidney function may be slowed or even reversed and these secondary complications averted¹⁴. Hence, there is a pressing clinical need for novel biomarkers that identify at-risk individuals at the earliest possible stage.

Early detection of people at risk for CKD progression provides a chance to put prompt therapies into place, which may decrease the illness's progression, lessen cardiovascular problems, and postpone the beginning of end-stage renal disease (ESRD). As a result, a lot of research has gone into finding new biomarkers that can diagnose kidney impairment before irreparable structural damage takes place. An ideal biomarker would be able to forecast the course of the illness, correlate with the severity of the condition, and properly represent continuing renal inflammation and fibrosis.^[14]

Galectin-3 has garnered significant attention among newly studied biomarkers due to its significant function in tissue healing, fibrosis, inflammation, and extracellular matrix remodelling. Galectin-3 is a multifunctional lectin that binds β -galactoside and is a member of the galectin family. Its biological activities are attributed to its carbohydrate recognition domain. Numerous organs, including as the heart, kidneys, vascular endothelium, macrophages, fibroblasts, and inflammatory cells, express it extensively. Galectin-3 controls cellular proliferation, apoptosis, adhesion, migration, immunological responses, and tissue remodelling depending on whether it is found within or outside of cells.^[15-18]

Galectin-3 is typically expressed in the kidney throughout embryonic nephrogenesis, especially in the collecting ducts and ureteric bud. In adult renal tubules, its expression is very modest. Galectin-3 expression significantly rises in pathological circumstances such as ischaemic damage, nephrotoxicity, or chronic inflammation. Galectin-3 is primarily produced by activated macrophages and then increases collagen production and fibroblast activation via TGF- β -dependent pathways. Chronic inflammation, extracellular matrix buildup, and progressive tubulointerstitial fibrosis—the clinical characteristic that causes permanent loss of renal function in chronic kidney disease (CKD)—are all influenced by persistent overexpression of Galectin-3.^[19]

Regardless of the underlying renal illness, tubulointerstitial fibrosis is now acknowledged as one of the best indicators of the advancement of chronic kidney disease. By encouraging fibroblast

proliferation, inflammatory cytokine release, epithelial-mesenchymal transition, and extracellular matrix deposition, galectin-3 appears to be involved in the entire spectrum from acute inflammation to chronic fibrosis. Therefore, even before a notable decline in GFR is clinically noticeable, high circulating Galectin-3 levels may indicate ongoing renal fibrogenesis. Investigating Galectin-3 as an early biomarker of chronic kidney disease is strongly justified by its biological function.

Galectin-3 has been linked to vascular inflammation and cardiovascular remodelling in addition to renal fibrosis. Since cardiovascular disease is the leading cause of death for people with chronic kidney disease (CKD), measuring blood Galectin-3 may simultaneously reveal information about cardiovascular risk and renal damage. As a result, Galectin-3 has become a potential biomarker that can integrate the cardiovascular, fibrotic, and inflammatory pathways implicated in the course of chronic kidney disease.

Evaluation of blood Galectin-3 may enhance early diagnosis, risk stratification, and disease progression monitoring given the limitations of traditional renal biomarkers and the crucial role fibrosis plays in the development of chronic kidney disease. In order to assess blood Galectin-3 levels in patients with chronic kidney disease, compare them with healthy persons, and ascertain their correlation with recognised markers of renal dysfunction, the current study was conducted. The study is to investigate the clinical value of serum Galectin-3 as an early biomarker of chronic kidney disease by looking at its correlation with serum creatinine, estimated glomerular filtration rate (eGFR), and other clinical factors.

Aim

To estimate serum Galectin-3 levels in patients with chronic kidney disease and compare them with healthy individuals in order to evaluate its clinical utility as an early biomarker of chronic kidney disease.

Objectives

1. To estimate serum Galectin-3 levels in patients with chronic kidney disease.
2. To compare serum Galectin-3 levels between CKD patients and healthy controls.
3. To correlate serum Galectin-3 levels with serum creatinine and estimated glomerular filtration rate (eGFR).
4. To evaluate the association of serum Galectin-3 with body mass index, blood pressure, random blood sugar, blood urea, and serum creatinine.

MATERIALS AND METHODS

Study Design: Hospital-based analytical case-control study.

Study Setting: The study was conducted in the Department of Biochemistry in collaboration with the Department of Nephrology at Thanjavur Medical

College Hospital, Thanjavur, after obtaining approval from the Institutional Ethics Committee.

Study Duration: The study was conducted during the postgraduate dissertation period after institutional ethical approval. (The thesis excerpt provided does not specify the exact study dates.)

Study Population: The study included 50 clinically diagnosed CKD patients (25 males and 25 females) attending the outpatient and inpatient services of the Department of Nephrology. Fifty apparently healthy age-matched individuals served as controls.

Sample Size

- Cases: 50 CKD patients
- Controls: 50 healthy individuals
- Total sample size: 100 participants.

Inclusion Criteria

- Patients diagnosed with chronic kidney disease.
- Age greater than 18 years.
- Patients with diabetes mellitus.

Exclusion Criteria

- Primary tubular diseases.
- Recent or concurrent administration of nephrotoxic drugs.
- Acute kidney injury.

Sample Collection: Written informed consent was obtained from all participants. Following an overnight fast of approximately 12 hours, 5 mL of venous blood was collected under aseptic precautions. Blood samples were allowed to clot and centrifuged at 2000 rpm for 15 minutes. Serum was separated, and an aliquot was stored at -20°C for estimation of serum Galectin-3. The remaining serum was utilized for estimation of blood glucose, blood urea, and serum creatinine.

Estimation of Serum Galectin-3

Serum Galectin-3 concentration was estimated using a commercially available sandwich Enzyme-Linked Immunosorbent Assay (ELISA) kit (Bioassay Technology Laboratory). The assay utilized wells pre-coated with anti-human Galectin-3 antibody. Serum samples were incubated with biotinylated anti-Galectin-3 antibody followed by streptavidin-horseradish peroxidase conjugate. After substrate addition, absorbance was measured at 450 nm using a microplate reader. Galectin-3 concentrations were calculated from the standard calibration curve according to the manufacturer's instructions.

Routine Biochemical Investigations

The following biochemical parameters were estimated:

- Blood glucose – Glucose oxidase-peroxidase (GOD-POD) method.
- Blood urea – Urease-GLDH method.
- Serum creatinine – Modified Jaffe's method.

eGFR Calculation

Estimated glomerular filtration rate (eGFR) was calculated using the Modification of Diet in Renal Disease (MDRD) equation:

$$\text{eGFR} = 186 \times (\text{Serum Creatinine})^{-1.154} \times (\text{Age})^{-0.203}$$

A correction factor of 0.742 was applied for females, and 1.21 for Black individuals, as described in the MDRD formula.

Statistical Analysis: Clinical and biochemical data were compiled and statistically analyzed to compare CKD patients with healthy controls. Correlation analyses were performed to evaluate the relationship between serum Galectin-3 concentrations and renal function parameters, including serum creatinine, eGFR, blood urea, blood glucose, body mass index, and blood pressure. (The thesis excerpt provided does not specify the statistical software or individual statistical tests.)

Ethical Approval: The study protocol received approval from the Institutional Ethics Committee of Thanjavur Medical College Hospital, Thanjavur, before commencement of the study. Written

informed consent was obtained from all study participants prior to sample collection.

RESULTS

A total of 100 study participants were included in the present study, comprising 50 patients with chronic kidney disease (CKD) and 50 healthy controls. Serum Galectin-3, serum creatinine, blood urea, random blood sugar (RBS), and estimated creatinine clearance (eGFR by MDRD formula) were evaluated in all participants. Statistical analysis was performed using Student's t-test for comparison between groups, while Pearson's correlation coefficient was used to assess the relationship between serum Galectin-3 and other clinical variables. A p-value of <0.05 was considered statistically significant.

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population

Parameter	Controls (n=50) Mean $\pm$ SD	CKD Cases (n=50) Mean $\pm$ SD	p-value
Age (years)	54.66 $\pm$ 5.72	52.48 $\pm$ 5.58	>0.05
Height (m)	1.62 $\pm$ 0.13	1.63 $\pm$ 0.15	>0.05
Weight (kg)	64.02 $\pm$ 8.66	71.60 $\pm$ 7.50	<0.05
BMI (kg/m ²)	24.50 $\pm$ 3.24	25.77 $\pm$ 3.39	>0.05
Systolic BP (mmHg)	118.44 $\pm$ 7.95	146.36 $\pm$ 14.38	<0.001*
Diastolic BP (mmHg)	77.04 $\pm$ 4.24	88.36 $\pm$ 9.41	<0.001*

[Table 1] presents the baseline demographic and clinical characteristics of the study population, comprising 50 healthy controls and 50 patients with chronic kidney disease (CKD). The mean age was comparable between the control group (54.66 $\pm$ 5.72 years) and CKD patients (52.48 $\pm$ 5.58 years), with no statistically significant difference ($p > 0.05$). Similarly, height and body mass index (BMI) did not differ significantly between the two groups ($p > 0.05$). However, CKD patients had a significantly higher mean body weight than controls (71.60 $\pm$ 7.50 kg vs. 64.02 $\pm$ 8.66 kg; $p < 0.05$). Both systolic and diastolic blood pressures were markedly elevated in CKD patients (146.36 $\pm$ 14.38 mmHg and 88.36 $\pm$ 9.41 mmHg, respectively) compared with healthy controls (118.44 $\pm$ 7.95 mmHg and 77.04 $\pm$ 4.24 mmHg), and these differences were highly statistically significant ($p < 0.001$). These findings

indicate that hypertension was significantly more prevalent among patients with CKD, while the demographic characteristics of the two groups remained comparable.

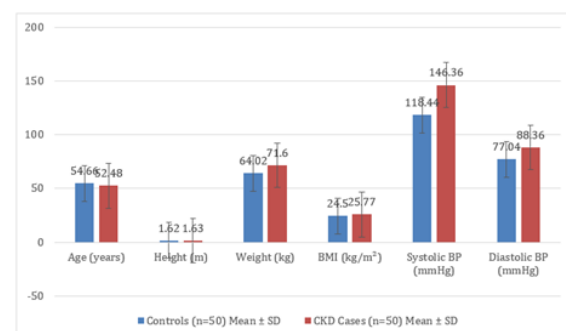


Figure 1: Baseline Demographic and Clinical Characteristics of the Study Population.

Table 2: Comparison of Serum Galectin-3 Levels Between CKD Patients and Healthy Controls

Study Group	Mean (ng/mL)	SD	p-value
Controls (n=50)	9.02	1.82	
CKD Cases (n=50)	28.88	9.27	<0.001*

[Table 2] compares serum Galectin-3 levels between patients with chronic kidney disease and healthy controls. The mean serum Galectin-3 concentration was significantly higher in CKD patients (28.88 $\pm$ 9.27 ng/mL) than in healthy controls (9.02 $\pm$ 1.82 ng/mL). The difference between the two groups was highly statistically significant ($p < 0.001$). These findings suggest that serum Galectin-3 levels increase substantially in patients with chronic kidney disease and support its potential role as a novel biomarker for the early detection and assessment of renal dysfunction.

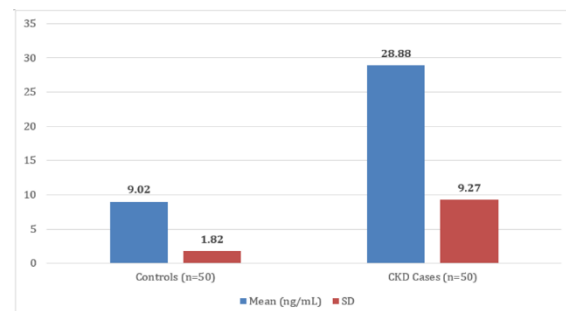


Figure 2: Comparison of Serum Galectin-3 Levels Between CKD Patients and Healthy Controls

[Table 3] compares the renal function parameters between healthy controls and patients with chronic kidney disease. The mean serum creatinine level was significantly higher in CKD patients (2.37 ± 0.79 mg/dL) than in controls (0.83 ± 0.18 mg/dL) ($p < 0.001$). Similarly, blood urea concentration was markedly elevated in CKD patients (80.56 ± 42.27 mg/dL) compared with controls (36.46 ± 9.48 mg/dL), with a highly significant difference ($p < 0.001$). In contrast, creatinine clearance was significantly reduced in CKD patients (31.01 ± 10.00 mL/min) compared to healthy controls (111.47 ± 9.90 mL/min) ($p < 0.001$). These findings confirm significant impairment of renal function among CKD patients and demonstrate the marked deterioration in

glomerular filtration associated with chronic kidney disease.

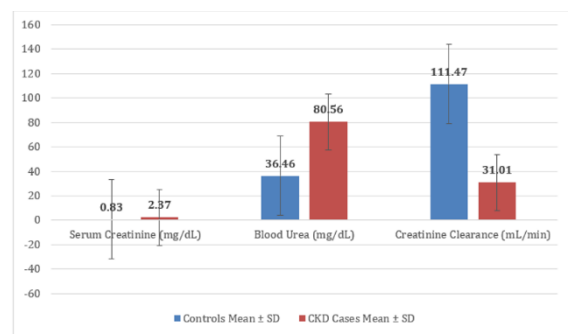


Figure 3: Mean

Table 3: Comparison of Renal Function Parameters Between CKD Patients and Controls

Parameter	Controls Mean $\pm$ SD	CKD Cases Mean $\pm$ SD	p-value
Serum Creatinine (mg/dL)	0.83 $\pm$ 0.18	2.37 $\pm$ 0.79	<0.001*
Blood Urea (mg/dL)	36.46 $\pm$ 9.48	80.56 $\pm$ 42.27	<0.001*
Creatinine Clearance (mL/min)	111.47 $\pm$ 9.90	31.01 $\pm$ 10.00	<0.001*

Table 4: Relationship Between Serum Galectin-3 and Severity of Renal Dysfunction

Creatinine Clearance (mL/min)	Number of Patients	Mean Serum Galectin-3 (ng/mL)
60–90	0	—
30–59	21	45.8 $\pm$ 20.1
15–29	28	72.1 $\pm$ 8.1
<15	1	81.0

[Table 4] illustrates the relationship between serum Galectin-3 levels and the severity of renal dysfunction based on creatinine clearance categories among CKD patients. No patients had creatinine clearance between 60 and 90 mL/min. Among patients with creatinine clearance of 30–59 mL/min ($n = 21$), the mean serum Galectin-3 level was 45.8 ± 20.1 ng/mL. Patients with creatinine clearance of 15–29 mL/min ($n = 28$) demonstrated a higher mean

serum Galectin-3 concentration of 72.1 ± 8.1 ng/mL, while the single patient with creatinine clearance below 15 mL/min had the highest serum Galectin-3 level of 81.0 ng/mL. These findings indicate a progressive increase in serum Galectin-3 concentration with declining renal function, suggesting that elevated Galectin-3 levels are closely associated with increasing severity of chronic kidney disease.

Table 5: Correlation of Serum Galectin-3 with Clinical and Biochemical Parameters in CKD Patients

Parameter	Pearson Correlation (r)	Correlation
Serum Creatinine	0.649	Positive
Creatinine Clearance	−0.541	Negative
Blood Urea	0.429	Positive
Random Blood Sugar	0.222	Positive
Systolic Blood Pressure	0.359	Positive
Diastolic Blood Pressure	0.194	Positive

[Table 5] presents the correlation between serum Galectin-3 levels and various clinical and biochemical parameters in patients with chronic kidney disease. Serum Galectin-3 demonstrated a strong positive correlation with serum creatinine ($r = 0.649$), indicating that Galectin-3 levels increased with worsening renal impairment. A moderate positive correlation was also observed with blood urea ($r = 0.429$), systolic blood pressure ($r = 0.359$), random blood sugar ($r = 0.222$), and diastolic blood pressure ($r = 0.194$). Conversely, serum Galectin-3 showed a moderate negative correlation with creatinine clearance ($r = -0.541$), indicating that Galectin-3 concentrations increased as renal function declined. These findings suggest that serum Galectin-3 is closely associated with both biochemical markers

of renal dysfunction and clinical indicators of disease severity, supporting its potential utility as an early biomarker of chronic kidney disease.

DISCUSSION

Chronic kidney disease (CKD) is a progressive disorder characterized by irreversible loss of renal function and persistent inflammation that ultimately leads to tubulointerstitial fibrosis and end-stage renal disease. Because traditional indicators like serum creatinine only rise after significant nephron loss has taken place, early identification continues to be a significant clinical issue. Finding biomarkers that indicate kidney damage and fibrosis at an earlier

stage is thus of increasing importance. By comparing blood Galectin-3 levels between CKD patients and healthy controls and evaluating its correlation with recognised indicators of renal failure, the current study assessed the clinical value of Galectin-3 as an early biomarker of CKD.

According to [Table 2], serum Galectin-3 concentrations in CKD patients were substantially higher than those in healthy controls (28.88 ± 9.27 ng/mL vs. 9.02 ± 1.82 ng/mL; $p < 0.001$). These results imply that Galectin-3 may be useful as a marker of renal damage and show that it is significantly higher in individuals with chronic kidney disease. De Boer et al. published similar findings, showing that Galectin-3 is strongly linked to tissue remodelling and fibrosis in chronic illnesses, such as renal disease.^[14] According to Liu et al., Galectin-3 is also strongly expressed in inflammatory cells and activated macrophages, which supports its function as a mediator of fibrosis and chronic inflammation.^[15] Henderson et al. also showed that Galectin-3 generated from macrophages stimulates extracellular matrix deposition and renal fibrogenesis, which leads to progressive renal failure.^[18]

While systolic and diastolic blood pressure were considerably higher in CKD patients, baseline demographic data revealed that both groups were similar in terms of age and body mass index [Table 1]. Chronic kidney disease is both a cause and an effect of hypertension, which also plays a major role in the increasing loss of nephrons. These results align with the findings of Schiffrin et al., who identified hypertension as a significant predictor of cardiovascular morbidity and the advancement of renal illness in chronic kidney disease.^[12] Increased blood pressure speeds up glomerular damage, which leads to a decrease in glomerular filtration rate and eventual renal fibrosis.

Additionally, there were notable differences in conventional renal biomarkers between patients and controls. In patients with chronic kidney disease (CKD), serum creatinine and blood urea were considerably raised, whereas creatinine clearance was dramatically decreased [Table 3]. These results are in line with earlier research that described serum creatinine as the conventional biochemical marker of chronic kidney disease (CKD) and validate the existence of decreased renal function in the study group.^[1,2] However, age, gender, muscle mass, and nutritional condition all affect blood creatinine, which lowers its sensitivity for early disease diagnosis. Therefore, rather of focusing only on decreased filtration capacity, the current findings provide justification for assessing other biomarkers such Galectin-3 that represent underlying renal inflammation and fibrosis.

The increasing rise in serum Galectin-3 levels with declining renal function was a significant finding of the current investigation [Table 4]. Galectin-3 concentrations were significantly greater in patients with reduced creatinine clearance, suggesting a

strong correlation between Galectin-3 and the severity of CKD. Henderson et al.'s experimental study, which showed that Galectin-3 produced by activated macrophages increases fibroblast proliferation and promotes tubulointerstitial fibrosis through transforming growth factor- β (TGF- β)-mediated pathways, is consistent with this result.^[18] According to Okamura et al., Galectin-3 is a crucial mediator of the advancement of chronic kidney disease (CKD) as it takes part in extracellular matrix remodelling and progressive renal fibrosis.^[19]

As shown in Table 5, correlation analysis also revealed a significant negative correlation with creatinine clearance ($r = -0.541$) and a strong positive correlation between serum Galectin-3 and serum creatinine ($r = 0.649$), blood urea ($r = 0.429$), random blood sugar ($r = 0.222$), systolic blood pressure ($r = 0.359$), and diastolic blood pressure ($r = 0.194$). These results suggest that deteriorating renal impairment is closely correlated with rising Galectin-3 levels. Similar connections have been reported by de Boer et al., who proposed that Galectin-3 functions as a biomarker of fibrosis and inflammation in renal and cardiovascular illness.^[14] Henderson et al. also showed that Galectin-3 expression directly contributes to fibrotic remodelling and rises with increasing renal damage.^[18]

The increased incidence of diabetic nephropathy in the CKD population is probably the cause of the raised random blood sugar seen in CKD patients. Chronic low-grade inflammation and increased renal fibrosis are linked to diabetes mellitus, which is still one of the primary causes of chronic kidney disease. Galectin-3 expression increases as a result of persistent hyperglycemia's stimulation of inflammatory cytokines and macrophage activation.^[13] This might account for the current study's finding of a positive connection between serum Galectin-3 and random blood sugar.

Galectin-3's biological function adds to its potential therapeutic value. Galectin-3 directly contributes to inflammatory reactions, fibroblast activation, collagen deposition, and extracellular matrix remodelling, in contrast to serum creatinine, which mainly indicates filtration performance. As a result, increased serum Galectin-3 may indicate renal damage earlier than detectable drops in glomerular filtration rate. Intense Galectin-3 overexpression has been shown in experiments after renal ischaemia and nephrotoxic damage, and continuous expression facilitates the transition from acute inflammation to chronic fibrosis.^[18,19]

Overall, the results of this investigation indicate that the fibrotic and inflammatory processes that underlie the development of CKD are reflected in serum Galectin-3. It may supplement traditional renal biomarkers for early diagnosis and disease monitoring, as evidenced by its notable rise in CKD patients [Table 2], increasing increase with declining renal function [Table 4], and high associations with existing biochemical indicators [Table 5]. These findings corroborate the mounting evidence that

blood Galectin-3 is a viable biomarker for identifying people who are more likely to develop chronic kidney disease (CKD) and may help improve clinical management by enabling earlier therapeutic intervention.

CONCLUSION

The present study demonstrated that serum Galectin-3 concentrations were significantly elevated in patients with chronic kidney disease compared with healthy controls and showed a strong association with declining renal function. Serum Galectin-3 levels showed a substantial negative link with creatinine clearance and a gradual rise with deteriorating renal impairment. They also showed significant positive relationships with blood urea, random blood sugar, blood pressure, and serum creatinine. These results suggest that serum Galectin-3 reflects not only decreased glomerular filtration but also the inflammatory and fibrotic mechanisms that cause CKD development. Because Galectin-3 is involved in tubulointerstitial fibrosis, extracellular matrix remodelling, and macrophage activation, it may be able to detect continuing renal damage earlier than traditional renal biomarkers, which only become aberrant after significant nephron loss.

As a result, serum Galectin-3 may be a useful supplementary biomarker for chronic kidney disease monitoring, risk assessment, and early detection. The current study's results validate serum Galectin-3's therapeutic relevance as a new biomarker for chronic kidney disease. Combining Galectin-3 estimate with standard biochemical indicators in regular clinical evaluation may enhance disease monitoring, enable earlier therapeutic intervention, and perhaps slow the development of end-stage renal disease. However, before widespread clinical use, further extensive prospective trials are needed.

Limitations of the study: The present study has certain limitations that should be considered while interpreting the findings. First, there were only 50 patients with chronic renal disease and 50 healthy controls in the sample, which may have limited the data's applicability to the larger CKD community. Second, the MDRD equation, which is based on serum creatinine and may not correctly represent early declines in glomerular filtration rate, was used to compute creatinine clearance, which was used to evaluate renal function. Third, serum Galectin-3 was not compared with other recognised indicators of renal failure, including urine biomarkers of tubular damage and serum cystatin C. Additionally, evaluation of longitudinal changes in Galectin-3 levels or their prognostic value for the development of CKD was not possible due to the cross-sectional research design. Lastly, the study did not assess the impact of various CKD stages or aetiologies independently. To confirm these results, further extensive multicenter prospective trials are needed.

REFERENCES

- Coresh J, Selvin E, Stevens LA, Manzi J, Kusek JW, Eggers P, Van Lente F, Levey AS: Prevalence of chronic kidney disease in the United States. *JAMA* 298: 2038–2047, 2007 [PubMed: 17986697]
- McClellan WM, Flanders WD: Risk factors for progressive chronic kidney disease. *J Am Soc Nephrol* 14[Suppl 2]: S65–S70, 2003 [PubMed: 12819305]
- Muntner P, Vupputuri S, Coresh J, Uribarri J, Fox CS: Metabolic abnormalities are present in adults with elevated serum cystatin C. *Kidney Int* 76: 81–88, 2009 [PMCID: PMC3049931] [PubMed: 19295502]
- Fried LF, Shlipak MG, Crump C, Bleyer AJ, Gottdiener JS, Kronmal RA, Kuller LH, Newman AB: Renal insufficiency as a predictor of cardiovascular outcomes and mortality in elderly individuals. *J Am Coll Cardiol* 41: 1364–1372, 2003 [PubMed: 12706933]
- Weiner DE, Tighiouart H, Amin MG, Stark PC, MacLeod B, Griffith JL, Salem DN, Levey AS, Sarnak MJ: Chronic kidney disease as a risk factor for cardiovascular disease and all-cause mortality: A pooled analysis of community-based studies. *J Am Soc Nephrol* 15: 1307–1315, 2004 [PubMed: 15100371]
- Joanne M. Bargman, Karl Skorecki : *Chronic kidney Disease : Harrison's Principles of Internal medicine*, 17th Edition; edited by Anthony s. Fauci, Engine Braunwald et al; Mc Graw Hill Medical Publications 2008, Vol.II- Pg. 1761.
- William J.Marshall : *The Kidneys, renal function and renal failure: Clinical Biochemistry*, I Edition ;edited by William J. Marshall,Stephen K.Bangert ; Churchill Livingston Publications 1995, Pg 135.
- Martin E Lascano, Martin J Schreiber and Saul Nurko. *Chronic Kidney Disease*. In: William D. Carey, editor. *Cleveland Clinic Current Clinical Medicine*. 2nd Edition. Philadelphia: Elsevier Saunders; 2010. p. 853
- Andrew S Levey, Kai-Uwe Eckardt, Yusuke Tsukamoto et al. *Definition and Classification of Chronic Kidney Disease: Improving Global Outcomes (KDIGO)*. *Kidney International*. 2005; Vol.67: 2089-2100
- S.K. Agarwal, R.K. Srivatsava : *CKD in India – Challenges & Solution ; Nephron clinical practice* 2009; 111, Pg 197-203.
- A. Ramesh Saxena, Robert D.Tota : *Approach to the patient with Kidney Disease*
- : Brenner & Rector's *The Kidney*, 8th Edition; edited by Barry M.Brenner
- ;Elsevier Publications 2008 Vol. I, Pg 713- 717. *olution ; Nephron clinical practice* 2009; 111, Pg 197-203
- Schiffrin EL, Lipman ML and Johannes FE Mann. *Cardiovascular Involvement in General Medical Conditions, Chronic Kidney Disease- Effects on the CVS*. *Circulation*. 2007;116: 85-97
- de Zeeuw D, Ramjit D, Zhang Z, Ribeiro AB, Kurokawa K, Lash JP, Chan J, Remuzzi G, Brenner BM, Shahinfar S: Renal risk and renoprotection among ethnic groups with type 2 diabetic nephropathy: A post hoc analysis of RENAAL. *Kidney Int* 69: 1675–1682, 2006 [PubMed: 16572114]
- de Boer RA, Yu L, van Veldhuisen DJ: Galectin-3 in cardiac remodeling and heart failure. *Curr Heart Fail Rep* 7: 1–8, 2010 [PMCID: PMC2831188] [PubMed: 20425490]
- Nieminen J, Kuno A, Hirabayashi J, Sato S: Visualization of galectin3 oligomerization on the surface of neutrophils and endothelial cells using fluorescence resonance energy transfer. *J Biol Chem* 282: 1374– 1383, 2007 [PubMed: 17082191]
- Ochieng J, Furtak V, Lukyanov P: Extracellular functions of galectin-3. *Glycoconj J* 19: 527–535, 2004 [PubMed: 14758076]
- Sharma UC, Pokharel S, van Brakel TJ, van Berlo JH, Cleutjens JP, Schroen B, André S, Crijns HJ, Gabius HJ, Maessen J, Pinto YM: Galectin-3 marks activated macrophages in failure-prone hypertrophied hearts and contributes to cardiac dysfunction. *Circulation* 110: 3121–3128, 2004 [PubMed: 15520318]
- Henderson NC, Mackinnon AC, Farnworth SL, Kipari T, Haslett C, Iredale JP, Liu FT, Hughes J, Sethi T: Galectin-3 expression and secretion links macrophages to the promotion of renal fibrosis. *Am J Pathol* 172: 288–298, 2008 [PMCID: PMC2312353] [PubMed: 18202187]
- Okamura DM, Pasichnyk K, Lopez-Guisa JM, Collins S, Hsu DK, Liu FT, Eddy AA: Galectin-3 preserves renal tubules and modulates extracellular matrix remodeling in progressive fibrosis. *Am J Physiol Renal Physiol* 300: F245–F253, 2011 [PMCID: PMC3023228] [PubMed: 20962111].