

Naogaon Medical College Journal

Frequency: Bi-Annual Vol-2 | Issue-2 | Jul-Dec 2025 https://nmcjournal.org

Research Article



pISSN: 3105-2568

Hematological and Urinary Biomarker Changes in Diabetic vs. Non-Diabetic Dialysis Patients: A Cross-Sectional Study

Traye Trapa^{1*}, Syeda Anisa Ali¹, Protity Roy Prome¹, Farhana Ferdaus²

15th Year MBBS, Khulna City Medical College, Khulna 2 Associate Professor and Head, Community Medicine and Public Health, Khulna City Medical College, Khulna



$* Corresponding \ author:$

Traye Trapa Email: trapatraye@gmail.com

How to cite this article:

Trapa T, Ali SA, Prome PR, Ferdaus F; Hematological and Urinary Biomarker Changes in Diabetic vs. Non-Diabetic Dialysis Patients: A Cross-Sectional Study. Naog. Med. Coll. J. 2025;2(2): 4-10

Article history:

Received: April 27, 2025 Accepted: July 29, 2025 Published: August 31, 2025

Peer Review Process:

The Journal abides by a double-blind peer review process such that the journal does not disclose the identity of the reviewer(s) to the author(s) and does not disclose the identity of the author(s) to the reviewer(s).

ABSTRACT: Background: Diabetes mellitus (DM) is associated with progressive kidney damage, anemia, and altered immune function, often complicating dialysis outcomes. Hematological and urinary biomarkers can reflect the severity of these complications. Objective: This study aimed to compare hematological and urinary biomarkers between diabetic and non-diabetic dialysis patients. Methods: A cross-sectional study was conducted at Khulna City Medical College (January-June 2025), including 250 dialysis patients (130 diabetic, 120 non-diabetic). Data were extracted from the most recent onemonth laboratory investigations. Hematological parameters (hemoglobin, hematocrit, RBC, WBC, neutrophils, lymphocytes, platelets) and urinary biomarkers (albumin, protein, creatinine, microalbuminuria) were analyzed. Statistical comparisons were performed using t-tests and chi-square tests; p<0.05 was considered significant. Results: Diabetic patients were older (58.3 ± 9.4 vs. 52.1 ± 10.8 years, p<0.001) and had longer dialysis duration (38.2 \pm 14.6 vs. 34.5 \pm 12.9 months, p=0.040). Hypertension (86.2% vs. 65.0%, p<0.001) and cardiovascular disease (31.5% vs. 18.3%, p=0.020) were more prevalent in diabetics. Hemoglobin $(9.1 \pm 1.2 \text{ vs. } 9.8 \pm 1.3 \text{ g/dL}, \text{ p} < 0.001)$, hematocrit (28.1 $\pm 3.8\%$ vs. $30.2 \pm 4.1\%$, p=0.002), and RBC count (3.0 ± 0.5 vs. $3.3 \pm 0.6 \times 10^6/\mu$ L, p=0.001) were lower in diabetic patients. WBC (7.9 \pm 2.1 vs. 7.3 \pm 1.8 \times 10³/ μ L, p=0.040) and neutrophil percentage (63.8 ± 8.4% vs. 61.1 ± 7.9%, p=0.030) were higher, while lymphocytes were lower (27.5 \pm 6.2% vs. 29.6 \pm 6.7%, p=0.020). Urinary albumin (1126.0 $\pm 347.0 \text{ vs. } 882.0 \pm 294.0 \text{ mg/L}, p<0.001), protein (86.0 \pm 24.0 \text{ vs. } 71.0 \pm 22.0 \text{ mg/dL}, p<0.001),$ and microalbuminuria (345.2 \pm 98.6 vs. 276.4 \pm 87.3 mg/g creatinine, p<0.001) were significantly higher, whereas urinary creatinine was lower (98.4 ± 21.2 vs. 104.7 ± 19.6 mg/dL, p=0.020) in diabetics. Conclusion: Diabetic dialysis patients demonstrate more pronounced anemia, altered leukocyte profiles, and greater proteinuria compared to non-diabetic patients, reflecting higher renal and inflammatory burden.

Keywords: Diabetes Mellitus, Dialysis, Hematological Biomarkers, Urinary Biomarkers, Proteinuria, Microalbuminuria.

Copyright © 2025 The Author(s): This is an open-access article distributed under the terms of the Creative Commons Attribution 4.0 International License (CC BY-NC 4.0) which permits unrestricted use, distribution, and reproduction in any medium for non-commercial use provided the original author and source are credited.

INTRODUCTION

Diabetes mellitus (DM) is a group of metabolic disorders characterized by high blood glucose levels

caused by problems with insulin production, action, or both.¹ The worldwide prevalence and incidence of DM have sharply increased in recent decades.² In 2013,

approximately 382 million people had diabetes globally, and this number is expected to reach 592 million by 2035, mostly in developing countries. For example, in Ethiopia, among adults aged 20-79 years, diabetes prevalence was 4.4% in 2013 and is projected to increase to 5.1% by.3 Globally, the number of adults with diabetes rose from 108 million in 1980 to 422 million in.4-6 The annual healthcare costs linked to DM range from to 1,099 billion.⁷⁻⁹ Chronic high blood sugar levels in DM can cause progressive damage, dysfunction, and failure of vital organs such as the kidneys, eyes, nerves, heart, and blood vessels.9 Longterm complications include nephropathy leading to kidney failure, retinopathy causing vision loss, autonomic neuropathy resulting in gastrointestinal cardiovascular issues, and peripheral neuropathy leading to foot ulcers.¹⁰ Most individuals with type 1 and type 2 DM develop these complications over time.¹¹ Furthermore, diabetes is often linked with hypertension, cardiovascular disease, and peripheral vascular disease, while also placing significant psychological and financial stress on patients and their families.^{12, 13} Regular monitoring and control of blood glucose, blood pressure, and lipid levels can help prevent or delay these complications.¹³. Diabetic kidney disease (DKD) is a common and serious complication, being the main cause of kidney failure and increased morbidity and death among diabetic patients. DKD affects 15-25% of those with type 1 diabetes and 30-40% of those with type 2 DM.14 Its development is influenced by environmental and genetic factors. Keeping blood glucose levels within target ranges greatly reduces the risk of DKD, and disease progression can be slowed by managing blood pressure effectively. 12-15

Early diagnosis of DKD enables timely intervention and improves prognosis. Identifying sensitive and reliable biomarkers for monitoring renal dysfunction in DM patients remains a challenge.13 Proteinuria serves as a primary marker of DKD and a key indicator of kidney disease progression.14, 17, 29 While microalbuminuria is an important biomarker of early kidney injury.^{18, 11} Microalbuminuria predicts renal impairment and is associated with premature morbidity and mortality among diabetic, hypertensive, and even healthy individuals.¹⁶ Evidence indicates that reducing urinary albumin levels lowers the risk of adverse renal outcomes. 20, 22-24, 26 Given the complex nature of diabetes, a multi-factorial management approach is essential. Comprehensive strategies-including blood glucose and

blood pressure control, lipid management, low-protein and low-salt diet, regular physical activity, weight management, and avoidance of smoking—can slow DKD progression and improve overall outcomes in diabetic patients. Patients at the study aims to evaluate hematological and urinary biomarker changes in diabetic versus non-diabetic dialysis patients, providing insights into the differential burden of renal injury, anemia, and inflammatory status in this high-risk population.

METHODOLOGY

Study Design

This cross-sectional study was conducted to evaluate hematological and urinary biomarker changes in diabetic versus non-diabetic dialysis patients at Khulna City Medical College, Khulna, Bangladesh. The study utilized a comparative approach, analyzing secondary data extracted from medical records of patients receiving dialysis. The study period spanned from January to June 2025, focusing on data collected from the most recent onemonth investigation reports prior to the study's data collection phase.

Study Population

The study population consisted of 250 patients undergoing dialysis at the dialysis unit of Khulna City Medical College. Patients were divided into two groups: diabetic dialysis patients and non-diabetic dialysis patients. Inclusion criteria encompassed adult patients (aged 18 years and above) receiving maintenance hemodialysis for at least three months and having complete investigation reports from the last one month available in their medical records. Exclusion criteria included patients with incomplete medical records, those with acute infections or malignancies, or those who had undergone dialysis for less than three months.

Data Collection

Secondary data were retrieved from the medical records of the dialysis unit at Khulna City Medical College. No primary laboratory tests were conducted for this study. Data were collected from investigation reports generated within the last one month of each patient's dialysis treatment, ensuring consistency and recency of the biomarker data. The following hematological and urinary biomarkers were extracted: Hematological Biomarkers: Hemoglobin, hematocrit, red blood cell count,

white blood cell count, platelet count, and mean corpuscular volume. Urinary Biomarkers: Urinary albumin, creatinine, and protein levels, where applicable, based on available records. Additional data collected included demographic details (age, sex), clinical characteristics (duration of dialysis, diabetes status), and relevant comorbidities. Data extraction was performed by trained personnel using a standardized data collection form to ensure accuracy and uniformity.

Data Management

Data were anonymized to protect patient confidentiality, with each patient assigned a unique identifier. Extracted data were entered into a secure electronic database, with double-entry verification to minimize errors. Any discrepancies in the records were resolved by cross-referencing with original patient files. Missing or incomplete data led to the exclusion of the respective patient's record from the analysis to maintain data integrity.

Statistical Analysis

Descriptive statistics were used to summarize demographic and clinical characteristics, with means and standard deviations for continuous variables and frequencies and percentages for categorical variables. Hematological and urinary biomarker levels were compared between diabetic and non-diabetic groups using appropriate statistical tests. For normally distributed data, independent t-tests were applied, while non-parametric tests (e.g., Mann-Whitney U test) were used for non-normally distributed data. Categorical data were analyzed using chi-square tests. A p-value of <0.05 was considered statistically significant. Statistical analyses were conducted using SPSS version 26.0.

Ethical Considerations

As the study relied on secondary data, informed consent was not required; however, strict measures were taken to ensure patient confidentiality and data security throughout the study.

RESULTS

A total of 250 dialysis patients were included, with 130 (52.0%) diabetic and 120 (48.0%) non-diabetics, based on medical records from Khulna City Medical College (January–June 2025). Data were extracted from the most recent one-month investigation reports. The results cover demographic, clinical, hematological, and urinary biomarkers, with statistical comparisons between diabetic and non-diabetic groups. Additional biomarkers (neutrophils, lymphocytes, and microalbuminuria) were included as they were available in the secondary data.

Table 1: Distribution of Patients by Demographic and Clinical Characteristics

Variable	Diabetic	Non-Diabetic	Total	Р -
	(n=130)	(n=120)	(n=250)	Value
Age (years, mean ± SD)	58.3 ± 9.4	52.1 ± 10.8	55.3 ± 10.5	<0.001*
Sex, n (%)				0.840
Male	72 (55.4%)	68 (56.7%)	140 (56.0%)	
Female	58 (44.6%)	52 (43.3%)	110 (44.0%)	
Duration of Dialysis (months,	38.2 ± 14.6	34.5 ± 12.9	36.4 ± 13.9	0.040*
mean ± SD)				
Comorbidities, n (%)				
Hypertension	112 (86.2%)	78 (65.0%)	190 (76.0%)	<0.001*
Cardiovascular Disease	41 (31.5%)	22 (18.3%)	63 (25.2%)	0.020*
Chronic Kidney Disease Stage 5	130 (100%)	120 (100%)	250 (100%)	1

Table 1 summarizes demographic and clinical characteristics. Diabetic patients were significantly older $(58.3 \pm 9.4 \text{ years vs. } 52.1 \pm 10.8 \text{ years, p} < 0.001)$ and had a longer dialysis duration $(38.2 \pm 14.6 \text{ months vs. } 34.5 \pm 12.9 \text{ months, p} = 0.040)$. Hypertension (86.2% vs. 65.0%, p < 0.001)

and cardiovascular disease (31.5% vs. 18.3%, p=0.020) were more prevalent in diabetic patients. Sex distribution was similar (p=0.840). All patients had stage 5 CKD, as expected for dialysis patients. These findings suggest a

higher burden of comorbidities in diabetic patients, potentially impacting biomarker profiles.

Table 2: Hematological Parameters Among Diabetic and Non-Diabetic Dialysis Patients

Parameter	Diabetic (n=130, mean	Non-Diabetic (n=120, mean	p-
	± SD)	± SD)	value
Hemoglobin (g/dL)	9.1 ± 1.2	9.8 ± 1.3	<0.001*
Hematocrit (%)	28.1 ± 3.8	30.2 ± 4.1	0.002*
Red Blood Cell Count	3.0 ± 0.5	3.3 ± 0.6	0.001*
(×10 ⁶ /μL)			
White Blood Cell Count	7.9 ± 2.1	7.3 ± 1.8	0.040*
$(\times 10^3/\mu L)$			
Neutrophils (%)	63.8 ± 8.4	61.1 ± 7.9	0.030*
Lymphocytes (%)	27.5 ± 6.2	29.6 ± 6.7	0.020*
Platelet Count (×10³/μL)	183.0 ± 52.0	196.0 ± 56.0	0.070
Mean Corpuscular Volume	88.2 ± 6.1	89.8 ± 5.7	0.110
(fL)			

Table 2 compares hematological biomarkers. Diabetic patients had significantly lower hemoglobin (9.1 \pm 1.2 g/dL vs. 9.8 \pm 1.3 g/dL, p<0.001), hematocrit (28.1 \pm 3.8% vs. 30.2 \pm 4.1%, p=0.002), and red blood cell count (3.0 \pm 0.5 ×106/µL vs. 3.3 \pm 0.6 ×106/µL, p=0.001). White blood cell count (7.9 \pm 2.1 ×103/µL vs. 7.3 \pm 1.8 ×103/µL, p=0.040) and neutrophil percentage (63.8 \pm 8.4% vs. 61.1 \pm 7.9%,

p=0.030) were higher in diabetics, while lymphocyte percentage ($27.5 \pm 6.2\%$ vs. $29.6 \pm 6.7\%$, p=0.020) was lower. Platelet count and MCV showed no significant differences (p=0.070 and p=0.110, respectively). These findings indicate more pronounced anemia and altered immune profiles in diabetic dialysis patients, potentially due to chronic inflammation.

Table 3: Urinary Biomarker Levels Among Diabetic and Non-Diabetic Dialysis Patients

Biomarker	Diabetic (n=130, mean	Non-Diabetic (n=120,	p-
	± SD)	mean ± SD)	value
Urinary Albumin (mg/L)	1126.0 ± 347.0	882.0 ± 294.0	<0.001*
Urinary Protein (mg/dL)	86.0 ± 24.0	71.0 ± 22.0	<0.001*
Urinary Creatinine (mg/dL)	98.4 ± 21.2	104.7 ± 19.6	0.020*
Microalbuminuria (mg/g	345.2 ± 98.6	276.4 ± 87.3	<0.001*
creatinine)			

Table 3 compares urinary biomarkers. Diabetic patients had significantly higher urinary albumin (1126.0 \pm 347.0 mg/L vs. 882.0 \pm 294.0 mg/L, p<0.001), urinary protein (86.0 \pm 24.0 mg/dL vs. 71.0 \pm 22.0 mg/dL, p<0.001), and microalbuminuria (345.2 \pm 98.6 mg/g creatinine vs. 276.4 \pm 87.3 mg/g creatinine, p<0.001). Urinary creatinine was lower in diabetics (98.4 \pm 21.2 mg/dL vs. 104.7 \pm 19.6 mg/dL, p=0.020). These results suggest greater proteinuria and kidney damage in diabetic dialysis patients, consistent with diabetic nephropathy.

DISCUSSION

Our cross-sectional analysis of 250 maintenance-dialysis patients—130 with diabetes (52.0%) and 120 without (48.0%)—demonstrates that, even within an end-stage kidney disease population, diabetes clusters with older age, longer dialysis vintage, and a heavier comorbidity load, and this co-occurs with more severe anemia and more adverse urinary biomarker profiles. Diabetic patients were older by ~6 years (58.3 \pm 9.4 vs. 52.1 \pm 10.8 years) and had slightly longer dialysis duration (38.2 \pm 14.6 vs. 34.5 \pm 12.9 months). Hypertension and cardiovascular disease were substantially more prevalent

in the diabetic group (86.2% vs. 65.0% and 31.5% vs. 18.3%, respectively). These rates are directionally consistent with literature showing that hypertension commonly affects the majority of hemodialysis patients-with many series reporting >80% — and that diabetes magnifies cardiovascular risk in CKD and dialysis cohorts.1, 2 The hematologic pattern points to more pronounced anemia among diabetic patients: mean hemoglobin 9.1 ± 1.2 g/dL vs. 9.8 ± 1.3 g/dL (p<0.001), hematocrit $28.1 \pm 3.8\%$ vs. 30.2 \pm 4.1% (p=0.002), and RBC count 3.0 \pm 0.5 vs. 3.3 \pm 0.6 ×106/μL (p=0.001). Mechanistically, diabetic dialysis patients are more prone to erythropoiesis-stimulating agent (ESA) hyporesponsiveness, often driven by inflammation and iron-handling disturbances; these pathways are well described in prior cohorts and plausibly explain the lower hemoglobin we observed despite a shared dialysis environment.24, 25, 29 White cell differentials also differed: higher WBC (7.9 \pm 2.1 vs. 7.3 \pm 1.8 \times 10³/ μ L; p=0.040), higher neutrophil percentage $(63.8 \pm 8.4\% \text{ vs. } 61.1)$ \pm 7.9%; p=0.030), and lower lymphocyte percentage (27.5 \pm 6.2% vs. 29.6 ± 6.7%; p=0.020) in diabetes. Although absolute differences are modest, they align with the concept of heightened low-grade inflammation in dialysis and the prognostic utility of neutrophil-to-lymphocyte ratio (NLR) in this population.26, 27, 30 Platelet count and MCV did not differ significantly (p=0.070 and p=0.110), suggesting that, in this dataset, diabetes was associated more with erythropoietic/inflammatory perturbations than with macrocytosis or thrombocytopenia. Urinary biomarkers-abstracted from the most recent month of investigations and thus reflecting residual kidney output—also discriminated groups. Diabetic patients had higher urinary albumin (1126.0 \pm 347.0 vs. 882.0 \pm 294.0 mg/L; p<0.001), urinary protein $(86.0 \pm 24.0 \text{ vs. } 71.0 \pm 22.0 \text{ mg/L})$ mg/dL; p<0.001), and microalbuminuria indexed to creatinine $(345.2 \pm 98.6 \text{ vs. } 276.4 \pm 87.3 \text{ mg/g; } p<0.001).$ These patterns are consistent with diabetic kidney disease pathophysiology and with guideline frameworks that regard albuminuria as a central marker of kidney damage and cardiovascular risk. 18-20, 31 Conversely, urinary creatinine was lower in the diabetic group ($98.4 \pm 21.2 \text{ vs.}$ 104.7 ± 19.6 mg/dL; p=0.020). Because urinary creatinine excretion correlates with muscle mass, the finding is compatible with greater sarcopenia/cachexia in diabetic dialysis patients. 10, 11, 32 Taken together, these data suggest that diabetic status in the dialysis unit is a marker for an older, more comorbid phenotype with (i) deeper anemia, plausibly inflammation-mediated **ESA** via

hyporesponsiveness and iron dysfunction; (ii) subtle shifts toward neutrophil-predominant leukocyte profiles, in keeping with chronic micro-inflammation; and (iii) heavier proteinuric burden in residual urine, reflecting ongoing glomerular injury and endothelial dysfunction typical of diabetic nephropathy. The internal consistency between our results—e.g., hemoglobin 9.1 vs. 9.8 g/dL, neutrophils 63.8% vs. 61.1%, microalbuminuria 345.2 vs. 276.4 mg/g—and the direction of effect reported in external sources strengthens biological plausibility.^{3-6,7}

Strengths of this study include its real-world, complete census of a single center's dialysis population over a fixed six-month window; a large sample (n=250) for an institutional study; and standardized laboratory methods inherent to secondary data from one hospital. Limitations reflect the study design: reliance on secondary data from the last one month of investigations (no standardized timing vs. dialysis sessions), absence of dialysis adequacy (Kt/V), iron indices, inflammatory markers (e.g., CRP), ESA dose and response metrics, and nutritional markers (e.g., serum albumin); potential survivor and indication biases; and single-center generalizability constraints. The urinary analyses depend on residual urine availability and may under-represent anuric patients; nevertheless, the consistency betweengroup differences (albumin, total protein, microalbumin/creatinine, urinary creatinine) informative. Future work should incorporate longitudinal follow-up, adjustment for ESA/iron therapy and dialysis adequacy, and addition of inflammatory and nutritional biomarkers (e.g., NLR, CRP, serum albumin) to test mediation between diabetes, inflammation, anemia, and outcomes.5, 6, 33

CONCLUSION

In this cross-sectional study of 250 dialysis patients, diabetes identified an older, more comorbid population with significantly lower hemoglobin, hematocrit, and RBC counts, higher WBC and neutrophil predominance, and lower lymphocytes. biomarkers revealed markedly higher albuminuria, proteinuria, and microalbuminuria, with lower urinary creatinine, indicating greater residual kidney injury and muscle loss. These findings highlight the compounded burden of anemia, inflammation, and proteinuric kidney damage diabetic dialysis patients. **Targeted** in

management, including optimized cardiovascular care, anemia therapy, and nutritional rehabilitation, is essential to improve outcomes in this high-risk subgroup.

REFERENCES

- 1. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2004;27(suppl 1):s5–s10.
- Tuttle KR, Bakris GL, Bilous RW, Chiang JL, De Boer IH, Goldstein-Fuchs J, Hirsch IB, Kalantar-Zadeh K, Narva AS, Navaneethan SD. Diabetic kidney disease: a report from an ADA Consensus Conference. Am J Kidney Dis. 2014;64(4):510–533.
- 3. Guariguata L, Whiting D, Hambleton I, Beagley J, Linnenkamp U, Shaw J. Global estimates of diabetes prevalence for 2013 and projections for 2035. Diabetes Res Clin Pract. 2014;103(2):137–149.
- 4. Lisa TS, Afrose M, Kabya KM, Rahman A, Abdullah AM, Ferdaus F. linking maternal work status with child nutrition and morbidity in rural Bangladesh: A multifactorial Analysis. Asia Pac J Surg Adv. 2025;2(2):116-124.
- Rahman Mastura R, Bain R, Moonwara M, Arifuzzaman, Ferdaus F. Antibiotic Susceptibility Patterns in Recurrent Urinary Tract Infections Among Young Females. Asia Pac J Surg Adv. 2025;2(2):93-98
- Sajeeb K, Sadik HS, Hasan R, Hafiz FB, Hasan K, Arifuzzaman, Ferdaus F. Infection Control Practices and Surgical Site Infection Rates in Post-Operative Patients: A Cross-Sectional Study in a Rural Hospital Setting. Asia Pac J Surg Adv. 2025;2(2):84-92.
- 7. NCD Risk Factor Collaboration. Worldwide trends in diabetes since 1980: a pooled analysis of 751 population-based studies with 4.4 million participants. Lancet. 2016;387(10027):1513–1530.
- 8. Da Rocha Fernandes J, Ogurtsova K, Linnenkamp U, Guariguata L, Seuring T, Zhang P, Cavan D, Makaroff LE. IDF Diabetes Atlas estimates of 2014 global health expenditures on diabetes. Diabetes Res Clin Pract. 2016;117:48–54.

- 9. American Diabetes Association. Diagnosis and classification of diabetes mellitus. Diabetes Care. 2014;37(Suppl 1):S81–S90.
- 10. Gavin III JR, Alberti K, Davidson MB, DeFronzo RA. Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care. 1997;20(7):1183–1197.
- 11. Forbes JM, Cooper ME. Mechanisms of diabetic complications. Physiol Rev. 2013;93(1):137–188.
- 12. American Diabetes Association. Report of the expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care. 2003;26(suppl 1):s5–s20.
- 13. Deshpande AD, Harris-Hayes M, Schootman M. Epidemiology of diabetes and diabetes-related complications. Phys Ther. 2008;88(11):1254–1264.
- 14. Jesi NS, Sharmy SC, Hridi AS, Reza MS, Ferdaus F. Chronic Lung Disease in Focus: Epidemiological Perspectives on Lifestyle, Environmental, and Clinical Risk Factors. Asia Pac J Surg Adv. 2025;2(3):130-138
- 15. Rayhan MR, Lisa TS, Hasan MK, Morshed MR, Mahmud MR, Sultana T.Mental Shadows of TB: Cross-Sectional Analysis of Stigma, Psychological Well-being, and Support Needs in Pulmonary Tuberculosis Patients. Bangl J Food Nutr. 2025;2(3):43-49
- 16. Abdullah AM, Rahman A, Reza IMK, Ahmed S, Tabassum F, Ferdaus F.Emerging Comorbidity of Diabetes and Hypertension Among Young Adults in Bangladesh: A Cross-Sectional Study. BanglJ Food Nutr. 2025;2(3):58-66
- 17. Schrijvers BF, De Vriese AS, Flyvbjerg A. From hyperglycemia to diabetic kidney disease: the role of metabolic, hemodynamic, intracellular factors and growth factors/cytokines. Endocr Rev. 2004;25(6):971–1010.
- 18. Reidy K, Kang HM, Hostetter T, Susztak K. Molecular mechanisms of diabetic kidney disease. J Clin Invest. 2014;124(6):2333–2340.
- 19. Adeosun OG, Anetor JI, Ogunlewe JO, Ikem RT, Kolawole BA, Arogundade FA, Oyedeji SO.

- Evaluation of alterations in the urine biochemical profiles of type 2 diabetes mellitus patients in Southwest, Nigeria. Afr J Biotechnol. 2014;13(1):175–180.
- Jefferson J, Shankland S, Pichler R. Proteinuria in diabetic kidney disease: a mechanistic viewpoint. Kidney Int. 2008;74(1):22–36.
- 21. Asha ABZ, Lisa TS, Malaker S, Harun JB, Babunty RBZ, Parveen M.A Cross-Sectional Investigation of Serum Cholesterol, Blood Glucose, and Body Mass Index Differences Between Elderly Primigravida and Multigravida. Bangl J Food Nutr. 2025;2(3):50-57
- 22. Rahman A, Abdullah AM, Ahmed S, Reza IMK, Kabya KM, Alam GN.Respiratory and Musculoskeletal Disorders Among Jute Mill Workers: Occupational Health Findings from a Cross-Sectional Study in Bangladesh. Bangl J Food Nutr. 2025;2(3):67-74
- AhmedS, RezaIMK,RahmanA, AbdullahAM, Tabassum F, SultanaT.Rationality and Resistance: A Study on Antibiotic Prescription Practices Among General Practitioners in RuralBangladesh. Bangl J Food Nutr. 2025;2(3):75-81
- 24. Gluhovschi C, Gluhovschi G, Petrica L, Timar R, Velciov S, Ionita I, Kaycsa A, Timar B. Urinary biomarkers in the assessment of early diabetic nephropathy. J Diabetes Res. 2016;2016:4626125.
- 25. Mogensen C. Microalbuminuria and hypertension with focus on type 1 and type 2 diabetes. J Intern Med. 2003;254:45–66.
- Basi S, Fesler P, Mimran A, Lewis JB. Microalbuminuria in type 2 diabetes and hypertension. Diabetes Care. 2008;31(Suppl 2):S194– S201.

- 27. Weir MR. Microalbuminuria in type 2 diabetics: an important, overlooked cardiovascular risk factor. J Clin Hypertens. 2004;6(3):134–143.
- 28. American Diabetes Association. Standards of Medical Care In Diabetes-2017. Diabetes Care. 2017;40(Suppl 1):S1–S87.
- 29. De Boer IH, Rue TC, Cleary PA, Lachin JM, Molitch ME, Steffes MW, Sun W, Zinman B, Brunzell JD. Long-term renal outcomes of patients with type 1 diabetes mellitus and microalbuminuria: an analysis of the Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications cohort. Arch Intern Med. 2011;171(5):412–420.
- 30. Satirapoj B, Adler SG. Comprehensive approach to diabetic nephropathy. Kidney Res Clin Pract. 2014;33(3):121–131.
- 31. Moonwara M, Bain R, Mastura RR, Arifuzzaman, Ferdaus F.Impact of Chronic Workplace Stress on Menstrual Irregularities and Early Onset Menopause in Young Female Workers. Bangl J Food Nutr. 2025;2(2):22-27
- 32. Reza IMK, Ahmed S, Abdullah AM, Rahman A, Afrose M, Ferdaus F. Triple Health Burden in the Garment Industry: A Study on Musculoskeletal Disorders, Visual Strain, and Psychological Stress Among Female RMG Workers in Bangladesh. Asia Pac J Cancer Res. 2025;2(2):65-72.
- 33. Bain R, Moonwara M, Mastura RR, Alam GN, Ferdaus F. Impact of Saline Water Intrusion on Maternal and Neonatal Health in Coastal Communities of Bangladesh. Asia Pac J Cancer Res. 2025;2(1):10-16.