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КЛИНИКО-ГЕНЕТИЧЕСКОЕ ПРОГНОЗИРОВАНИЕ РИСКА РАЗВИТИЯ ХРОНИЧЕСКОЙ БОЛЕЗНИ ПОЧЕК У БОЛЬНЫХ С МЕТАБОЛИЧЕСКИМ СИНДРОМОМ

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АННОТАЦИЯ

В связи с возрастанием частоты метаболического синдрома (МС) в мире особо актуальным становится вопрос прогнозирования риска хронической болезни почек (ХБП), являющейся одним из частых осложнений МС. Цель этой обзорной статьи — осветить современные данные о связи МС и ХБП, проведенных многоцентровых исследованиях и генетических факторах, а также перспективах клинко-генетического прогнозирования.

Метаболический синдром включает сочетание абдоминального ожирения, артериальной гипертензии, нарушения углеводного обмена и дислипидемии. Этот синдром оказывает независимое влияние на риск поражения почек. По данным иранского когортного исследования (15 255 чел.), поражение почек на фоне МС обнаружено с $HR = 1,51$. В метаанализе 32 когорт (более 400 тыс. наблюдений) установлено, что МС повышает риск хронической почечной недостаточности на 47% ($RR = 1,47$).

Генетически обусловленное снижение eGFR подтверждено в исследованиях Mendelian randomization. Связь генотипов ALPK1, CDH4, PTPRN2 с повышенным риском ХБП наблюдалась в кохортах в Японии. Эти гены влияют на метаболизм жиров, инсулинорезистентность и хроническое воспаление.

Современные подходы включают комплексную оценку: клинические параметры (eGFR, альбуминурия, СРБ, HOMA-IR), генотипирование и искусственный интеллект для построения моделей риска. Это открывает возможности для ранней диагностики, таргетной терапии (блокада RAAS, SGLT2-ингибиторы, метаболическая коррекция).

Ключевые слова: метаболический синдром, хроническая болезнь почек, генетические маркеры, прогнозирование риска, полиморфизмы генов, Mendelian randomization, инсулинорезистентность, альбуминурия, eGFR, клинко-генетические модели

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CLINICAL AND GENETIC PREDICTION OF CHRONIC KIDNEY DISEASE RISK IN PATIENTS WITH METABOLIC SYNDROME

ANNOTATION

Due to the increasing prevalence of metabolic syndrome (MS) worldwide, predicting the risk of chronic kidney disease (CKD), one of the frequent complications of MS, has become particularly relevant. The purpose of this review article is to highlight current data on the relationship between MS and CKD, multicenter studies conducted, and genetic factors, as well as prospects for clinical and genetic prediction.

Metabolic syndrome comprises a combination of abdominal obesity, arterial hypertension, carbohydrate metabolism disorders, and dyslipidemia. This syndrome independently affects the risk of kidney damage. According to an Iranian cohort study (15,255 participants), kidney damage associated with MS was detected with a hazard ratio (HR) of 1.51. A meta-analysis of 32 cohorts (over 400,000 observations) established that MS increases the risk of chronic renal failure by 47% (relative risk, $RR = 1.47$).

A genetically determined decrease in estimated glomerular filtration rate (eGFR) has been confirmed in Mendelian randomization studies. An association between ALPK1, CDH4, and PTPRN2 genotypes and increased risk of CKD was observed in Japanese cohorts. These genes influence fat metabolism, insulin resistance, and chronic inflammation.

Modern approaches include comprehensive assessment: clinical parameters (eGFR, albuminuria, C-reactive protein, HOMA-IR), genotyping, and artificial intelligence for constructing risk models. This opens up opportunities for early diagnosis and targeted therapy (RAAS blockade, SGLT2 inhibitors, metabolic correction).

Keywords: metabolic syndrome, chronic kidney disease, genetic markers, risk prediction, gene polymorphisms, Mendelian randomization, insulin resistance, albuminuria, eGFR, clinical-genetic models

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METABOLIK SINDROMLI BEMORLARDA SURUNKALI BUYRAK KASALLIGI RIVOJLANISH XAVFINI KLINIK-GENETIK BASHORATLASH

ANNOTATSIYA

Dunyoda metabolik sindrom (MS) chastotasining ko'payishi munosabati bilan MSning eng ko'p uchraydigan asoratlardan biri bo'lgan surunkali buyrak kasalligi (SBK) xavfini bashorat qilish masalasi alohida dolzarblik kasb etmoqda. Ushbu sharh maqolasining maqsadi MS va SBK o'rtasidagi aloqa, o'tkazilgan ko'p markazli tadqiqotlar va genetik omillar, shuningdek, klinik-genetik prognozlash istiqbollari haqidagi zamonaviy ma'lumotlarni yoritishdan iborat.

Metabolik sindrom abdominal semizlik, arterial gipertoniya, uglevod almashinuvining buzilishi va dislipidemiyaning uyg'unlashuvini o'z ichiga oladi. Bu sindrom buyrak shikastlanishi xavfiga mustaqil ta'sir ko'rsatadi. Eron kogorta tadqiqoti (15 255 kishi) ma'lumotlariga ko'ra, MS fonida buyrak shikastlanishi HR = 1,51 ko'rsatkich bilan aniqlangan. 32 ta kogortani (400 mingdan ortiq kuzatuv) qamrab olgan meta-tahlilda MS surunkali buyrak yetishmovchiligi xavfini 47% ga oshirishi aniqlangan (RR = 1,47).

Mendelian randomizatsiya tadqiqotlarida eGFRning genetik sabablarga ko'ra pasayishi tasdiqlangan. ALPK1, CDH4, PTPRN2 genotiplarining SBK yuqori xavfi bilan bog'liqligi Yaponiya kogortalarida kuzatilgan. Bu genlar yog'lar almashinuviga, insulina rezistentlikka va surunkali yallig'lanishga ta'sir ko'rsatadi.

Zamonaviy yondashuvlar kompleks baholashni o'z ichiga oladi: klinik ko'rsatkichlar (eGFR, albuminuriya, CRO, HOMA-IR), genotiplash va xavf modellarini yaratish uchun sun'iy intellekt. Bu erta tashxis qo'yish, maqsadli davolash (RAAS blokadasi, SGLT2-ingibitorlari, metabolik korreksiya) imkoniyatlarini ochib beradi.

Kalit so'zlar: metabolik sindrom, surunkali buyrak kasalligi, genetik markerlar, xavfni bashorat qilish, gen polimorfizmlari, Mendelian randomizatsiyasi, insulinrezistentlik, albuminuriya, eGFR, klinik-genetik modellar

Chronic kidney disease (CKD) is one of the most common and socially significant complications of metabolic syndrome. According to epidemiological studies, up to 30-50% of patients with metabolic syndrome (MS) have signs of renal dysfunction, especially in the early, subclinical stages. CKD contributes to increased cardiovascular mortality, decreased quality of life, and progression of metabolic disorders[1,2,6].

Given the heterogeneity of clinical manifestations and differences in the rate of CKD progression in the context of MS, the task of identifying predisposition to renal pathology using molecular genetic and clinical laboratory markers becomes relevant. Chronic kidney disease is currently one of the global health problems, with growing prevalence and high risk of fatal outcomes, primarily due to cardiovascular complications. According to the Global Burden of Disease study, CKD is among the top ten leading causes of death worldwide, showing steady growth over the past two decades. At the same time, metabolic syndrome - a complex of metabolic and cardiovascular disorders including insulin resistance, visceral obesity, arterial hypertension, dyslipidemia, and carbohydrate metabolism disorders - has become extremely common in both industrialized countries and those with transitional economies[3,5,11].

The number of patients with MS is increasing annually, and accumulated scientific data indicate its significant contribution to the development and progression of CKD. Metabolic disorders lead to structural and functional changes in the kidneys, including the development of albuminuria, decreased glomerular filtration rate (GFR), activation of the renin-angiotensin-aldosterone system (RAAS), oxidative stress, and interstitial fibrosis. The role of systemic chronic inflammation accompanying MS is especially significant, as it contributes to damage of the endothelium and nephrons.

Despite the evident clinical relationship between MS components and CKD development, the molecular and genetic mechanisms of this interaction remain incompletely understood. Modern approaches, including genomics, epigenetics, metabolomics, as well as tools such as Mendelian randomization analysis and genetic polymorphism panels, open up opportunities for more accurate risk stratification and individualization of preventive and treatment strategies[4,6,9].

The relevance of studying clinical and genetic factors of CKD development in patients with MS is determined by the need for early identification of high-risk groups, development of personalized prediction models, and optimization of nephroprotective therapy. A review of current scientific literature, including large-scale multicenter studies, systematic reviews, and genetic analyses, allows for a comprehensive examination of the problem and outlines solutions in clinical practice[7,8,12,16].

Pathogenetic mechanisms of CKD in metabolic syndrome

The main pathogenetic links are:

- Hyperglycemia and insulin resistance leading to glucose toxicity and impaired glomerular filtration.
- Activation of the renin-angiotensin-aldosterone system (RAAS), contributing to kidney fibrosis.
- Oxidative stress and chronic inflammation that provoke endothelial damage and interstitial nephritis.
- Hypertension and microalbuminuria, which are early manifestations of kidney damage.

Genetic predisposition to CKD in patients with MS

Among the genes associated with the risk of developing chronic kidney disease, the most attention is paid to the following:

Gen	Polymorphism	Associated effects
ACE	I/D	The D-allele is associated with RAAS hyperactivity and progressive nephropathy
AGT, AGTR1	M235T, A1166C	Elevated vascular tone, kidney sclerosis
eNOS (NOS3)	G894T	Decreased NO production, deterioration of endothelial function
SLC2A9, SLC22A2	Different	Uric acid transport disorder, hyperuricemia
TNF- α , IL-6	-308G>A, -174G>C	Increased production of pro-inflammatory cytokines
PPAR γ	Pro12Ala	Regulation of lipid and glucose metabolism, insulin resistance

Polymorphisms of these genes contribute to the formation of an unfavorable metabolic and inflammatory background, accelerating the development of nephropathy [13,19,26].

Clinical and laboratory predictors of CKD risk in patients with MS

- Decrease in glomerular filtration rate (GFR) below 90 ml/min/1.73 m²

- Microalbuminuria (30-300 mg/day)
- Hyperuricemia
- Elevated levels of CRP, IL-6, TNF- α
- Insulin resistance index (HOMA-IR)
- Increased body mass index (BMI) and waist circumference

The integration of these parameters into prognostic models, taking into account the genetic background, allows for risk stratification of patients and individualization of the approach to the prevention and treatment of CKD.

Prospects of clinical and genetic prediction
Creating personalized risk models, including:

- Genotyping of polymorphisms
- Metabolic and biochemical indicators
- Artificial intelligence and machine learning

...allows for more accurate prediction of the development and progression of CKD, as well as the selection of individual treatment regimens, including antihypertensive and metabolic therapy, and nephroprotection [14,17,20].

The effect of metabolic syndrome (MS) on the risk of CKD - major studies

A large prospective Iranian study (15,255 people, observation 1999-2018) showed that patients with MS, diabetes, and hypertension have a 51% higher risk of developing CKD (HR = 1.51; 95% CI 1.32-1.71) compared to the group without MS. At the same time, obesity and low HDL were not significant factors. A meta-analysis of 24 cohorts (6.6 million people) revealed that MS is associated with an increased risk of CKD (OR = 1.42; 95% CI 1.28-1.57), albuminuria/proteinuria (OR = 1.43), rapid decrease in eGFR (OR = 1.25). The "dose effect" is demonstrated: the more MS components present, the higher the risk. Carte blanche: a meta-analysis of 32 cohorts with 413,621 participants showed an increase in the risk of new cases of CKD (RR = 1.47), ESRD (RR = 1.55), and, most significantly, an increase in blood pressure (RR = 1.37). A cross-sectional Chinese study (1,969 people, age ≥ 40 years) revealed an independent relationship between MS or its components (from blood pressure levels to CRP, HOMA-IR) and CKD even in the absence of diabetes or obesity. Genetic and causal data from Mendelian randomization (MR-analyses): Using GWAS data, it was shown that genetically determined MS components (blood pressure, obesity, diabetes, TG) indeed cause decreased kidney function (eGFR), increased BUN, and albuminuria. This confirms the quasi-causal link between MS and CKD. A TW MR-study in March 2024 confirms that individual components of MS (weight, blood pressure, diabetes) have a causal effect on kidney function decline. Pathophysiology: From Mechanism to Clinic [25,26].

A comprehensive systematic review (2024) described key mechanisms: activation of the RAAS system, chronic inflammation, oxidative stress, and endothelial dysfunction. Adipose tissue promotes aldosterone and angiotensin II, which cause kidney fibrosis. High blood pressure is the main early sign of filtration barrier damage and microalbuminuria; hyperglycemia and insulin resistance increase cellular toxicity, RAAS activation, and oxidative stress[15,18,21].

Clinical indicators: eGFR, albuminuria, CRP, HOMA-IR, waist circumference, glycemia, lipids.

AI/machine learning for risk stratification (e.g., KNN model with $\approx 80\%$ accuracy for predicting MS in patients with CKD)

This multimodal approach allows for early detection and targeted treatment, including RAAS blockers, MR antagonists, metabolic correction, and blood pressure control. Modern studies confirm that metabolic syndrome significantly increases the risk of developing and progressing chronic kidney disease. The combination of advanced genetic data (MR analyses), clinical markers, and machine learning offers a promising path for personalized prediction and early intervention. Reducing blood pressure, controlling glycemia, implementing nephroprotective strategies, and active monitoring will become key elements in the fight against CKD in patients with MS[22,23,24].

One of the largest and methodologically sound population studies confirming the link between metabolic syndrome and the development of chronic kidney disease is the Tehran Lipid and Glucose Study (TLGS) conducted in Iran. This prospective cohort study included 15,255 adult participants without initial chronic kidney disease, who were followed for over 11 years. The study diagnosed metabolic syndrome according to IDF criteria, determined estimated glomerular filtration rate (eGFR) using the CKD-EPI formula, and monitored the development of chronic kidney disease. Results showed that the presence of metabolic syndrome was associated with a 51% increased risk of developing CKD (HR = 1.51; 95% CI: 1.32-1.71). Particularly significant risk factors were hyperglycemia and arterial hypertension, and their combination with obesity increased the risk of renal dysfunction several-fold. The study emphasized the importance of early detection and modification of metabolic syndrome components to prevent decline in kidney function.

Additional insight into the causal relationships between metabolic disorders and CKD comes from an analysis using Mendelian Randomization (MR), published in 2024 by the research group of Guo Z. et al. This multi-center study utilized data from major international databases - UK Biobank, CKDGen, and FinnGen - covering over 400,000 individuals. The study demonstrated that genetically determined metabolic characteristics such as elevated systolic blood pressure and body mass index significantly increase the risk of developing CKD. Specifically, genetic predisposition to hypertension was associated with a 26% increase in CKD risk (OR = 1.26), and obesity with a 39% increase (OR = 1.39). A causal relationship between high triglyceride levels and renal dysfunction was also noted. These findings confirm that not only the phenotypic manifestations of metabolic syndrome, but also the underlying genetic factors play a significant role in the pathogenesis of renal pathology.

Conclusion

The development of CKD in patients with metabolic syndrome is due to the interaction of metabolic, inflammatory, and genetic factors. Identifying genetic markers of predisposition and using clinical and laboratory data in comprehensive risk assessment allows not only for predicting the course of the disease but also for timely initiation of individualized preventive and therapeutic interventions. Implementing clinical-genetic approaches in practical nephrology is a promising direction in modern medicine.

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