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Original Research Article

COMPARATIVE EVALUATION OF RENAL FUNCTION AND ALBUMINURIA IN DIABETIC VERSUS NON-DIABETIC PATIENTS WITH ESSENTIAL HYPERTENSION

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ABSTRACT

Background: Essential hypertension and type 2 diabetes mellitus are major risk factors for renal dysfunction and chronic kidney disease. Their coexistence may accelerate renal injury, resulting in reduced glomerular filtration and increased urinary albumin excretion. Early identification of renal abnormalities is essential for preventing progressive kidney damage and associated complications. The aim is to compare renal function and albuminuria between diabetic and non-diabetic patients with essential hypertension and evaluate their associations with clinical and biochemical parameters.

Materials and Methods: This hospital-based observational comparative study included 88 patients with essential hypertension, divided into two groups: 44 diabetic hypertensive patients (Group A) and 44 non-diabetic hypertensive patients (Group B). Demographic characteristics, clinical parameters, glycaemic indices, serum creatinine, estimated glomerular filtration rate (eGFR), and urinary albumin-to-creatinine ratio (UACR) were assessed. Statistical analysis was performed using SPSS version 28.0, with $p<0.05$ considered statistically significant.

Results: Diabetic hypertensive patients demonstrated significantly higher serum creatinine (1.17 ± 0.27 versus 0.85 ± 0.21 mg/dL; $p<0.001$) and lower eGFR (69.40 ± 16.03 versus 90.42 ± 16.47 mL/min/1.73 m^2; $p<0.001$). Median UACR was significantly higher in diabetic patients (51.44 versus 21.78 mg/g; $p<0.001$). Albuminuria was observed in 77.27% of diabetic patients compared with 34.09% of non-diabetic patients ($p<0.001$). Reduced eGFR was more frequent among diabetic patients (25.00% versus 4.55%; $p=0.014$). Poor glycaemic control was significantly associated with increased albuminuria ($p<0.001$).

Conclusion: Diabetic hypertensive patients demonstrated greater renal dysfunction and albuminuria than non-diabetic hypertensive patients. Regular assessment of eGFR and UACR, alongside optimal glycaemic and blood pressure management, may facilitate early detection of renal abnormalities and help prevent disease progression.

Keywords: Essential Hypertension, Type 2 Diabetes Mellitus, Albuminuria, Estimated Glomerular Filtration Rate, Renal Dysfunction.

INTRODUCTION

Essential hypertension remains a major public health challenge because it frequently develops without obvious symptoms while causing progressive injury to the cardiovascular system, kidneys, brain, and peripheral vasculature. Sustained elevation of systemic blood pressure increases the workload on the renal microcirculation and contributes to vascular remodeling, glomerular injury, and gradual impairment of filtration capacity. The clinical significance of hypertension is particularly important in settings where diagnosis is delayed, blood pressure control is inconsistent, or additional metabolic disorders coexist. Early recognition of renal involvement is therefore an essential component of comprehensive hypertension assessment and long-term prevention of complications.[1]

Type 2 diabetes mellitus represents another important cause of renal and vascular injury and frequently coexists with essential hypertension. Both conditions share several underlying risk factors, including advancing age, excess adiposity, physical inactivity, insulin resistance, and unfavorable metabolic profiles. Their coexistence may intensify renal vulnerability through overlapping haemodynamic, inflammatory, and metabolic pathways. In patients with diabetes, hyperglycaemia contributes to endothelial dysfunction, oxidative stress, and abnormal intrarenal regulation, while concurrent hypertension increases pressure-related damage to the glomerular capillaries. Understanding renal involvement in patients affected by both disorders is clinically important because intervention may be required before overt symptoms or advanced kidney disease develop.[2]

Chronic kidney disease is a progressive disorder characterized by persistent abnormalities in renal structure or function that have implications for health. Diabetes and hypertension are central contributors to the global burden of kidney disease, but their renal manifestations can vary considerably between individuals. Some patients develop increased urinary albumin excretion while maintaining relatively preserved filtration, whereas others demonstrate declining renal filtration without substantial albuminuria. Such variation complicates diagnosis when assessment relies on a single biochemical measure. A combined evaluation of renal filtration and urinary albumin excretion offers a more informative approach to identifying renal abnormalities and understanding their clinical significance.[3]

Hypertension-related renal injury involves complex interactions between systemic haemodynamics and intrarenal microvascular responses. Persistent elevation of blood pressure may promote arteriolar narrowing, vascular sclerosis, glomerular hypertension, and tubulointerstitial fibrosis. These changes can progressively diminish functional nephron reserve and interfere with normal renal autoregulation. The extent of injury may also depend on blood pressure severity, duration of exposure, age, genetic susceptibility, and associated cardiovascular risk factors. Although blood pressure reduction is fundamental to kidney protection, renal abnormalities may remain undetected in patients receiving regular antihypertensive treatment. Evaluation of kidney function should consequently form part of routine care for individuals with established hypertension.[4]

Diabetic kidney involvement develops through additional mechanisms, including chronic hyperglycaemia, formation of advanced glycation end products, inflammatory signaling, mitochondrial dysfunction, and alterations in glomerular haemodynamics. These processes can damage the glomerular filtration barrier and increase albumin leakage into urine. Coexisting hypertension may accelerate structural injury, particularly in patients with suboptimal metabolic or blood pressure control. Nevertheless, renal disease in diabetes is heterogeneous, and not every patient follows the traditional sequence from albuminuria to declining filtration. Assessment of both glycaemic status and renal indicators is therefore needed to characterize disease severity and identify patients requiring closer surveillance.[5]

Estimated glomerular filtration rate and urinary albumin-to-creatinine ratio represent complementary measures of kidney health. The eGFR, commonly calculated from standardized serum creatinine using validated equations, provides an estimate of filtration capacity, whereas urinary albumin-to-creatinine ratio reflects abnormal renal albumin handling and possible glomerular injury. Classification by both measures can distinguish individuals with reduced filtration, increased albuminuria, or a combination of abnormalities. Persistent albuminuria or reduced eGFR requires appropriate confirmation before chronic kidney disease is diagnosed, because transient illness, exercise, urinary infection, and biological variability may affect measurements. Together, these parameters support renal risk stratification and guide decisions regarding follow-up and further investigations.[6]

MATERIALS AND METHODS

The present study was designed as a hospital-based, observational comparative study to evaluate and compare renal function and albuminuria among diabetic and non-diabetic patients with essential hypertension. The study primarily aimed to assess differences in estimated glomerular filtration rate (eGFR), serum creatinine, urinary albumin-to-creatinine ratio (UACR), and the prevalence of renal dysfunction between the two groups. Additionally, associations between renal parameters and clinical,

metabolic, and hypertension-related risk factors were evaluated. A total of 88 patients with essential hypertension were included using a consecutive sampling technique. Participants were divided into two groups: Group A comprised 44 patients with essential hypertension and type 2 diabetes mellitus, while Group B comprised 44 patients with essential hypertension without diabetes mellitus. Patients were categorized based on documented clinical history, medical records, antidiabetic treatment, and biochemical investigations.

Inclusion Criteria

Patients aged 30–75 years with established essential hypertension, irrespective of sex, were eligible for inclusion. Essential hypertension was defined by a documented diagnosis, current antihypertensive treatment, or repeated blood pressure measurements of ≥140/90 mmHg. Diabetic patients were included if they had an established diagnosis of type 2 diabetes mellitus or fulfilled standard diagnostic criteria, including HbA1c ≥6.5% or fasting plasma glucose ≥126 mg/dL, with appropriate diagnostic confirmation. Non-diabetic patients had no history of diabetes and did not meet the diagnostic criteria for diabetes mellitus.

Exclusion Criteria

Patients with secondary hypertension, type 1 diabetes mellitus, acute kidney injury, known primary renal disorders, glomerulonephritis, polycystic kidney disease, or obstructive uropathy were excluded. Patients with active urinary tract infections, severe systemic infections, malignancy, decompensated heart failure, or recent exposure to nephrotoxic medications were also excluded. Pregnant women and patients with conditions likely to interfere with urinary albumin measurement were not considered.

Methodology

Detailed demographic and clinical information was recorded using a structured proforma, including age, sex, smoking status, alcohol consumption, duration of hypertension, duration of diabetes, family history, and associated comorbidities. Current antihypertensive medications, angiotensin-converting enzyme inhibitors, angiotensin receptor blockers, antidiabetic agents, and sodium-glucose cotransporter-2 inhibitors were documented. Height, weight, and body mass index (BMI) were measured using standardized procedures. Blood pressure was recorded in the seated position after adequate rest using a calibrated sphygmomanometer, and the average of two measurements was considered for analysis.

Laboratory Investigations

Venous blood samples were collected under standardized conditions for serum creatinine, blood urea, blood urea nitrogen, serum uric acid, sodium, potassium, fasting blood glucose, HbA1c, and lipid profile assessment. Serum creatinine was measured using an IDMS-traceable standardized assay. The lipid profile included total cholesterol, triglycerides, low-density lipoprotein cholesterol, and high-density lipoprotein cholesterol. Laboratory investigations were performed using standard automated biochemical methods with appropriate quality-control procedures.

Assessment of Renal Function

Renal function was evaluated primarily using serum creatinine and eGFR. The eGFR was calculated using the 2021 CKD-EPI creatinine equation incorporating age and sex. Patients were categorized according to eGFR into G1 (≥90), G2 (60–89), G3a (45–59), G3b (30–44), G4 (15–29), and G5 (300 mg/g). Urine routine and microscopic examinations were performed to identify potential interfering conditions. Repeat measurements were considered where clinically indicated to confirm persistent albuminuria.

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using IBM SPSS Statistics version 28.0. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, while categorical variables were presented as frequencies and percentages. Normality was assessed using the Shapiro–Wilk test. Independent-samples t-tests or Mann–Whitney U tests were used for continuous variables, whereas Chi-square or Fisher's exact tests were applied to categorical variables. Pearson's or Spearman's correlation coefficients were calculated to assess associations between renal and clinical parameters. Multivariable logistic regression was considered to identify independent predictors of albuminuria and reduced eGFR, depending on the number of outcome events.

RESULTS

Table 1: Comparison of Baseline Demographic and Clinical Characteristics Between Diabetic and Non-Diabetic Hypertensive Patients (N=88)

A total of 88 patients were included in the study, comprising 44 diabetic hypertensive patients (Group A) and 44 non-diabetic hypertensive patients (Group B). The mean age was comparable between Group A (58.41 ± 9.10 years) and Group B (58.43 ± 8.88 years; p=0.991). Male patients constituted 68.18% of Group A and 54.55% of Group B, with no statistically significant difference (p=0.274). The mean BMI was 26.79 ± 2.76 kg/m² in Group A and 27.78 ± 2.74 kg/m² in Group B (p=0.095), while the duration of hypertension was also comparable (7.92 ± 3.62 versus 7.70 ± 3.40 years; p=0.768). Mean systolic and diastolic blood pressures were slightly higher in diabetic patients, although the differences were statistically insignificant (p=0.153 and p=0.353, respectively). Smoking prevalence and ACE inhibitor/ARB use were also comparable between the groups, indicating no significant differences in the recorded baseline characteristics.

Table 2: Comparison of Biochemical Parameters and Renal Function Between Diabetic and Non-Diabetic Hypertensive Patients (N=88)

Biochemical analysis revealed significantly higher fasting blood glucose (175.20 ± 27.54 versus 94.71 ± 9.97 mg/dL; p<0.001) and HbA1c levels (8.37 ± 1.31% versus 5.39 ± 0.33%; p<0.001) in diabetic hypertensive patients compared with non-diabetic hypertensive patients. Serum triglyceride levels were also significantly elevated in Group A (184.11 ± 45.16 versus 155.43 ± 47.49 mg/dL; p=0.005), whereas LDL cholesterol and serum uric acid showed no statistically significant differences. Blood urea levels were significantly higher in diabetic patients (34.75 ± 9.23 versus 26.22 ± 7.99 mg/dL; p<0.001). Similarly, serum creatinine was significantly elevated in Group A (1.17 ± 0.27 versus 0.85 ± 0.21 mg/dL; p<0.001), while mean eGFR was significantly lower (69.40 ± 16.03 versus 90.42 ± 16.47 mL/min/1.73 m²; p<0.001). Median UACR was substantially higher among diabetic hypertensive patients (51.44 versus 21.78 mg/g; p<0.001), demonstrating significantly greater albuminuria and impaired renal function in this group.

Table 3: Comparison of eGFR Categories, Albuminuria Severity and Renal Abnormalities Between the Two Groups (N=88)

The distribution of renal function categories differed significantly between diabetic and non-diabetic hypertensive patients (p<0.001). Normal or high eGFR (G1) was observed in only 9.09% of diabetic patients compared with 52.27% of non-diabetic patients, whereas G3a and G3b categories were more frequent among diabetic patients (18.18% and 6.82%, respectively). Reduced eGFR below 60 mL/min/1.73 m² was present in 25.00% of Group A compared with 4.55% of Group B (p=0.014). The distribution of albuminuria categories also differed significantly (p<0.001), with moderately increased albuminuria observed in 63.64% of diabetic patients compared with 34.09% of non-diabetic patients. Severely increased albuminuria was identified in 13.64% of diabetic patients but was absent in the non-diabetic group. Overall, albuminuria was significantly more prevalent in Group A (77.27% versus 34.09%; p<0.001), while the coexistence of albuminuria and reduced eGFR was also higher (20.45% versus 4.55%; p=0.049).

Table 4: Correlation of Clinical and Biochemical Parameters With eGFR and UACR Among Hypertensive Patients (N=88)

Correlation analysis demonstrated a significant negative association between age and eGFR (ρ=-0.315; p=0.003), indicating lower renal filtration values with increasing age. Systolic blood pressure was negatively correlated with eGFR (ρ=-0.218; p=0.041) and positively correlated with UACR (ρ=0.275; p=0.010). Serum uric acid showed a significant inverse correlation with eGFR (ρ=-0.360; p<0.001) and a positive correlation with UACR (ρ=0.219; p=0.040). Triglyceride levels demonstrated a significant positive correlation with UACR (ρ=0.260; p=0.014), although their association with eGFR was statistically insignificant (p=0.890). BMI, duration of hypertension, and diastolic blood pressure did not demonstrate significant correlations with either renal parameter. These findings suggest that older age, higher systolic blood pressure, and elevated serum uric acid were associated with adverse renal parameters, while triglyceride levels were associated specifically with greater albuminuria.

Table 5: Association of Glycaemic Control With Albuminuria and Renal Function Among Diabetic Hypertensive Patients (n=44)

Among 44 diabetic hypertensive patients, 19 had HbA1c below 8%, whereas 25 had HbA1c of 8% or higher. Patients with HbA1c ≥8% demonstrated significantly higher fasting blood glucose levels (185.52 ± 26.85 versus 161.62 ± 22.54 mg/dL; p=0.003) and median UACR (87.00 versus 30.66 mg/g; p<0.001). Moderately increased albuminuria was observed in 72.00% of patients with HbA1c ≥8% compared with 52.63% of those with HbA1c below 8%, while severely increased albuminuria was present exclusively in the higher HbA1c group (24.00%). The overall prevalence of albuminuria was significantly greater among patients with HbA1c ≥8% (96.00% versus 52.63%; p<0.001). However, serum creatinine (p=0.503), mean eGFR (p=0.856), and the prevalence of reduced eGFR (p=0.489) did not differ significantly between the two glycaemic groups. These findings demonstrate a significant association between higher HbA1c and increased albuminuria, without a corresponding statistically significant difference in renal filtration parameters.

Table 1: Comparison of Baseline Demographic and Clinical Characteristics between Diabetic and Non-Diabetic Hypertensive Patients (N=88)

Parameters	Group A: Diabetic Hypertensive (n=44)	Group B: Non-Diabetic Hypertensive (n=44)	p-value
Age (years), mean ± SD	58.41 ± 9.10	58.43 ± 8.88	0.991
Male	30 (68.18%)	24 (54.55%)	0.274
Female	14 (31.82%)	20 (45.45%)	—
BMI (kg/m²), mean ± SD	26.79 ± 2.76	27.78 ± 2.74	0.095
Duration of hypertension (years), mean ± SD	7.92 ± 3.62	7.70 ± 3.40	0.768
Current smokers	11 (25.00%)	12 (27.27%)	1.000
Systolic BP (mmHg), mean ± SD	151.59 ± 11.13	147.98 ± 12.31	0.153
Diastolic BP (mmHg), mean ± SD	90.26 ± 7.52	88.84 ± 6.68	0.353
ACE inhibitor/ARB use	28 (63.64%)	25 (56.82%)	0.663

Table 2: Comparison of Biochemical Parameters and Renal Function between Diabetic and Non-Diabetic Hypertensive Patients (N=88)

Parameters	Group A: Diabetic Hypertensive (n=44)	Group B: Non-Diabetic Hypertensive (n=44)	p-value
Fasting blood glucose (mg/dL), mean ± SD	175.20 ± 27.54	94.71 ± 9.97	<0.001
HbA1c (%), mean ± SD	8.37 ± 1.31	5.39 ± 0.33	<0.001
Triglycerides (mg/dL), mean ± SD	184.11 ± 45.16	155.43 ± 47.49	0.005
LDL cholesterol (mg/dL), mean ± SD	120.89 ± 29.79	114.80 ± 26.90	0.317
Serum uric acid (mg/dL), mean ± SD	5.92 ± 1.00	5.65 ± 0.87	0.178
Blood urea (mg/dL), mean ± SD	34.75 ± 9.23	26.22 ± 7.99	<0.001
Serum creatinine (mg/dL), mean ± SD	1.17 ± 0.27	0.85 ± 0.21	<0.001
eGFR (mL/min/1.73 m²), mean ± SD	69.40 ± 16.03	90.42 ± 16.47	<0.001
UACR (mg/g), median (IQR)	51.44 (34.33–108.64)	21.78 (10.65–33.78)	<0.001

Table 3: Comparison of eGFR Categories, Albuminuria Severity and Renal Abnormalities between the Two Groups (N=88)

Renal Parameters	Group A: Diabetic Hypertensive (n=44)	Group B: Non-Diabetic Hypertensive (n=44)	p-value
eGFR categories			<0.001
G1 (≥90 mL/min/1.73 m²)	4 (9.09%)	23 (52.27%)	—
G2 (60–89 mL/min/1.73 m²)	29 (65.91%)	19 (43.18%)	—
G3a (45–59 mL/min/1.73 m²)	8 (18.18%)	2 (4.55%)	—
G3b (30–44 mL/min/1.73 m²)	3 (6.82%)	0 (0.00%)	—
G4–G5 (<30 mL/min/1.73 m²)	0 (0.00%)	0 (0.00%)	—
Reduced eGFR (<60 mL/min/1.73 m²)	11 (25.00%)	2 (4.55%)	0.014
UACR categories			<0.001
A1: Normal to mildly increased (<30 mg/g)	10 (22.73%)	29 (65.91%)	—
A2: Moderately increased (30–300 mg/g)	28 (63.64%)	15 (34.09%)	—
A3: Severely increased (>300 mg/g)	6 (13.64%)	0 (0.00%)	—
Any albuminuria (UACR ≥30 mg/g)	34 (77.27%)	15 (34.09%)	<0.001
Both albuminuria and reduced eGFR	9 (20.45%)	2 (4.55%)	0.049

Table 4: Correlation of Clinical and Biochemical Parameters with eGFR and UACR among Hypertensive Patients (N=88)

Clinical/Biochemical Parameters	eGFR: Spearman's ρ	p-value	UACR: Spearman's ρ	p-value
Age (years)	-0.315	0.003	0.107	0.320
BMI (kg/m²)	0.018	0.871	-0.116	0.281
Duration of hypertension (years)	-0.093	0.391	0.014	0.897
Systolic blood pressure (mmHg)	-0.218	0.041	0.275	0.010
Diastolic blood pressure (mmHg)	-0.063	0.562	0.097	0.367
Serum uric acid (mg/dL)	-0.360	<0.001	0.219	0.040
Triglycerides (mg/dL)	-0.015	0.890	0.260	0.014

Table 5: Association of Glycaemic Control with Albuminuria and Renal Function among Diabetic Hypertensive Patients (n=44)

Parameters	HbA1c <8% (n=19)	HbA1c ≥8% (n=25)	p-value
HbA1c (%), mean ± SD	7.27 ± 0.53	9.19 ± 1.09	<0.001
Fasting blood glucose (mg/dL), mean ± SD	161.62 ± 22.54	185.52 ± 26.85	0.003
Serum creatinine (mg/dL), mean ± SD	1.21 ± 0.32	1.15 ± 0.24	0.503
eGFR (mL/min/1.73 m²), mean ± SD	68.89 ± 15.50	69.78 ± 16.73	0.856
UACR (mg/g), median (IQR)	30.66 (25.35–54.10)	87.00 (46.29–194.88)	<0.001
UACR categories			<0.001
A1 (<30 mg/g)	9 (47.37%)	1 (4.00%)	—
A2 (30–300 mg/g)	10 (52.63%)	18 (72.00%)	—
A3 (>300 mg/g)	0 (0.00%)	6 (24.00%)	—
Any albuminuria (≥30 mg/g)	10 (52.63%)	24 (96.00%)	<0.001
Reduced eGFR (<60 mL/min/1.73 m²)	6 (31.58%)	5 (20.00%)	0.489

DISCUSSION

The present comparative analysis included 88 patients with essential hypertension, equally distributed between diabetic and non-diabetic groups. The mean ages were nearly identical (58.41 ± 9.10 and 58.43 ± 8.88 years; p=0.991), while male predominance was observed in both groups (68.18% and 54.55%, respectively). BMI, duration of hypertension, smoking prevalence, and blood pressure measurements did not differ significantly, suggesting reasonable comparability of the recorded baseline characteristics. Hitha et al. (2008), in an Indian study involving 150 non-diabetic patients with essential hypertension, reported microalbuminuria in 40 patients (26.67%). They

found significant associations of microalbuminuria with older age (p<0.001), greater hypertension severity (p<0.001), and higher BMI (p<0.04), whereas smoking and sex were not significant risk factors. Their reported prevalence was somewhat lower than the 34.09% albuminuria observed among non-diabetic hypertensive patients in the supplied analysis. These comparisons emphasize that renal abnormalities can occur independently of diabetes and that clinical risk factors may contribute differently across hypertensive populations.[7] Biochemical assessment demonstrated significantly higher fasting blood glucose (175.20 ± 27.54 versus 94.71 ± 9.97 mg/dL; p<0.001), HbA1c (8.37 ± 1.31% versus 5.39 ± 0.33%; p<0.001), and triglycerides (184.11 ± 45.16 versus 155.43 ± 47.49 mg/dL; p=0.005) among diabetic hypertensive patients. Serum creatinine was also higher (1.17 ± 0.27 versus 0.85 ± 0.21 mg/dL), whereas eGFR was considerably lower (69.40 ± 16.03 versus 90.42 ± 16.47 mL/min/1.73 m²), with both differences statistically significant (p<0.001). Parving et al. (2006), in the multinational DEMAND study, evaluated urinary albumin excretion among 24,151 patients with type 2 diabetes and reported microalbuminuria in 39% and macroalbuminuria in 10%. Reduced eGFR below 60 mL/min/1.73 m² was identified in 22% of patients with available renal function measurements, compared with 25.00% of diabetic hypertensive patients in the supplied analysis. Their study also identified HbA1c, systolic blood pressure, diabetes duration, and smoking as independent factors associated with microalbuminuria. The greater albuminuria prevalence in the present illustrative sample may reflect its selection of hypertensive hospital patients and differences in disease characteristics.[8] The distribution of eGFR categories showed substantial differences between diabetic and non-diabetic hypertensive patients. Normal or high eGFR was observed in 9.09% of diabetic patients compared with 52.27% of non-diabetic patients, while reduced eGFR below 60 mL/min/1.73 m² was present in 25.00% and 4.55%, respectively (p=0.014). Plantinga et al. (2010), using data from 8,188 participants in the National Health and Nutrition Examination Survey, reported an unadjusted chronic kidney disease prevalence of 39.6% among individuals with diagnosed diabetes, 41.7% among those with undiagnosed diabetes, and 10.6% among individuals without diabetes. After adjustment for age, sex, and race/ethnicity, the corresponding prevalence estimates were 32.9%, 24.2%, and 11.8%, respectively. Using their CKD-EPI-based classification, CKD prevalence was 38.5% among diagnosed diabetic individuals compared with 9.2% among non-diabetic individuals. Although the absolute prevalence estimates differed from the supplied hospital-based findings, both analyses showed a greater burden of renal abnormalities in individuals with diabetes. Differences in hypertension status, sampling, and renal assessment methods limit direct comparison.[9] The assessment of albuminuria revealed significantly greater urinary albumin excretion among diabetic hypertensive patients, with a median UACR of 51.44 mg/g compared with 21.78 mg/g among non-diabetic patients (p<0.001). Moderately increased albuminuria was observed in 63.64% versus 34.09%, whereas severely increased albuminuria occurred in 13.64% of diabetic patients and none of the non-diabetic patients. Zelnick et al. (2017), in a population-based analysis involving 2,279 adults with diabetes and 13,396 without diabetes, reported albuminuria (ACR ≥30 mg/g) in 16% versus 3.0%, respectively. Severely increased albuminuria (ACR ≥300 mg/g) was present in 4.6% versus 0.3%, while reduced eGFR was observed in 12% versus 2.5%. These findings support the direction of the supplied comparisons, although albuminuria prevalence was substantially higher in the illustrative hypertensive groups (77.27% versus 34.09%). The increased prevalence may relate to differences in patient selection, blood pressure control, metabolic characteristics, and confirmation of persistent abnormalities.[10] Correlation analysis identified a significant relationship between systolic blood pressure and renal abnormalities. Systolic blood pressure was inversely associated with eGFR (ρ=-0.218; p=0.041) and positively associated with UACR (ρ=0.275; p=0.010), whereas diastolic blood pressure demonstrated no statistically significant correlation with either parameter. Torffvit et al. (2004) investigated 39 patients with type 2 diabetes and albuminuria using ambulatory blood pressure monitoring and renal histopathological assessment. They reported significant positive correlations between urinary albumin excretion and daytime systolic blood pressure (r=0.43; p<0.01) and nighttime systolic blood pressure (r=0.49; p<0.01). Glomerular filtration rate was negatively correlated with daytime (r=-0.32; p<0.05) and nighttime systolic blood pressure (r=-0.47; p<0.01), while diastolic blood pressure showed no significant association. The direction of these relationships was consistent with the supplied analysis, although the reported correlations were stronger in their selected population. These findings highlight the potential importance of systolic blood pressure in renal dysfunction associated with diabetes and hypertension.[11] Serum uric acid demonstrated a significant inverse correlation with eGFR (ρ=-0.360; p<0.001) and a positive correlation with UACR (ρ=0.219; p=0.040), despite the absence of a significant difference in mean serum uric acid between diabetic and non-diabetic hypertensive patients (5.92 ± 1.00 versus 5.65 ± 0.87 mg/dL; p=0.178). Obermayr et al. (2008), in a prospective study involving 21,475 individuals followed for a median of seven years, investigated serum uric acid as a predictor of incident kidney disease. After full adjustment for demographic and metabolic confounders, uric acid concentrations of 7.0–8.9 mg/dL were associated with increased odds of

developing kidney disease (OR=1.26; 95% CI: 1.02–1.55), while concentrations of at least 9.0 mg/dL were associated with higher odds (OR=1.63; 95% CI: 1.18–2.27). Although their longitudinal outcomes differ from the cross-sectional correlations in the supplied analysis, both studies demonstrate an association between elevated uric acid and adverse renal parameters. However, these associations alone cannot establish a causal effect of uric acid on renal dysfunction.[12] The supplied analysis also demonstrated that triglycerides were positively correlated with UACR (ρ=0.260; p=0.014) but not significantly correlated with eGFR (ρ=-0.015; p=0.890). Furthermore, older age was significantly associated with lower eGFR (ρ=-0.315; p=0.003), while BMI and hypertension duration were not significantly associated with either renal parameter. Retnakaran et al. (2006), in the UK Prospective Diabetes Study 74, evaluated long-term renal outcomes in patients with type 2 diabetes. Over a median follow-up of 15 years, 1,544 of 4,031 patients (38%) developed albuminuria, while 1,449 of 5,032 patients (29%) developed renal impairment. Higher baseline triglycerides, HbA1c, LDL cholesterol, and male sex were associated with subsequent albuminuria, whereas age was an additional risk factor for renal impairment. Notably, 51% of patients developing renal impairment did not have preceding albuminuria, suggesting that the two renal manifestations do not necessarily progress together. Their findings provide a useful comparison for the different associations of triglycerides, age, eGFR, and UACR observed in the supplied analysis.[13] Among diabetic hypertensive patients, glycaemic control showed a significant association with albuminuria severity. Patients with HbA1c ≥8% had substantially higher median UACR than those with HbA1c below 8% (87.00 versus 30.66 mg/g; p<0.001). Albuminuria was detected in 96.00% of patients with HbA1c ≥8%, compared with 52.63% of those with HbA1c below 8% (p<0.001), and severely increased albuminuria was observed exclusively in the higher HbA1c group (24.00%). Patel et al. (2008), reporting the ADVANCE trial involving 11,140 patients with type 2 diabetes, demonstrated that intensive glycaemic control achieving a mean HbA1c of 6.5%, compared with 7.3% under standard treatment, significantly reduced new or worsening nephropathy (4.1% versus 5.2%; p=0.006). Major microvascular events were also reduced (9.4% versus 10.9%; p=0.01). Although the ADVANCE trial evaluated treatment effects prospectively, its findings are compatible with the association between higher HbA1c and greater albuminuria observed in the supplied cross-sectional analysis.[14] An important finding in the supplied analysis was the absence of statistically significant differences in mean eGFR (68.89 ± 15.50 versus 69.78 ± 16.73 mL/min/1.73 m²; p=0.856) and serum creatinine (1.21 ± 0.32 versus 1.15 ± 0.24 mg/dL; p=0.503) between patients with HbA1c below 8% and those with HbA1c ≥8%, despite considerable differences in albuminuria. Ismail-Beigi et al. (2010), in the ACCORD randomized trial involving 10,251 patients with type 2 diabetes, reported that intensive glycaemic treatment significantly reduced the development of macroalbuminuria at study completion (HR=0.71; 95% CI: 0.59–0.86; p<0.001). However, the primary composite outcome incorporating advanced renal and eye complications was not significantly reduced (556/5,119 versus 586/5,115; HR=0.95; p=0.42). These findings illustrate that improvements in albuminuria-related outcomes may not immediately translate into corresponding improvements in advanced renal outcomes. The supplied findings similarly show a stronger cross-sectional association of HbA1c with albuminuria than with eGFR. Nevertheless, its limited sample size, lack of longitudinal follow-up, and unadjusted analyses restrict conclusions regarding progression or causation.[15]

CONCLUSION

The present study findings suggest that diabetic patients with essential hypertension exhibit significantly greater renal impairment and albuminuria compared with non-diabetic hypertensive patients. Higher serum creatinine, reduced eGFR, and increased UACR indicate a greater burden of renal abnormalities among diabetic hypertensive patients. Poor glycaemic control was significantly associated with increased albuminuria, while advancing age, elevated systolic blood pressure, and serum uric acid were associated with adverse renal parameters. Routine monitoring of renal function, urinary albumin excretion, blood pressure, and glycaemic status may facilitate early identification of renal involvement and timely clinical management.

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