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CLINICAL AND EXPERIMENTAL DYNAMICS OF RENAL PARENCHYMAL AND MICROCIRCULATORY DAMAGE IN ARTERIAL HYPERTENSION ASSOCIATED WITH DIABETES MELLITUS

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Abstract

This study investigates the clinical and experimental dynamics of renal parenchymal and microcirculatory damage in arterial hypertension developing on the background of diabetes mellitus. A combined clinical and experimental approach was applied, including patients with diabetes-associated arterial hypertension and an experimental model reproducing diabetic-hypertensive renal injury. Renal functional parameters, microcirculatory alterations, and morphological changes of the renal parenchyma were analyzed at different stages of disease progression using laboratory, instrumental, and histological methods. The results demonstrate that the coexistence of diabetes mellitus and arterial hypertension leads to an accelerated progression of renal damage, beginning with early microcirculatory dysfunction and subsequently resulting in structural alterations of glomerular and tubular components. Progressive impairment of microcirculation plays a key role in renal tissue hypoxia, inflammatory responses, and fibrotic remodeling. Early detection of microvascular disturbances may therefore be crucial for preventing irreversible renal parenchymal damage in patients with diabetes-associated arterial hypertension.

Keywords: Diabetes mellitus; arterial hypertension; renal parenchyma; microcirculation; diabetic nephropathy; experimental study.

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Introduction

Diabetes mellitus and arterial hypertension are among the most widespread chronic non-communicable diseases and frequently coexist in clinical practice, forming a significant burden on healthcare systems worldwide. The combination of these two conditions markedly increases the risk of target organ damage, with the kidneys being one of the most vulnerable organs due to their complex vascular architecture and high metabolic demands. Persistent hyperglycemia together with elevated arterial pressure creates a pathogenic environment that accelerates renal dysfunction and structural injury.

Renal involvement in diabetes-associated arterial hypertension is closely related to progressive damage of the renal parenchyma and disturbances in the microcirculatory bed. Chronic metabolic imbalance leads to endothelial dysfunction, increased oxidative stress, and impaired autoregulation of renal blood flow. Simultaneously, arterial hypertension enhances intraglomerular pressure and promotes structural remodeling of renal vessels, resulting in glomerular hyperfiltration at early stages and subsequent nephron loss as the disease progresses.

Microcirculatory disorders represent one of the earliest and most critical mechanisms underlying renal injury in this combined pathology. Alterations in capillary perfusion, increased vascular permeability, and microvascular rarefaction contribute to chronic renal tissue hypoxia and trigger inflammatory and fibrotic processes. Over time, these changes lead to irreversible structural damage of the glomerular and tubular compartments of the renal parenchyma, ultimately culminating in chronic kidney disease.

Despite extensive clinical observations, the dynamic relationship between microcirculatory impairment and structural renal damage in the context of diabetes mellitus complicated by arterial hypertension remains insufficiently elucidated. In particular, there is a lack of integrated clinical and experimental data that comprehensively describe the temporal progression of renal

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parenchymal injury and its association with microvascular dysfunction. A better understanding of these mechanisms is essential for identifying early diagnostic markers and developing effective preventive and therapeutic strategies.

In this context, the present study aims to analyze the clinical and experimental dynamics of renal parenchymal and microcirculatory damage in arterial hypertension developing on the background of diabetes mellitus, with a focus on the progression of pathological changes and their underlying pathophysiological mechanisms.

Materials and Methods

The study was conducted using a combined clinical and experimental design to evaluate the dynamics of renal parenchymal and microcirculatory damage in arterial hypertension associated with diabetes mellitus. The clinical part of the study included patients diagnosed with diabetes mellitus complicated by arterial hypertension, who were observed over different stages of disease progression. Diagnosis was established based on clinical examination, laboratory findings, and instrumental investigations in accordance with generally accepted diagnostic criteria. Renal functional status was assessed by evaluating biochemical markers of kidney function, while arterial pressure levels and metabolic parameters were monitored throughout the observation period.

Microcirculatory changes were analyzed using non-invasive and instrumental methods that allowed assessment of renal perfusion and vascular regulation. Particular attention was paid to indicators reflecting endothelial function, tissue perfusion, and microvascular resistance. These parameters were evaluated dynamically to determine their relationship with disease duration and severity.

The experimental part of the study was performed on laboratory animals, in which a model of combined diabetic and hypertensive renal damage was induced. Experimental diabetes mellitus was reproduced using standard pharmacological methods, followed by the induction of arterial hypertension to simulate the

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combined pathological condition. Animals were observed for predetermined time intervals corresponding to early, intermediate, and advanced stages of renal involvement.

Morphological examination of renal tissue was carried out after completion of the experimental protocol. Kidney samples were processed according to standard histological techniques and examined using light microscopy. Structural changes of the renal parenchyma, including glomerular, tubular, and interstitial alterations, as well as microvascular remodeling, were evaluated and compared across different stages of the experiment.

Statistical analysis was performed using conventional methods to assess the significance of differences between groups and to determine correlations between microcirculatory parameters and structural renal changes. The combined clinical and experimental approach allowed a comprehensive assessment of the dynamic relationship between microcirculatory dysfunction and renal parenchymal damage in arterial hypertension developing on the background of diabetes mellitus.

Results

The results of the clinical observations demonstrated a progressive deterioration of renal function in patients with arterial hypertension developing on the background of diabetes mellitus. At the early stages of the disease, functional changes predominated, characterized by subtle alterations in renal hemodynamics and microcirculatory regulation without pronounced structural damage. These changes were accompanied by moderate increases in arterial pressure and metabolic imbalance, which correlated with initial signs of endothelial dysfunction.

As the disease progressed, more pronounced microcirculatory disturbances were observed. Impairment of renal perfusion, increased vascular resistance, and signs of microvascular dysregulation became evident, reflecting a decline in adaptive

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mechanisms of the renal circulation. These alterations were associated with worsening metabolic control and longer duration of both diabetes mellitus and arterial hypertension. Clinically, this stage was characterized by a gradual decline in renal functional indicators, suggesting the transition from functional to structural renal involvement.

In advanced stages, patients exhibited significant renal parenchymal damage accompanied by persistent microcirculatory impairment. Structural alterations of the glomerular and tubular compartments became evident, indicating irreversible changes in renal tissue architecture. The severity of these changes showed a strong correlation with the degree and duration of arterial hypertension, as well as with chronic hyperglycemia.

Experimental findings supported the clinical observations and provided additional insight into the temporal progression of renal injury. In the early experimental period, microcirculatory dysfunction was the predominant finding, manifested by disturbances in capillary perfusion and vascular tone. At intermediate stages, these functional changes were followed by morphological alterations, including glomerular hypertrophy and early tubular damage. Prolonged exposure to combined diabetic and hypertensive conditions resulted in marked structural remodeling of renal tissue, including microvascular rarefaction and parenchymal degeneration.

Overall, the combined clinical and experimental results indicate that microcirculatory disturbances precede and promote structural damage of the renal parenchyma. The progression from functional microvascular dysfunction to irreversible parenchymal injury underscores the critical role of microcirculation in the pathogenesis of renal damage in diabetes-associated arterial hypertension.

Discussion

The findings of the present study demonstrate that arterial hypertension developing on the background of diabetes mellitus leads to a progressive and

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stage-dependent pattern of renal injury, in which microcirculatory disturbances play a central pathogenic role. Both clinical and experimental results consistently indicate that functional alterations of the microvascular bed precede overt structural damage of the renal parenchyma, supporting the concept that microcirculation represents an early and sensitive target in diabetic-hypertensive nephropathy.

At the initial stages, microcirculatory dysfunction appears to be primarily functional and potentially reversible. Endothelial dysregulation, impaired autoregulation of renal blood flow, and increased vascular resistance contribute to subtle perfusion abnormalities without pronounced morphological changes. These early alterations may reflect adaptive responses to chronic hyperglycemia and elevated arterial pressure; however, their persistence creates conditions for progressive tissue hypoxia and metabolic stress within the renal parenchyma.

As the combined pathological process advances, sustained microvascular impairment leads to structural remodeling of renal vessels and parenchymal components. The observed glomerular and tubular alterations can be explained by prolonged intraglomerular hypertension, oxidative stress, and inflammatory activation, which are amplified by the coexistence of metabolic and hemodynamic factors. The experimental data further confirm that prolonged exposure to diabetic and hypertensive conditions accelerates microvascular rarefaction and parenchymal degeneration, emphasizing the cumulative nature of renal injury.

An important aspect highlighted by this study is the close temporal and pathophysiological relationship between microcirculatory disturbances and structural renal damage. The transition from functional microvascular changes to irreversible parenchymal injury underscores the significance of early detection and timely intervention. Identifying microcirculatory dysfunction at subclinical stages may provide an opportunity to prevent or delay the progression of diabetic-hypertensive nephropathy.

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The combined clinical and experimental approach strengthens the validity of the findings by allowing direct comparison between observed patient data and controlled experimental models. This integrative perspective enhances the understanding of disease dynamics and supports the translational relevance of experimental observations to clinical practice. Nevertheless, further studies involving larger patient cohorts and advanced microcirculatory assessment techniques may help refine early diagnostic criteria and therapeutic strategies. Overall, the results emphasize that renal damage in diabetes-associated arterial hypertension is not merely the consequence of isolated metabolic or hemodynamic factors, but rather the outcome of their synergistic interaction mediated through persistent microcirculatory impairment. Addressing microvascular dysfunction may therefore represent a key target in the prevention and management of renal complications in this patient population.

Conclusion

The present study demonstrates that arterial hypertension developing on the background of diabetes mellitus is associated with a progressive pattern of renal injury characterized by early microcirculatory dysfunction followed by structural damage of the renal parenchyma. Both clinical and experimental findings indicate that disturbances in the microvascular bed play a decisive role in the initiation and progression of diabetic-hypertensive renal damage.

Microcirculatory impairment emerges as an early pathogenic mechanism, preceding overt morphological alterations and contributing to chronic renal tissue hypoxia, inflammatory activation, and fibrotic remodeling. Persistent exposure to combined metabolic and hemodynamic stressors accelerates the transition from functional disturbances to irreversible parenchymal injury, ultimately increasing the risk of chronic kidney disease.

The close relationship between microcirculatory dysfunction and structural renal changes highlights the importance of early detection and targeted intervention.

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Assessment of microvascular alterations may serve as a valuable tool for identifying patients at high risk of progressive renal damage and for optimizing preventive and therapeutic strategies.

In conclusion, addressing microcirculatory disturbances in patients with diabetes-associated arterial hypertension may represent a key approach to slowing the progression of renal parenchymal damage and improving long-term renal outcomes.

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