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MORPHOLOGICAL FEATURES OF RENAL CORTICAL BLOOD VESSELS IN EXPERIMENTAL METABOLIC SYNDROME

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Abstract. Metabolic syndrome is a complex pathological condition characterized by a combination of obesity, insulin resistance, arterial hypertension, and dyslipidemia, which significantly affect the structure and function of internal organs, particularly the kidneys. Vascular alterations within the renal cortex play a crucial role in the progression of renal dysfunction associated with metabolic disturbances.

The aim of this study was to investigate the morphological features of renal cortical blood vessels in experimental metabolic syndrome. An experimental study was conducted using laboratory animals with induced metabolic syndrome. Histological examination of renal tissue was performed to evaluate vascular structural changes within the cortical layer of the kidneys. The results demonstrated significant morphological alterations in renal cortical vessels, including vascular wall thickening, narrowing of the vascular lumen, endothelial damage, and perivascular edema. Microcirculatory disturbances and signs of sclerosis were also observed in some specimens. These changes were more pronounced in the experimental group compared to controls.

In conclusion, experimental metabolic syndrome leads to substantial morphological remodeling of renal cortical blood vessels, contributing to impaired renal microcirculation and progression of renal pathology. Early detection of vascular alterations may be important for preventing chronic kidney damage associated with metabolic disorders.

Keywords. Metabolic syndrome, kidney, renal cortex, blood vessels, morphology, microcirculation, endothelial damage, nephropathy

Introduction

Metabolic syndrome is a complex metabolic disorder characterized by the coexistence of obesity, insulin resistance, arterial hypertension, and lipid metabolism disturbances. In recent decades, the prevalence of this condition has increased considerably, becoming one of the major medical and social problems worldwide. The syndrome affects multiple organ systems and is closely associated with the development of cardiovascular and renal complications.

The kidneys are among the primary target organs affected by metabolic disturbances due to their intensive blood supply and high sensitivity to hemodynamic and biochemical changes. Persistent hyperglycemia, oxidative stress, and chronic low-grade inflammation contribute to progressive vascular injury within renal tissue. These pathological processes lead to structural remodeling of blood vessels and impairment of renal microcirculation.

The renal cortex contains a highly specialized vascular network responsible for maintaining filtration processes and tissue perfusion. Structural integrity of cortical blood vessels is essential for normal nephron function. Under conditions of metabolic syndrome, vascular walls undergo gradual pathological changes, including thickening, endothelial damage, narrowing of

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the vascular lumen, and sclerosis. Such alterations reduce blood flow efficiency and may initiate progressive renal dysfunction.

Microcirculatory disturbances within the renal cortex are considered one of the early manifestations of metabolic kidney injury. Endothelial dysfunction plays a central role in this process by disrupting vascular tone regulation and increasing vascular permeability. As a result, tissue hypoxia and inflammatory reactions develop, contributing to further structural damage of renal tissue.

Experimental models of metabolic syndrome provide important opportunities for studying the mechanisms of vascular remodeling in the kidneys. Histological and morphometric analysis of renal cortical vessels allows objective evaluation of pathological alterations and helps clarify the relationship between metabolic disturbances and renal injury.

Understanding the morphological features of renal cortical blood vessels in metabolic syndrome is important for early diagnosis and prevention of chronic kidney disease. Identification of early vascular changes may contribute to the development of more effective therapeutic and preventive strategies aimed at preserving renal function.

Therefore, the aim of the present study was to investigate the morphological characteristics of renal cortical blood vessels in experimental metabolic syndrome and evaluate the degree of vascular remodeling associated with metabolic disturbances.

Materials and Methods.

This experimental study was conducted to evaluate the morphological characteristics of renal cortical blood vessels under conditions of metabolic syndrome. The investigation included 24 mature white laboratory rats weighing 180–220 g. The animals were divided into two groups: a control group and an experimental group, with 12 animals in each group.

Experimental metabolic syndrome was induced through long-term administration of a high-calorie diet enriched with fats and carbohydrates over a 12-week period. During the experiment, body weight, fasting blood glucose levels, and arterial blood pressure were monitored to assess the development of metabolic disturbances.

At the end of the experimental period, the animals were euthanized under standard laboratory conditions. Kidney tissue samples were collected immediately and fixed in 10% neutral buffered formalin. After fixation, the specimens were processed using conventional histological techniques and embedded in paraffin blocks.

Histological sections measuring 5–7 µm in thickness were prepared using a rotary microtome. The sections were stained with hematoxylin and eosin for general morphological assessment. Microscopic examination was performed using a light microscope equipped with a digital imaging system.

The study focused on morphological evaluation of renal cortical blood vessels, including:

- interlobular arteries
- arterioles
- capillary structures

Special attention was paid to the following parameters:

- vascular wall thickness
- vascular lumen diameter
- endothelial condition
- presence of perivascular edema
- signs of sclerosis and microcirculatory disorders

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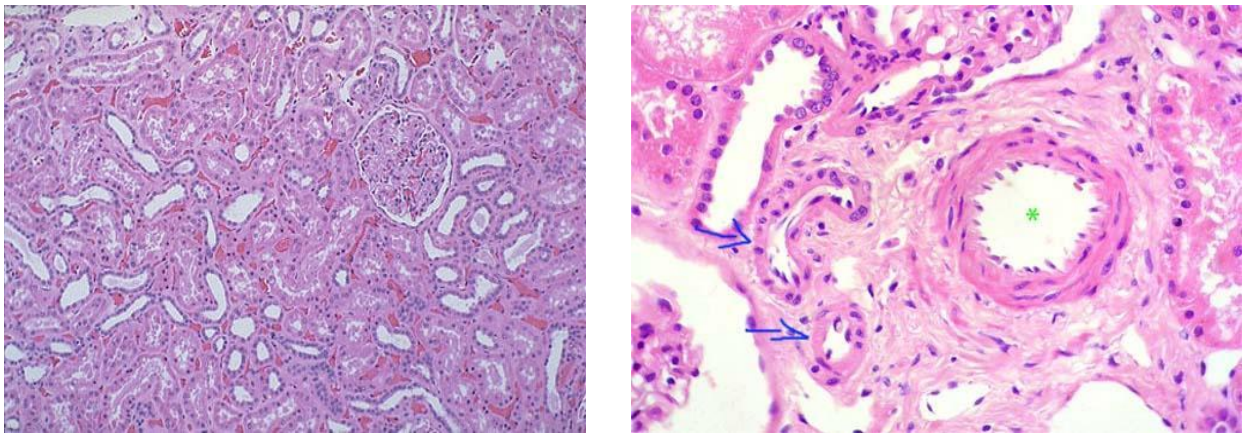
Morphometric measurements were performed using digital image analysis software. Multiple measurements were obtained from each specimen, and mean values were calculated for comparative analysis between groups.

Table 1. Morphological Parameters Evaluated in Renal Cortical Blood Vessels

Parameter	Evaluation Criteria	Diagnostic Significance
Vascular wall thickness	Microscopic morphometry	Indicates vascular remodeling
Vascular lumen diameter	Measurement of arterial lumen	Reflects circulatory impairment
Endothelial integrity	Histological assessment	Evaluates endothelial damage
Perivascular edema	Presence of fluid accumulation	Indicates microcirculatory disorder
Sclerotic alterations	Structural changes in vessel walls	Suggests chronic vascular injury

Note. Morphological assessment was performed using standard histological and morphometric methods.



Note. Representative histological images of renal cortical blood vessels obtained during microscopic examination.

All obtained data were processed using descriptive statistical analysis. Quantitative values were expressed as mean ± standard deviation, and comparative evaluation between groups was performed to identify the severity of vascular alterations.

Results

Microscopic examination of renal cortical tissue demonstrated significant structural alterations in the blood vessels of the experimental group compared with the control group. The most pronounced pathological changes were observed in interlobular arteries, arterioles, and cortical capillary networks.

In the control group, renal cortical vessels maintained normal histological architecture. Vascular walls were thin and uniform, endothelial cells were intact, and vascular lumens remained well preserved. No signs of edema, sclerosis, or circulatory disturbances were identified within the cortical layer.

In contrast, the experimental group with induced metabolic syndrome exhibited marked vascular remodeling. Thickening of vascular walls was observed in the majority of specimens,

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accompanied by narrowing of the vascular lumen. Endothelial cells showed structural irregularities, including swelling and partial desquamation. In several sections, perivascular edema and congestion of capillary vessels were detected.

Morphometric analysis revealed a significant increase in vascular wall thickness in the experimental group, while lumen diameter was considerably reduced. These findings indicate progressive impairment of renal microcirculation associated with metabolic disturbances.

Table 2. Morphometric Characteristics of Renal Cortical Blood Vessels

Parameter	Control Group	Experimental Group
Vascular wall thickness (μm)	12.3 ± 1.1	19.5 ± 2.3
Vascular lumen diameter (μm)	29.1 ± 2.4	18.2 ± 1.9
Endothelial condition	Preserved	Partially damaged
Perivascular edema	Absent	Moderate
Signs of vascular sclerosis	Not detected	Present

Note. Morphometric data obtained from histological analysis of renal cortical blood vessels.

Additional histological findings included focal thickening of the basal membrane and areas of vascular sclerosis within cortical tissue. Capillary congestion and impaired blood flow were more pronounced in animals with severe manifestations of metabolic syndrome.

The observed vascular alterations suggest that metabolic syndrome induces progressive structural remodeling of renal cortical vessels, leading to deterioration of renal microvascular circulation and potential impairment of kidney function.

Figure 2. Histological Changes in Renal Cortical Blood Vessels

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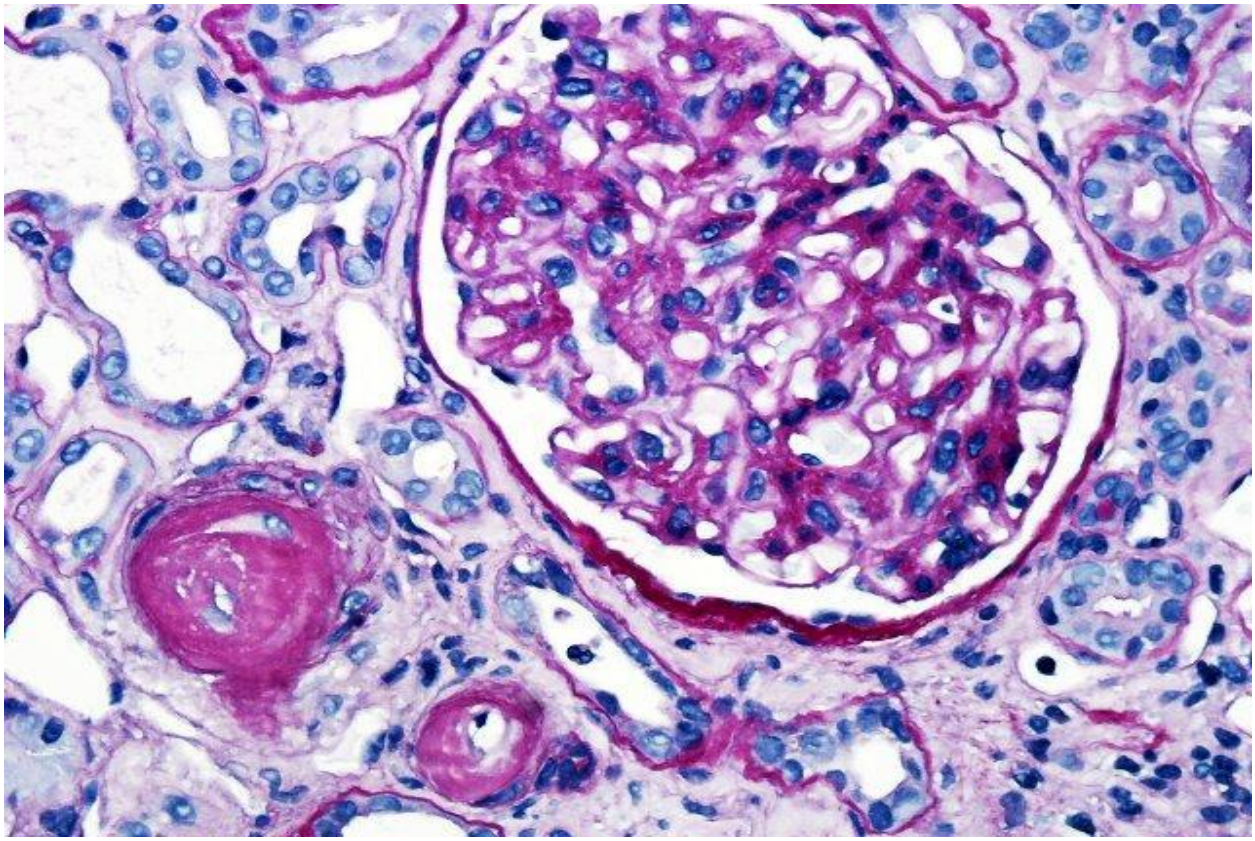

Note. Representative histological images demonstrating vascular wall thickening, endothelial injury, and microcirculatory disturbances in the renal cortex under experimental metabolic syndrome conditions.

Discussion

The results of the present study demonstrate that experimental metabolic syndrome leads to significant structural remodeling of renal cortical blood vessels. The observed vascular alterations, including wall thickening, narrowing of vascular lumens, endothelial damage, and perivascular edema, indicate progressive impairment of renal microcirculation under metabolic stress conditions.

One of the major findings of this study was the increase in vascular wall thickness in the experimental group. This change may be associated with chronic hyperglycemia, oxidative stress, and persistent hemodynamic overload characteristic of metabolic syndrome. Long-term metabolic disturbances promote hypertrophy of vascular smooth muscle cells and accumulation of connective tissue components within the vascular wall, resulting in reduced vascular elasticity and impaired blood flow.

Endothelial damage identified in the experimental group is of particular importance because endothelial dysfunction is considered one of the earliest pathological mechanisms in metabolic syndrome. Structural disruption of endothelial cells contributes to altered vascular permeability, impaired regulation of vascular tone, and activation of inflammatory pathways. These processes aggravate tissue hypoxia and accelerate vascular remodeling within the renal cortex.

The narrowing of vascular lumens observed during morphometric analysis suggests a progressive reduction in renal perfusion. Impaired cortical blood supply may contribute to

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glomerular ischemia, decreased filtration efficiency, and gradual deterioration of renal function. In addition, capillary congestion and perivascular edema detected in this study indicate disturbances of local microcirculation and increased vascular permeability.

Another important observation was the presence of focal sclerosis in renal cortical vessels. Sclerotic alterations reflect chronic vascular injury and may represent an early stage in the development of nephropathy associated with metabolic syndrome. Progressive fibrosis and vascular rigidity further compromise renal hemodynamics and contribute to chronic kidney damage.

The findings of this study confirm that metabolic syndrome affects not only systemic metabolism but also the structural integrity of renal microvascular networks. Morphological and morphometric evaluation of renal cortical vessels provides valuable information regarding the severity of vascular injury and progression of renal pathology.

Despite the informative results, several limitations should be acknowledged. The study was conducted on an experimental animal model, which may not fully reproduce all aspects of human metabolic syndrome. In addition, the duration of the experimental period may influence the degree of vascular alterations observed. Further investigations involving longer observation periods and additional molecular analysis may provide deeper understanding of the mechanisms underlying renal vascular remodeling.

In conclusion, experimental metabolic syndrome causes pronounced morphological changes in renal cortical blood vessels, leading to impaired microcirculation and structural renal injury. Early identification of vascular alterations may play an important role in preventing progression of metabolic nephropathy and preserving renal function.

Conclusion

In conclusion, experimental metabolic syndrome causes significant morphological alterations in the blood vessels of the renal cortex. The study demonstrated pronounced vascular wall thickening, narrowing of vascular lumens, endothelial injury, perivascular edema, and focal sclerotic changes within renal cortical tissue.

These structural alterations indicate progressive impairment of renal microcirculation and may contribute to the development of metabolic nephropathy. Morphometric analysis confirmed that metabolic disturbances are associated with substantial remodeling of renal cortical vessels and deterioration of vascular integrity.

The findings emphasize the importance of early detection of vascular changes in metabolic syndrome. Histological and morphometric evaluation of renal blood vessels may provide valuable diagnostic information for assessing the severity of renal involvement and predicting progression of kidney damage.

Overall, metabolic syndrome exerts a significant negative effect on the renal microvascular system, highlighting the need for timely preventive and therapeutic strategies aimed at preserving renal function and reducing the risk of chronic kidney disease.

References

1. Alberti, K. G. M. M., Zimmet, P., & Shaw, J. (2006). Metabolic syndrome — a new worldwide definition. *The Lancet, 366*(9491), 1059–1062.
2. Reaven, G. M. (1988). Banting lecture 1988: Role of insulin resistance in human disease. *Diabetes, 37*(12), 1595–1607.
3. Eckel, R. H., Grundy, S. M., & Zimmet, P. Z. (2005). The metabolic syndrome. *The Lancet, 365*(9468), 1415–1428.

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https://westerneuropeanstudies.com/index.php/3
ISSN (E): 2942-1918 Open Access| Peer Reviewed

4. DeFronzo, R. A. (2010). Insulin resistance, lipotoxicity, type 2 diabetes and atherosclerosis. *Diabetes Care, 33*(2), 453–461.
5. Zakirova, G., Masharipova, D., Boboev, Q., & Tagaeva, D. (2026). T-786C Polymorphism of the NOS3 Gene and Its Role in the Development of Renal Dysfunction in Patients of the Uzbek Population with Chronic Heart Failure. *Turk Kardiyoloji Dernegi Arsivi: Turk Kardiyoloji Derneginin Yayin Organidir*.
6. Arutyunov, G. P., Tarlovskaya, E. I., Arutyunov, A. G., Batluk, T. I., Koziolova, N. A., Chesnikova, A. I., ... & Shchukin, Y. V. (2024). Patients With Non-Obstructive Coronary Artery Disease and Polyvascular Disease. Sub-Analysis of the Real-World Registry KAMMA (Clinical Registry on Patient Population With Polyvascular Disease in the Russian Federation and Eurasian Countries). *Kardiologiia*, *64*(8), 13-23.
7. Arutyunov, G. P., Tarlovskaya, E., Arutyunov, A. G., Belenkov, Y. N., Konradi, A. O., Lopatin, Y. M., ... & Omarova, Y. (2022). ANALYSIS OF THE IMPACT OF COMORBID CARDIOVASCULAR PATHOLOGY TO THE COURSE OF COVID-19 AND ITS'OUTCOMES IN INPATIENTS ADMITTED TO HOSPITAL DURING THE FIRST AND THE SECOND WAVES OF THE NOVEL CORONAVIRUS INFECTION IN THE EURASIAN REGIONE. *KARDIOLOGIYA*, *62*(12), 37-48.
8. Alisherovna, Z. G., Kabirovna, K. U., Dadaevna, R. Z., & Akhmedjanovna, K. R. (2019). Role of the neurohumoral factors in the progression of chronic heart failure and kidney dysfunction. *European science review*, *2*(1-2), 104-107.
9. Arutyunov, G. P., Tarlovskaya, E. I., Arutyunov, A. G., Polyakov, D. S., Grigorieva, N. Y., Gubareva, I. V., ... & Shcherbakov, S. Y. (2023). Lipid profile changes after the acute COVID-19 period. Sub-analysis of the International Registry" Dynamics Analysis of Comorbidities in SARS-CoV-2 Survivors"(AKTIV SARS-CoV-2)"(12-month follow-up). *Russian Journal of Cardiology*, *29*(3), 5716.
10. ZAKIROVA, G., MASHARIPOVA, D., DUSANOVA, N., MIRZAEV, R., & SADIEVA, Z. ASSESSMENT OF RENAL FUNCTION IMPAIRMENT IN INDIVIDUALS SUFFERING FROM CHRONIC HEART FAILURE. Scientific publishing house Infinity, Ufa КОНФЕРЕНЦИЯ: SCIENTIFIC RESEARCH OF THE SCO COUNTRIES: SYNERGY AND INTEGRATION Beijing, 12 февраля 2025 года Организаторы: Scientific publishing house Infinity, Ufa.
11. ZAKIROVA, G., MASHARIPOVA, D., DUSANOVA, N., MIRZAEV, R., & SADIEVA, Z. ASSESSMENT OF RENAL FUNCTION IMPAIRMENT IN INDIVIDUALS SUFFERING FROM CHRONIC HEART FAILURE. Scientific publishing house Infinity, Ufa КОНФЕРЕНЦИЯ: SCIENTIFIC RESEARCH OF THE SCO COUNTRIES: SYNERGY AND INTEGRATION Beijing, 12 февраля 2025 года Организаторы: Scientific publishing house Infinity, Ufa.
12. Иргашева, С. У., & Гуламмахмудова, Д. В. (2014). Анализ эффективности лечения нарушений репродуктивного здоровья у больных с СПКЯ. *Молодой ученый*, (11), 145-149.
13. Valijanovna, G. D. (2025). REPRODUKTIV YOSHDAGI AYOLLARDA POLIKISTOZ TUXUMDON SINDROMINI TASHXISLASHDA ZAMONAVIY YONDASHUVLAR. *Лучшие интеллектуальные исследования*, *53*(1), 361-369.
14. Valijanovna, G. D. (2025). GIPERANDROGENIYA RIVOJLANISHINING PATOGENETIK JIHATLARI VA U BILAN BOG ‘LIQ BO ‘LGAN BIR QATOR

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METABOLIK ASORATLAR VA TPKS BILAN OG ‘RIGAN AYOLLARDA ULARNI TUZATISH. *Лучшие интеллектуальные исследования*, *53*(1), 370-382.
15. Valijanovna, G. D. (2016). Modern methods of treatment of Polycystic Ovary Syndrome. *European science review*, (11-12), 47-48.
16. Гуламмахмудова, Д. В. (2015). Состояние репродуктивного здоровья и гормонального статуса у женщин с синдромом поликистозных яичников. *ББК 60 М 43*, 63.
17. Valijanovna, G. D. (2015). Reproductive health and hormonal status of women with polycystic ovary syndrome. *European science review*, (9-10), 57-60.
18. Ахмеджанова, Х. З., & Шукуров, Ф. И. (2024). Инновационные подходы к ведению женщин с бесплодием позднего репродуктивного возраста с учетом овариального резерва. *TAHRIRIYAT HAY’ATI*, 29.
19. Олимова, К. Ж., & Ахмеджанова, Х. З. (2024). Инновационный метод в диагностике синдрома пустых фолликулов.
20. Ахмеджанова, Х. З., & Шукуров, Ф. И. (2024). Эффективность экзосомальной терапии у женщин позднего репродуктивного возраста с низким овариальным резервом. *Москва*, *4*, 07.
21. Ахмеджанова, Х. З. (2025). 3D-эластография овариальной стромы при низком овариальном резерве: клинико-прогностическое значение и роль в выборе терапевтической тактики.
22. Маликова, Ш. Н., & Ахмеджанова, Х. З. (2026). ТУРЛИ РЕПРОДУКТИВ ЁШДАГИ АЁЛЛАРДА ОВАРИАЛ ЗАХИРАНИНГ БИОКИМЁВИЙ ВА ЭХОГРАФИК КЎРСАТКИЧЛАРИНИНГ ҚИЁСИЙ КЛИНИК ТАҲЛИЛИ. *Eurasian Journal of Medical and Natural Sciences*, *6*(1), 43-50.
23. Ахмеджанова, Х. З. (2025). ПАСТ ОВАРИАЛ ЗАХИРАЛИ КЕЧ РЕПРОДУКТИВЁШДАГИ АЁЛЛАРДА ЭНДОМЕТРИЙ РЕЦЕПТИВЛИГИНИНГ КЛИНИК ВА ЭХОГРАФИК ХУСУСИЯТЛАРИ. *Eurasian Journal of Medical and Natural Sciences*, *5*(12), 270-280.
24. Рашидова, К. Б., & Ахмеджанова, Х. З. (2026). ҲАЙЗЦИКЛИ БУЗИЛИШЛАРИ БИЛАН КЕЧУВЧИ АЁЛЛАРДА ГОРМОНАЛ ПРОФИЛ ЎЗГАРИШЛАРИ. *Eurasian Journal of Medical and Natural Sciences*, *6*(1), 34-42.
25. Мадолимова, Н. Ҳ., & Ахмеджанова, Х. З. (2025). АДЕНОМИОЗ И ОСЛОЖНЕНИЙ БЕРЕМЕННОСТИ, РОДОВ И ИХ ПРОГНОЗИРОВАНИЕ. *ОСНОВЫ МЕДИЦИНЫ*, *1*(7), 177-187.
26. Ахмеджанова, Х. З., & Шукуров, Ф. И. (2024). Гормональный статус у женщин позднего репродуктивного возраста с низким овариальным резервом. *Журнал гуманитарных и естественных наук*, (16 [1]), 91-99.
27. Шукуров, Ф. И., Ахмеджанова, Х. З., & Мадолимова, Н. Х. (2026). Современные подходы к восстановлению фертильности у женщин позднего репродуктивного возраста с низким овариальным резервом: обзор литературы.
28. Tervaert, T. W. C., Mooyaart, A. L., Amann, K., et al. (2010). Pathologic classification of diabetic nephropathy. *Journal of the American Society of Nephrology, 21*(4), 556–563.
29. Forbes, J. M., & Cooper, M. E. (2013). Mechanisms of diabetic complications. *Physiological Reviews, 93*(1), 137–188.

Western European Journal of Medicine and Medical Science
Volume 4, Issue 05, May 2026
https://westerneuropeanstudies.com/index.php/3
ISSN (E): 2942-1918 Open Access| Peer Reviewed

30. Thomas, M. C., Brownlee, M., Susztak, K., et al. (2015). Diabetic kidney disease. *Nature Reviews Disease Primers, 1*, 15018.
31. Gilbert, R. E. (2017). Proximal tubulopathy: Prime mover and key therapeutic target in diabetic kidney disease. *Diabetes, 66*(4), 791–800.
32. Vallon, V., & Komers, R. (2011). Pathophysiology of the diabetic kidney. *Comprehensive Physiology, 1*(3), 1175–1232.
33. Magee, G. M., Bilous, R. W., Cardwell, C. R., et al. (2009). Is hyperfiltration associated with the future risk of developing diabetic nephropathy? *Diabetologia, 52*(4), 691–697.