Consequences of Damage to Renal Parenchyma and Microcirculatory Bed in Arterial Hypertension Associated with Diabetes Mellitus

Xatamov Vohidjon Baxtiyorovich

Central Asian Medical University

Abstract

Objective. To evaluate the clinical and laboratory consequences of damage to the renal parenchyma and microcirculatory bed in patients with arterial hypertension associated with diabetes mellitus, and to determine the relationship between albuminuria, eGFR, renal Doppler parameters, and microcirculation markers.A conditional clinical observational study included 90 patients. The patients were divided into three groups: Group 1 — patients with type 2 diabetes mellitus and arterial hypertension, n=45; Group 2 — patients with type 2 diabetes mellitus without arterial hypertension, n=25; Group 3 — patients with arterial hypertension without diabetes mellitus, n=20.In all patients, blood pressure, HbA1c, creatinine, eGFR, urinary albumin-to-creatinine ratio, renal ultrasound, renal parenchymal thickness, renal Doppler resistive index, and peripheral microcirculation parameters were assessed. Statistical analysis included ANOVA, the Kruskal–Wallis test, the χ^2 test, Spearman correlation analysis, multivariate regression analysis, and ROC analysis.

Keywords: *diabetes mellitus, arterial hypertension, diabetic nephropathy, renal parenchyma, microcirculation, albuminuria, eGFR, renal resistive index.*

Introduction

Diabetes mellitus and arterial hypertension are among the most important risk factors for chronic kidney disease. Diabetic kidney disease is often manifested by albuminuria, reduced glomerular filtration rate, and structural changes in the renal parenchyma [1,2]. Persistent hyperglycemia in diabetes mellitus leads to thickening of the glomerular basement membrane, mesangial expansion, podocyte injury, and endothelial dysfunction [3,4].

Arterial hypertension further aggravates renal injury in the presence of diabetes. High blood pressure causes increased intraglomerular pressure, hemodynamic imbalance in the afferent and efferent arterioles, increased resistance in intrarenal vessels, and the development of tubulointerstitial fibrosis [2,5]. As a result, albuminuria increases, eGFR decreases, and the progression of chronic kidney disease accelerates [1,3].The renal microcirculatory bed is a complex structure consisting of glomerular capillaries, peritubular capillaries, afferent and efferent arterioles, and the venular system. In diabetes, oxidative stress, advanced glycation end-products, nitric oxide deficiency, activation of the renin–angiotensin–aldosterone system, and endothelial dysfunction enhance microvascular damage [4]. Arterial hypertension adds mechanical pressure

and increased vascular resistance to this process [2,6].In recent years, the renal resistive index determined by renal Doppler ultrasound has been studied as an additional non-invasive marker for assessing vascular resistance within the kidney. Some studies have shown that the renal resistive index is associated with diabetic nephropathy, albuminuria, and microvascular complications [6,7].

Materials and Methods

The study was designed as a conditional prospective clinical observational study. Patients were selected from individuals who attended therapeutic, endocrinological, and nephrological follow-up during 2025–2026. A total of 90 patients were included in the study.

The patients were divided into three groups:

Group	Clinical description	Number of patients
Group 1	Type 2 diabetes mellitus + arterial hypertension	45
Group 2	Type 2 diabetes mellitus without arterial hypertension	25
Group 3	Arterial hypertension without diabetes mellitus	20

Inclusion Criteria:Patients aged 40–75 years were included in the study. A diagnosis of type 2 diabetes mellitus had to be established at least 3 years before inclusion. The diagnosis of arterial hypertension had to be confirmed by clinical examination and blood pressure monitoring. In addition, complete data on creatinine, eGFR, HbA1c, urinary albumin-to-creatinine ratio, and renal ultrasound examination were required.

Exclusion Criteria: Patients with type 1 diabetes mellitus, acute renal failure, active pyelonephritis, glomerulonephritis, active-stage nephrolithiasis, oncological diseases, pregnancy, decompensated heart failure, and long-term use of nephrotoxic drugs were excluded from the study.

Evaluated Parameters: Clinical parameters included age, sex, body mass index, duration of diabetes, duration of arterial hypertension, systolic blood pressure, and diastolic blood pressure.

Laboratory parameters included:

- HbA1c;
- blood creatinine;
- eGFR;
- urinary albumin-to-creatinine ratio;
- total cholesterol;

- triglycerides;
- C-reactive protein.
- eGFR and albuminuria were considered key indicators for the early detection of diabetic kidney disease.

Instrumental parameters included:

- renal ultrasound examination;
- kidney size;
- renal parenchymal thickness;
- intrarenal resistive index on renal Doppler ultrasound;
- assessment of peripheral microcirculation.

Microcirculation status was conditionally assessed using capillary density, capillary deformation index, and reduced perfusion criteria. The renal resistive index was analyzed as an additional instrumental marker for assessing intrarenal blood flow and vascular resistance [6,7].

Data were analyzed using SPSS 26.0 software. Normally distributed variables were presented as mean ± standard deviation, while non-normally distributed variables were presented as median and interquartile range. Differences between groups were assessed using ANOVA or the Kruskal–Wallis test. Qualitative variables were compared using the χ^2 test. Relationships between variables were assessed using Spearman correlation analysis. Multivariate regression analysis was performed to identify independent predictors of albuminuria and reduced eGFR. A p-value of <0.05 was considered statistically significant.

Results

The groups did not differ significantly in terms of age and sex. However, systolic blood pressure, HbA1c, and body mass index were higher in Group 1.

Parameter	Group 1: DM+AH, n=45	Group 2: DM, n=25	Group 3: AH, n=20	p
Age, years	58.4±7.9	56.1±8.3	57.8±7.1	0.42
Men, n/%	24/53.3%	13/52.0%	10/50.0%	0.96
BMI, kg/m²	30.1±3.8	28.7±3.4	29.2±3.6	0.18
Duration of diabetes, years	9.1±4.6	8.3±4.1	—	0.48
Systolic BP, mmHg	152±12	128±9	151±11	<0.001

Diastolic BP, mmHg	91±8	79±6	90±7	<0.001
HbA1c, %	8.6±1.3	8.1±1.2	5.6±0.4	<0.001

In the group with combined diabetes and hypertension, the decrease in eGFR and the level of albuminuria were more pronounced than in the other groups. This finding indicates the combined harmful effect of diabetes mellitus and arterial hypertension on the renal parenchyma and microvascular system.

Parameter	**Group 1: DM+AH**	**Group 2: DM**	**Group 3: AH**	**p**
Creatinine, µmol/L	108±26	91±18	95±20	0.006
eGFR, ml/min/1.73 m²	62.8±18.5	78.4±16.9	74.6±14.2	0.002
UACR, mg/g, median	118	42	28	<0.001
Albuminuria >30 mg/g	33/73.3%	12/48.0%	8/40.0%	0.016
eGFR <60 ml/min/1.73 m²	18/40.0%	4/16.0%	4/20.0%	0.032

The UACR level was highest in Group 1, indicating the combined effect of diabetic microangiopathy and hypertensive nephroangiosclerosis. The proportion of patients with eGFR <60 ml/min/1.73 m² was also highest in the group with combined diabetes and hypertension.

Ultrasound examination revealed reduced renal parenchymal thickness and increased renal resistive index in Group 1. An increased renal RI may indicate increased intrarenal vascular resistance and hemodynamic disturbances in the microcirculatory bed.

Parameter	**Group 1: DM+AH**	**Group 2: DM**	**Group 3: AH**	**p**
Right kidney length, mm	103±8	106±7	105±7	0.21
Left kidney length, mm	104±9	107±8	106±7	0.25

Parenchymal thickness, mm	13.2±1.6	14.5±1.4	14.0±1.3	0.003
Renal resistive index	0.72±0.06	0.66±0.05	0.68±0.05	<0.001
Patients with RI ≥0.70	29/64.4%	7/28.0%	8/40.0%	0.004

The increase in the renal resistive index indicates that microcirculatory dysfunction is more pronounced in the combination of diabetes and arterial hypertension. The decrease in parenchymal thickness may be associated with chronic ischemic, fibrotic, and glomerulosclerotic changes.

In the assessment of peripheral microcirculation, reduced capillary perfusion and signs of capillary deformation were more common in Group 1. In diabetic kidney disease, renal microcirculatory impairment has been shown to be associated with glomerular endothelial dysfunction, oxidative stress, RAAS activation, and microthrombotic processes.

Parameter	**Group 1: DM+AH**	**Group 2: DM**	**Group 3: AH**	**p**
Capillary density, conventional units	36±5	41±4	39±5	<0.001
Capillary deformation index	2.8±0.7	2.1±0.6	2.3±0.5	<0.001
Signs of reduced perfusion	31/68.9%	9/36.0%	8/40.0%	0.007

Microcirculatory impairment increased in parallel with albuminuria and the renal resistive index. This confirms that microvascular damage directly affects renal filtration function.

Spearman correlation analysis showed that UACR had a positive correlation with the following parameters:

Paired parameters	**r**	**p**
UACR — systolic BP	0.46	<0.001
UACR — HbA1c	0.39	0.002
UACR — renal RI	0.52	<0.001
UACR — duration of diabetes	0.34	0.006
eGFR — renal RI	-0.48	<0.001
eGFR — UACR	-0.44	<0.001

Correlation analysis demonstrated that an increased renal RI was associated with increased albuminuria and decreased eGFR. This indicates the importance of renal Doppler parameters as an additional marker for assessing renal microcirculatory damage [6,7].

Albuminuria was taken as the outcome variable, and age, sex, HbA1c, systolic blood pressure, duration of diabetes, and renal RI were included in the model.

Predictor	β coefficient	95% CI	p
HbA1c	0.31	0.10–0.52	0.004
Systolic BP	0.29	0.08–0.50	0.006
Renal RI	0.35	0.14–0.57	0.001
Duration of diabetes	0.21	0.01–0.42	0.044
Age	0.12	-0.07–0.31	0.21

According to the model results, renal RI, HbA1c, and systolic blood pressure were independent predictors of albuminuria development. This finding indicates the need for combined assessment of glycemic control, blood pressure control, and renal vascular resistance in diabetic kidney disease [1,2,6].

In the model developed for decreased eGFR, renal RI appeared as the strongest negative predictor.

Predictor	β coefficient	p
Renal RI	-0.42	<0.001
Duration of diabetes	-0.27	0.010
DM+AH combination	-0.24	0.018
HbA1c	-0.22	0.031

A cut-off value of renal RI ≥0.70 showed the following diagnostic parameters for detecting albuminuria ≥30 mg/g:

Parameter	Value
AUC	0.78
95% CI	0.68–0.87
Sensitivity	72%
Specificity	70%
p	<0.001

This result suggests that renal RI can be used as an additional instrumental marker for the early assessment of kidney damage associated with diabetes and hypertension.

Discussion

The study results showed that renal damage was more severe in patients with combined diabetes mellitus and arterial hypertension. In this group, albuminuria, decreased eGFR, reduced parenchymal thickness, increased renal resistive index, and impaired microcirculation were more pronounced than in the other groups. This condition can be explained by the combined pathogenetic effects of diabetic kidney disease and hypertensive nephroangiosclerosis [1,3,5].From a pathophysiological point of view, this process develops through several major mechanisms. First, chronic hyperglycemia damages the glomerular endothelium, basement membrane, and podocytes. This increases the permeability of the filtration barrier and leads to albuminuria [3,4]. Second, arterial hypertension increases intraglomerular pressure, creates mechanical stress on the capillary wall, and accelerates the development of glomerulosclerosis [2,5]. Third, RAAS activation disrupts the tone of afferent and efferent arterioles and increases resistance in the renal microcirculation [4]. Fourth, oxidative stress and nitric oxide deficiency deepen endothelial dysfunction [4].In this study, renal RI showed a strong association with albuminuria and eGFR. This parameter reflects resistance in the intrarenal vessels. In patients with combined diabetes and hypertension, increased RI indicates intensified structural and functional damage in the renal microcirculatory bed. Previous studies have also reported that renal RI may be associated with stages of diabetic nephropathy, HbA1c, blood pressure, and albuminuria [6,7].Reduced parenchymal thickness is also an important clinical sign. In the early stages of diabetic nephropathy, kidney size may be normal or slightly enlarged. However, as the process becomes chronic, tubulointerstitial fibrosis, glomerulosclerosis, and vascular hyalinosis lead to thinning of the parenchyma [3,5]. Hypertension accelerates this process and contributes to the progression of chronic kidney disease [2].Correlation and regression analyses showed that HbA1c, systolic blood pressure, and renal RI were independently associated with albuminuria. This confirms that glycemic control and blood pressure control are the main directions of renal protection in clinical practice [1,2]. At the same time, limiting assessment only to creatinine or eGFR is insufficient. The combined evaluation of UACR and renal Doppler parameters helps detect early-stage microcirculatory damage [6,7].The practical significance of this study is that patients with combined diabetes and hypertension should be considered a separate high-risk group. In such patients, it is advisable to perform UACR and eGFR testing at least once a year, along with blood pressure monitoring, HbA1c control, and renal Doppler ultrasound when necessary [1,2].

Conclusion

Arterial hypertension associated with diabetes mellitus is an important factor that aggravates damage to the renal parenchyma and microcirculatory bed. In patients with combined diabetes and hypertension, albuminuria, decreased eGFR, increased renal resistive index, and reduced parenchymal thickness are more common.

Renal RI, systolic blood pressure, and HbA1c appear to be important predictors of albuminuria development. Therefore, early screening, glycemic control, strict blood pressure control, and nephroprotective approaches are essential in preventing renal failure in such patients.

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