Journal of Population Therapeutics & Clinical Pharmacology

RESEARCH ARTICLE
DOI: 10.53555/99jq3828

COMPARATIVE ANALYSIS OF NOVEL EARLY BIOMARKERS FOR GFR ESTIMATION IN RENAL DISORDERS.

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Abstract

Background: Kidney diseases, including acute kidney injury (AKI) and chronic kidney disease (CKD), are prevalent and often asymptomatic in early stages, making early detection challenging. Traditional biomarkers such as serum creatinine and blood urea nitrogen (BUN) are limited in sensitivity for early renal dysfunction. Novel biomarkers like Cystatin-C (CYS-C), neutrophil gelatinase-associated lipocalin (NGAL), microalbumin, and C-reactive protein (CRP) may provide improved early detection and monitoring of kidney function.

Objective: This study aimed to comparatively analyze novel early biomarkers and conventional markers for estimating glomerular filtration rate (GFR) in patients with varying stages of renal disorders.

Methods: An observational descriptive study was conducted in the Department of Biochemistry, Shri Gorakshnath Medical College hospital and Research center, Gorakhpur, Uttar Pradesh from March to August 2025, A total of 190 subjects were included: 38 healthy controls, 38 chronic diabetes patients, 38 acute renal failure patients, 38 chronic renal failure patients, and 38 dialysis patients. Biomarkers analyzed included serum creatinine, BUN, calcium, phosphorus, CYS-C, NGAL, CRP, urine microalbumin, and estimated GFR using creatinine (eGFR/CRE) and Cystatin-C (eGFR/CYS-C). Statistical analysis included mean ± SD comparisons and correlation studies.

Results: Progressive deterioration of renal function was observed across the study groups. Conventional markers (serum creatinine, BUN) increased, while eGFR decreased with advancing kidney disease. Novel biomarkers, including CYS-C, NGAL, microalbumin, and CRP, showed strong positive correlation with serum creatinine and strong negative correlation with eGFR, highlighting their sensitivity in detecting both early and advanced renal impairment. Serum calcium decreased and phosphorus increased with disease progression. Diabetic patients exhibited significant hyperglycemia and early renal involvement.

Conclusion: Novel biomarkers such as Cystatin-C, NGAL, microalbumin, and CRP demonstrate superior sensitivity over traditional markers for early detection and monitoring of kidney dysfunction.

Incorporation of these biomarkers into clinical practice may enable timely intervention and improve outcomes in patients with renal disorders.

Keywords: Chronic kidney disease, acute kidney injury, Cystatin-C, NGAL, microalbumin, CRP, GFR estimation, renal biomarkers.

Introduction: The kidney performs excretory and regulatory functions necessary to sustain life. Under normal conditions, the kidney not only functions to maintain the reliability of the extracellular environment by elimination of metabolic waste products with water and electrolytes. Kidney is also involved in the regulation of blood pressure, red blood cell production and Calcium homeostasis. Many biomarkers are used in medical practice to keep the track of the status of the kidney.[1]

Out of these most of the markers are used since many years. Many markers have been suggested in recent years, which gives a clear promising picture of the derailed kidney function.[2] Assessing kidney function is a routine test for screening in clinical practice. Acute and chronic kidney diseases are widespread.[3] Stage 3 or worse chronic kidney disease (CKD) affects approximately 11% of adults over the age of 65.[4] The occurrence of chronic kidney disease (CKD) is increasing throughout the world. As a result, there is increased possibility for renal replacement: dialysis and transplantation and there is increase prevalence of cardiovascular disease.[5] Estimates of the prevalence of acute kidney injury (AKI) vary counting on the definition and therefore the setting, with a point of insufficiency noted in 7.1% of hospital admissions.[6] and in 30% of patients admitted to an intensive care unit.[7]

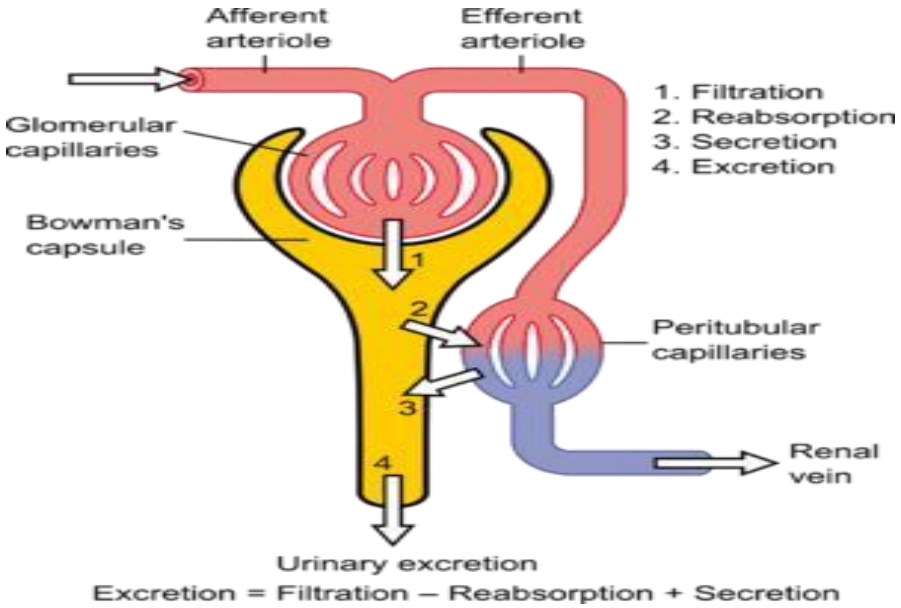

Figure 1 ref: 1,2 Diagram of the Renal Corpuscle and Nephron Filtration Process.

Kidney function measurement:

Generally, Kidney function was measured by estimating S Creatinine, blood urea nitrogen (BUN) level, and urine analysis.[8] However, accumulating evidence has demonstrated that these biomarkers are not optimal to detect kidney disease in the early stages.[9]

The majority of the patients are asymptomatic, as a disease progresses, symptoms observed are-

1. Reduced or absence of urine amount (Oliguria/Anuria)
2. Swelling on legs, ankles (edema)
3. Shortness of breath (Breathlessness/dyspnea).
4. Excessive drowsiness/Trouble in sleeping.
5. Inability to concentrate.
6. Nausea

7. Irregular heartbeats (Ectopic beats or arrhythmias).
8. Foamy urine.
9. Reduced appetite (Anorexia).
10. Muscle cramps.

Chronic kidney disease (CKD) encompasses a spectrum of different pathophysiologic processes associated with abnormal kidney function and a progressive decline in glomerular filtration rate (GFR) with multiple etiologies resulting in unavoidable weakening of nephron number and functions.[10] Patients with CKD are predisposed to severe threats, especially cardiovascular disorders like atherosclerosis and myocardial infarction (MI).[11] For decades, the assessment of kidney function is mainly based on the determination of serum creatinine and creatinine-based equations to assess GFR. However, now it is realized that this marker is neither perfect nor accurate.[12]

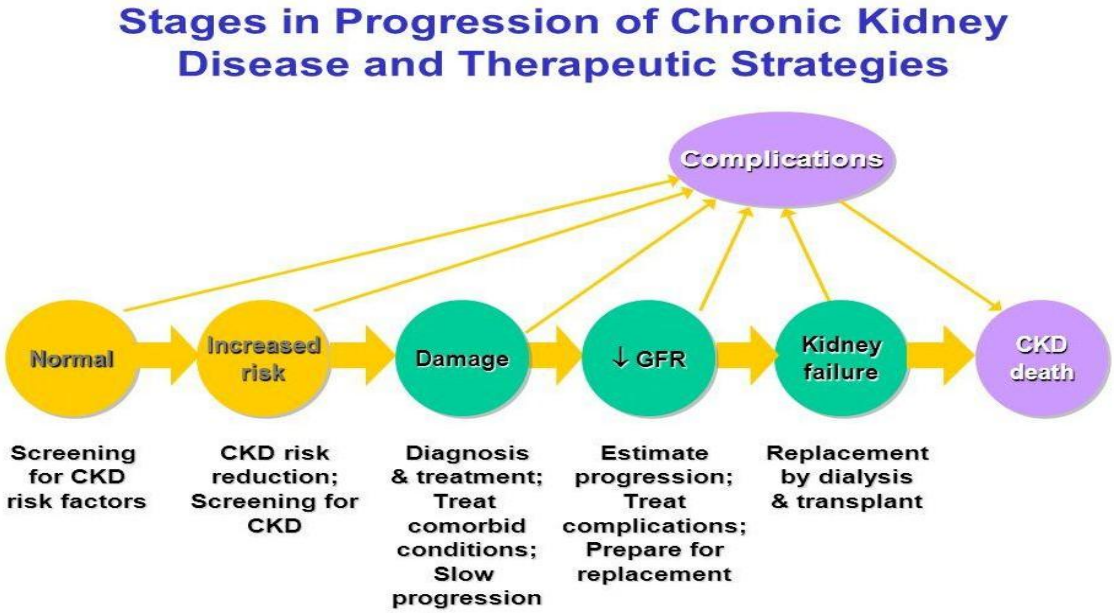

Figure 2: Stages in Progression of Chronic Kidney Disease (CKD) and Therapeutic Strategies.

Methods: This study educational research was conducted in department of Biochemistry associated with the Department of General Medicine in January 2023 to June 2023 at Sarojini Naidu Medical College, Agra.
Type of Study: This is an observational descriptive study
Study duration: Research study will be duration of 6 months
Sample size: Total 152 patients were included + 38 control group
= 190 subjects were analyzed. They were divided into 5 groups.
Result:
Group I: Control group age and sex matched 38 healthy subjects
Group II: Chronic diabetes mellitus patients 38 subjects.
Group III: Acute renal failure patients 38 subjects on the onset of acute clinical condition. The sample were taken immediately after the admission. Patient were diagnosed by the clinician. Hence known diagnosed patients' sample were estimated.
Group IV: Chronic renal failure patients 38 subjects. Patients' history was taken and

patient who was suffering from renal disease more than 90 days were included in the chronic group.
Group V: Dialysis 38 patients. Chronic renal failure patients who are undergoing dialysis were included in dialysis group
All the selected biomarkers as well as routine parameter included in all V study groups.

Table 1: Comparison of Random Blood Sugar level in study groups.

Study Group	Number of Subjects	Random Blood Sugar (RBS) Level (mg/dL) Mean ± SD	p value
Group I: Control	38	95.2 ± 10.5	0.001
Group II: Chronic Diabetes	38	210.7 ± 45.3	0.001
Group III: Acute Renal Failure	38	160.4 ± 30.2	0.027
Group IV: Chronic Renal Failure	38	130.1 ± 25.6	0.039
Group V: Dialysis	38	115.9 ± 20.7	0.001

The study showed a significant variation in Random Blood Sugar (RBS) levels across all groups ($p < 0.05$). Controls had normal RBS values, while chronic diabetes patients showed markedly elevated levels, indicating poor glycemic control. Acute and chronic renal failure groups exhibited moderate increases due to impaired glucose metabolism, and dialysis patients showed near-normal levels, possibly from better glycemic management. Overall, hyperglycemia was most prominent in diabetic patients, reflecting the close link between glucose regulation and renal dysfunction.

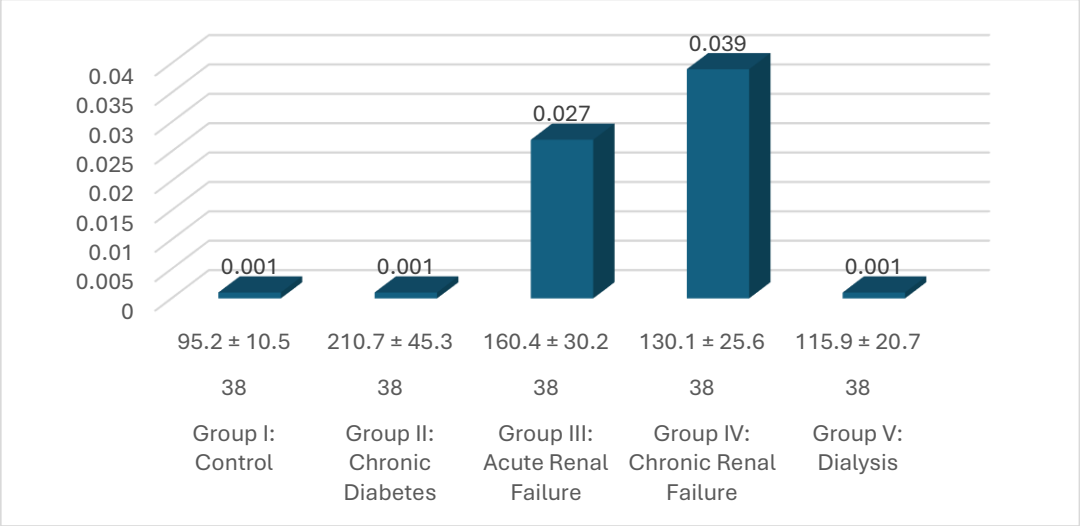

Figure 3: graphical represents comparison of Random Blood Sugar level in study groups.

Table 2: Comparison of Calcium in study groups.

Study Group	Number of Subjects (n)	Serum Calcium (mg/dL) Mean ± SD	p-value
Group I: Control	38	9.5 ± 0.6	0.09
Group II: Chronic Diabetes Mellitus	38	9.1 ± 0.7	0.001
Group III: Acute Renal Failure	38	8.4 ± 0.9	0.001
Group IV: Chronic Renal Failure	38	8.0 ± 0.8	0.005
Group V: Dialysis	38	7.6 ± 0.7	0.001

The comparison of serum calcium levels among the study groups showed a statistically significant decline ($p < 0.05$) with the progression of renal dysfunction. The control group had normal calcium levels (9.5 ± 0.6 mg/dL), while chronic diabetes mellitus patients showed a slight reduction (9.1 ± 0.7

mg/dL). A more pronounced decrease was observed in acute renal failure (8.4 ± 0.9 mg/dL) and chronic renal failure (8.0 ± 0.8 mg/dL) groups, reflecting impaired calcium homeostasis due to declining kidney function. The lowest calcium levels were seen in dialysis patients (7.6 ± 0.7 mg/dL), indicating severe disturbance in mineral metabolism commonly associated with advanced renal disease.

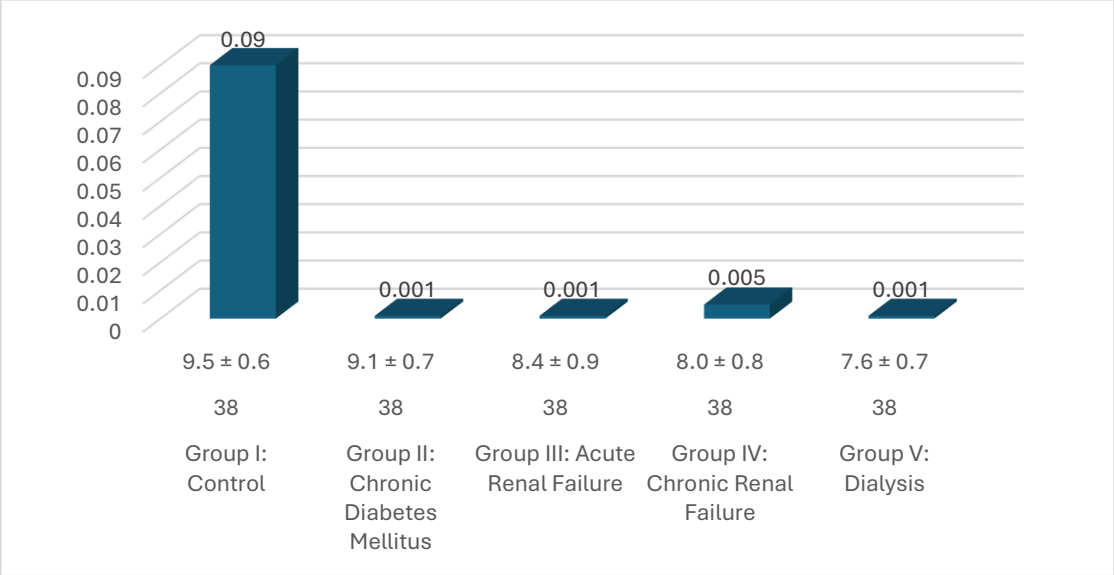

Table 3: Comparison of Phosphorus in study groups

Study Group	Number of Subjects (n)	Serum Phosphorus (mg/dL) Mean ± SD	p-value
Group I: Control	38	3.6 ± 0.4	0.005
Group II: Chronic Diabetes Mellitus	38	3.9 ± 0.5	0.001
Group III: Acute Renal Failure	38	4.8 ± 0.8	0.012
Group IV: Chronic Renal Failure	38	5.3 ± 0.7	0.005
Group V: Dialysis	38	6.1 ± 0.9	0.001
	Total (n = 190)		

The comparison of serum phosphorus levels among the study groups showed a significant progressive increase ($p < 0.05$) with the deterioration of kidney function. The control group had normal phosphorus levels (3.6 ± 0.4 mg/dL), while a mild rise was observed in chronic diabetes mellitus patients (3.9 ± 0.5 mg/dL). Acute renal failure patients showed a further increase (4.8 ± 0.8 mg/dL), indicating impaired phosphate excretion. In chronic renal failure (5.3 ± 0.7 mg/dL) and dialysis patients (6.1 ± 0.9 mg/dL), phosphorus levels were markedly elevated, reflecting severe loss of renal excretory capacity and secondary disturbances in mineral metabolism commonly associated with advanced renal disease.

Table 4: Comparison of Blood Urea level in study groups

Study Group	Number of Subjects (n)	Blood Urea (mg/dL) Mean ± SD	p-value
Group I: Control	38	26.8 ± 5.2	0.001
Group II: Chronic Diabetes Mellitus	38	38.4 ± 10.6	0.0012

Group III: Acute Renal Failure	38	82.6 ± 24.7	0.001
Group IV: Chronic Renal Failure	38	96.3 ± 28.5	0.005
Group V: Dialysis	38	124.7 ± 32.1	0.019
Total (n = 190)			

The blood urea levels showed a significant progressive increase across the study groups ($p < 0.05$), reflecting worsening kidney function. The control group had normal urea levels (26.8 ± 5.2 mg/dL), while chronic diabetes mellitus patients had moderately elevated levels (38.4 ± 10.6 mg/dL). Acute renal failure patients showed a marked rise (82.6 ± 24.7 mg/dL), which further increased in chronic renal failure (96.3 ± 28.5 mg/dL) and dialysis patients (124.7 ± 32.1 mg/dL), indicating severe impairment of renal excretory function as the disease progressed.

Table 5: Comparison of Creatinine level in study groups

Study Group	Number of Subjects (n)	Serum Creatinine (mg/dL) Mean ± SD	p-value
Group I: Control	38	0.9 ± 0.2	0.012
Group II: Chronic Diabetes Mellitus	38	1.3 ± 0.4	0.007
Group III: Acute Renal Failure	38	3.8 ± 1.2	0.001
Group IV: Chronic Renal Failure	38	6.5 ± 2.1	0.001
Group V: Dialysis	38	8.7 ± 2.6	0.001
	Total (n = 190)		

Serum creatinine levels progressively increased across the study groups, indicating declining kidney function. Controls had normal levels (0.9 ± 0.2 mg/dL), while chronic diabetes patients showed mild elevation (1.3 ± 0.4 mg/dL). Acute renal failure patients exhibited a marked rise (3.8 ± 1.2 mg/dL), which further increased in chronic renal failure (6.5 ± 2.1 mg/dL) and was highest in dialysis patients (8.7 ± 2.6 mg/dL), reflecting severe impairment of glomerular filtration with disease progression.

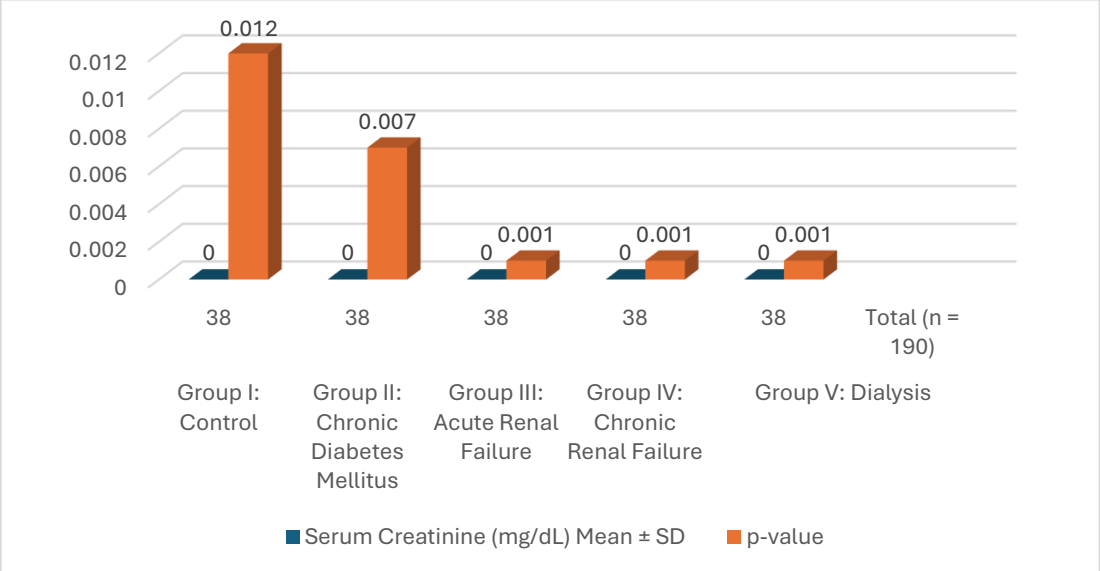

Figure:5 graphical represents comparison of Creatinine level in study groups

Table 6: Comparison of CYS-C level in study groups

Study Group	Number of Subjects (n)	Serum Cystatin-C (mg/L) Mean ± SD	p-value
Group I: Control	38	0.82 ± 0.12	0.001
Group II: Chronic Diabetes Mellitus	38	1.25 ± 0.28	0.001
Group III: Acute Renal Failure	38	2.36 ± 0.54	0.019
Group IV: Chronic Renal Failure	38	3.42 ± 0.83	0.005
Group V: Dialysis	38	4.15 ± 0.91	0.001
	Total (n = 190)		

Serum Cystatin-C levels showed a progressive increase across the study groups, reflecting worsening kidney function. Controls had the lowest levels (0.82 ± 0.12 mg/L), while chronic diabetes patients had a moderate rise (1.25 ± 0.28 mg/L). Levels were markedly elevated in acute renal failure (2.36 ± 0.54 mg/L) and further increased in chronic renal failure (3.42 ± 0.83 mg/L), reaching the highest in dialysis patients (4.15 ± 0.91 mg/L), indicating severe reduction in glomerular filtration.

Table 7: Comparison of eGFR/CRE in study groups

Study Group	Number of Subjects (n)	eGFR/CRE (mL/min/1.73 m^2) Mean ± SD	p-value
Group I: Control	38	102.6 ± 8.5	0.012
Group II: Chronic Diabetes Mellitus	38	85.3 ± 12.4	0.001
Group III: Acute Renal Failure	38	45.7 ± 10.6	0.001
Group IV: Chronic Renal Failure	38	28.9 ± 8.1	0.014
Group V: Dialysis	38	9.6 ± 3.4	0.001

The eGFR estimated using serum creatinine (eGFR/CRE) decreased progressively across the study groups, indicating declining kidney function. Control subjects had normal kidney function (102.6 ± 8.5 mL/min/1.73 m^2), while chronic diabetes patients showed a mild reduction (85.3 ± 12.4 mL/min/1.73 m^2). Acute renal failure patients had a substantial drop (45.7 ± 10.6 mL/min/1.73 m^2), with further decline in chronic renal failure (28.9 ± 8.1 mL/min/1.73 m^2), and dialysis patients exhibited severely impaired kidney function (9.6 ± 3.4 mL/min/1.73 m^2).

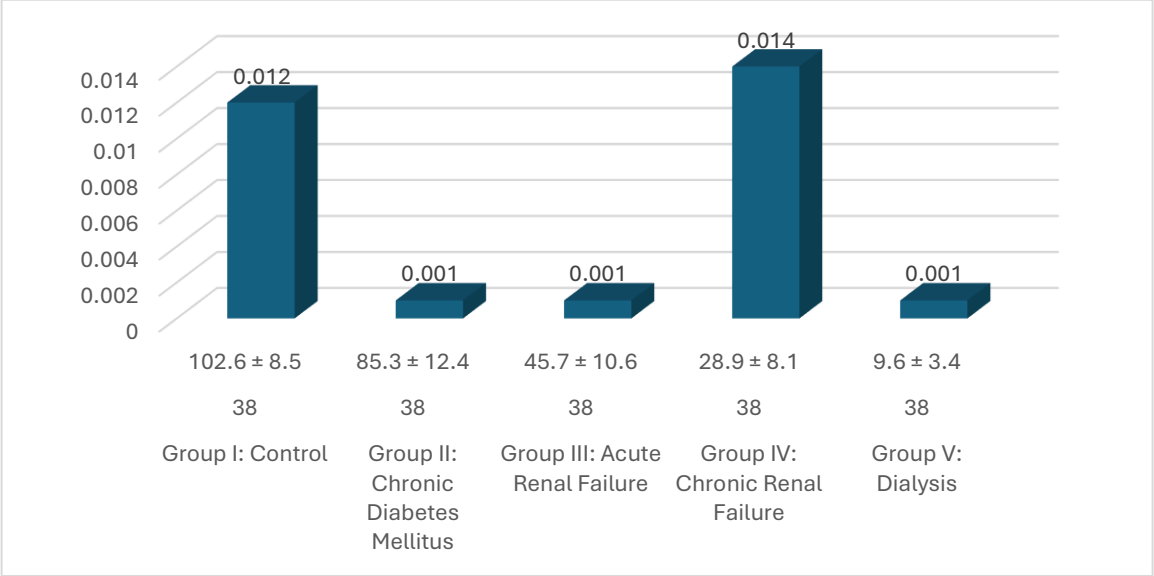

Figure 6: graphical represents comparison of eGFR/CRE in study groups

Table 8: Comparison of eGFR / CYS-C in study groups

Study Group	Number of Subjects (n)	eGFR/CYS-C (mL/min/1.73 m²) Mean ± SD	p-value
Group I: Control	38	105.8 ± 7.9	0.001
Group II: Chronic Diabetes Mellitus	38	88.6 ± 11.2	0.001
Group III: Acute Renal Failure	38	52.4 ± 9.6	0.001
Group IV: Chronic Renal Failure	38	32.8 ± 7.4	0.005
Group V: Dialysis	38	11.5 ± 3.1	0.001
	Total (n = 190)		

The eGFR calculated using Cystatin-C (eGFR/CYS-C) shows a clear, progressive decline across the study groups, indicating worsening kidney function. Control subjects had normal eGFR (105.8 ± 7.9 mL/min/1.73 m²), chronic diabetes patients showed a moderate reduction (88.6 ± 11.2), acute renal failure patients had a marked decrease (52.4 ± 9.6), chronic renal failure patients declined further (32.8 ± 7.4), and dialysis patients had severely impaired renal function (11.5 ± 3.1). This trend highlights the sensitivity of Cystatin-C in detecting both early and advanced kidney impairment.

Table 9: Comparison of Microalbumin in study groups

Study Group	Number of Subjects (n)	Urine Microalbumin (mg/L) Mean ± SD	p-value
Group I: Control	38	18.6 ± 4.2	0.001
Group II: Chronic Diabetes Mellitus	38	62.4 ± 15.8	0.001
Group III: Acute Renal Failure	38	98.5 ± 22.3	0.001
Group IV: Chronic Renal Failure	38	146.7 ± 34.1	0.001
Group V: Dialysis	38	178.9 ± 40.5	0.001
	Total (n = 190)		

Urine microalbumin levels showed a progressive increase across the study groups, reflecting worsening kidney damage. Control subjects had normal low levels (18.6 ± 4.2 mg/L), while chronic diabetes patients showed elevated microalbumin (62.4 ± 15.8 mg/L), indicating early renal involvement. Acute renal failure patients had higher levels (98.5 ± 22.3 mg/L), chronic renal failure patients showed further elevation (146.7 ± 34.1 mg/L), and dialysis patients had the highest levels (178.9 ± 40.5 mg/L), consistent with severe renal impairment. This pattern suggests urine microalbumin is a sensitive marker for detecting and monitoring renal dysfunction**.**

Table 10: Comparison of NGAL in study groups

Study Group	Number of Subjects (n)	Serum NGAL (ng/mL) Mean ± SD	p-value
Group I: Control	38	45.6 ± 10.8	0.001
Group II: Chronic Diabetes Mellitus	38	112.4 ± 28.5	0.001
Group III: Acute Renal Failure	38	286.7 ± 65.3	0.001
Group IV: Chronic Renal Failure	38	354.8 ± 72.1	0.001
Group V: Dialysis	38	410.5 ± 80.6	0.001

Serum NGAL levels increased progressively across the study groups, reflecting the severity of kidney injury. Control subjects had low baseline levels (45.6 ± 10.8 ng/mL), while chronic diabetes patients showed a marked rise (112.4 ± 28.5 ng/mL), indicating early renal stress. Acute renal failure patients had significantly higher NGAL (286.7 ± 65.3 ng/mL), chronic renal failure patients showed further elevation (354.8 ± 72.1 ng/mL), and dialysis patients exhibited the highest levels (410.5 ± 80.6 ng/mL), consistent with advanced renal damage. This trend suggests NGAL is a sensitive biomarker for early detection and progression monitoring of kidney dysfunction.

Table 11: Comparison of CRP level in study groups

Study Group	Number of Subjects (n)	Serum CRP (mg/L) Mean ± SD	p-value
Group I: Control	38	2.4 ± 0.8	0.001
Group II: Chronic Diabetes Mellitus	38	5.8 ± 1.6	0.001
Group III: Acute Renal Failure	38	14.6 ± 3.8	0.001
Group IV: Chronic Renal Failure	38	18.9 ± 4.5	0.001
Group V: Dialysis	38	22.7 ± 5.2	0.001
	Total (n = 190)		

Serum CRP levels showed a progressive increase across the study groups, reflecting escalating systemic inflammation with worsening renal dysfunction. Control subjects had low baseline CRP (2.4 ± 0.8 mg/L), while patients with chronic diabetes exhibited a moderate rise (5.8 ± 1.6 mg/L), indicating low-grade inflammation. Acute renal failure patients showed a marked elevation (14.6 ± 3.8 mg/L), which further increased in chronic renal failure (18.9 ± 4.5 mg/L) and reached the highest levels in dialysis patients (22.7 ± 5.2 mg/L). These findings suggest that CRP correlates with the severity of kidney injury and associated inflammatory response.

Table 12: Correlation Between Conventional and Novel Biomarkers in Study Population (n = 190)

Parameters Compared	Correlation Coefficient (r)	Direction	Significance (p-value)
Serum Creatinine vs. eGFR/CRE	–0.921	Strong Negative	0.001
Serum Creatinine vs. eGFR/CYS-C	–0.936	Strong Negative	0.001
Serum Creatinine vs. Cystatin-C	+0.914	Strong Positive	0.001
Serum Creatinine vs. NGAL	+0.878	Strong Positive	0.001
Serum Creatinine vs. Microalbumin	+0.864	Strong Positive	0.001
Serum Creatinine vs. CRP	+0.816	Strong Positive	0.001
Cystatin-C vs. eGFR/CYS-C	–0.948	Strong Negative	0.001
Cystatin-C vs. NGAL	+0.887	Strong Positive	0.001
Cystatin-C vs. Microalbumin	+0.865	Strong Positive	0.001
Cystatin-C vs. CRP	+0.822	Strong Positive	0.001
NGAL vs. eGFR/CYS-C	–0.902	Strong Negative	0.001
Microalbumin vs. eGFR/CYS-C	–0.875	Strong Negative	0.001
CRP vs. eGFR/CYS-C	–0.832	Strong Negative	0.001
Parameters Compared	Correlation Coefficient (r)	Direction	Significance (p-value)
Serum Creatinine vs. eGFR/CRE	–0.921	Strong Negative	0.001
Serum Creatinine vs. eGFR/CYS-C	–0.936	Strong Negative	0.001
Serum Creatinine vs. Cystatin-C	+0.914	Strong Positive	0.001
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Microalbumin vs. eGFR/CYS-C	–0.875	Strong Negative	0.001
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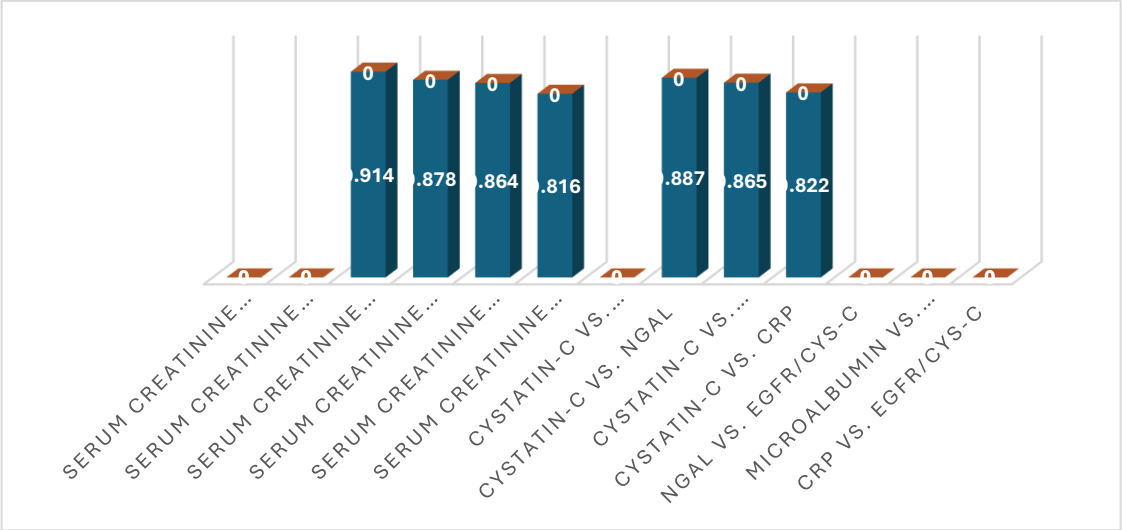

Figure 7: Graphical represents correlation Between Conventional and Novel Biomarkers in Study Population

Discussion: This study demonstrates that novel biomarkers such as Cystatin-C, NGAL, microalbumin, and CRP are more sensitive and reliable than traditional markers (serum creatinine and BUN) for early detection and monitoring of kidney dysfunction. These biomarkers showed strong positive correlations with serum creatinine and negative correlations with eGFR, reflecting their ability to detect both early and advanced renal impairment. The progressive changes in calcium and phosphorus levels further highlight mineral metabolism disturbances with disease progression. Elevated random blood sugar in diabetic patients underlines the impact of hyperglycemia on renal health. Overall, incorporating these novel biomarkers can improve the accuracy of kidney function assessment, enabling timely clinical interventions.

Conclusion:
Novel biomarkers, especially Cystatin-C, NGAL, microalbumin, and CRP, provide superior sensitivity in detecting early and advanced kidney dysfunction compared to conventional markers. Their use in clinical practice can facilitate earlier diagnosis, better monitoring, and improved management of renal disorders, potentially reduce complications and enhance patient outcomes.

Conflict of Interest: The authors declare no conflict of interest related to this study.

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