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S.V. Fedorov, N.R. Malinina, N.Z. Pozur
Ivano-Frankivsk National Medical University, Ivano-Frankivsk

FEATURES OF THE EFFECT OF TRIMETAZIDINE ON THE INDICATORS OF INFLAMMATION AND FIBROSIS IN PATIENTS WITH STABLE CORONARY ARTERY DISEASE WITH CONCOMITANT NON-ALCOHOLIC FATTY LIVER DISEASE

e-mail: m.natalia2511@gmail.com

The issue of finding ways to optimize the treatment of patients with stable coronary artery disease in combination with non-alcoholic fatty liver disease is very relevant today. The aim of this study was to evaluate the effectiveness of drug therapy for coronary artery disease in combination with non-alcoholic fatty liver disease using trimetazidine, taking into account the effect on components of the low-intensity systemic inflammatory response, as well as the progression of fibrosis and steatosis. The study involved patients with combined pathology who, depending on the treatment they received, were divided into two groups. All patients underwent anamnestic, clinical, anthropometric, instrumental (abdominal ultrasound), and laboratory tests (complete blood count with leukocyte indices, determination of levels of high-sensitivity C-reactive protein, matrix metalloproteinase-6, galectin-3, and monocyte chemotactic protein-1). Patients in the second group received trimetazidine for one month. The use of trimetazidine improved exercise tolerance, slowed the progression of fibrosis and steatosis, reduced body mass index, and reduced the intensity of the systemic inflammatory response.

Key words: stable coronary artery disease, non-alcoholic fatty liver disease, trimetazidine, inflammation, liver fibrosis.

С.В. Федоров, Н.Р. Малініна, Н.З. Позур

ОСОБЛИВОСТІ ВПЛИВУ ТРИМЕТАЗИДИНУ НА ПОКАЗНИКИ ЗАПАЛЕННЯ ТА ФІБРОЗУ В ХВОРИХ НА СТАБІЛЬНУ ІШЕМІЧНУ ХВОРОБУ СЕРЦЯ ПРИ СУПУТНІЙ НЕАЛКОГОЛЬНІЙ ЖИРОВІЙ ХВОРОБІ ПЕЧІНКИ

Питання пошуку шляхів оптимізації лікування пацієнтів із стабільною ішемічною хворобою серця у поєднанні з неалкогольною жировою хворобою печінки є на сьогоднішній день дуже актуальним. Метою даної роботи була оцінка ефективності медикаментозної терапії ішемічної хвороби серця у поєднанні з неалкогольною жировою хворобою печінки з використанням триметазидину, з урахуванням впливу на компоненти системної запальної відповіді низької інтенсивності, прогресування процесів фіброзу та стеатозу. У дослідженні взяли участь пацієнти з поєднаною патологією, які, залежно від отриманого лікування, були розподілені на дві групи. Усім пацієнтам проводили анамнестичні, клінічні, антропометричні, інструментальні (тредміл-тест, ультразвукове дослідження органів черевної порожнини) та лабораторні дослідження (загальний аналіз крові з обчисленням лейкоцитарних індексів, визначення рівнів високочутливого С-реактивного білка, матриксної металопротеїнази-6, галектину-3, моноцитарного хемотаксичного протеїну-1). Пацієнти першої групи отримували стандартизоване лікування. Пацієнти другої групи отримували триметазидин протягом одного місяця. Застосування триметазидину сприяло покращенню толерантності до фізичного навантаження, сповільненню процесів фіброзу та стеатозу, зменшенню індексу маси тіла та зниженню інтенсивності системної запальної відповіді.

Ключові слова: стабільна ішемічна хвороба серця, неалкогольна жирова хвороба печінки, триметазидин, запалення, фіброз печінки.

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Comorbidity is a significant concern in internal medicine, particularly when coexisting conditions have a considerable impact on patients' quality of life and life expectancy. Non-alcoholic fatty liver disease (NAFLD) is the most common chronic liver condition and is recognized as an important risk factor for the development of coronary artery disease (CAD) and cerebrovascular disorders. NAFLD is associated with subclinical cardiovascular alterations that may precede major cardiovascular events [4]. Liver imaging, elastography, and biochemical tests are employed to detect hepatic steatosis, particularly in relation to left ventricular function [2, 11].

Studies have demonstrated a strong association between hepatic steatosis and left ventricular abnormalities, as well as an increased risk of arterial hypertension [1]. Low-grade systemic inflammation, a hallmark of NAFLD, contributes to the development of atherosclerosis and its complications, such as CAD and hypertension. Chronic inflammation accompanying NAFLD is also linked to the progression of liver fibrosis—an essential stage in the development of hepatic failure [7].

Stable coronary artery disease (SCAD), driven by the progression of atherosclerosis in the coronary arteries, is commonly comorbid with NAFLD. In these patients, chronic systemic inflammation may lead to structural and functional changes in the left ventricle, further increasing the risk of cardiovascular complications. Low-grade systemic inflammation characteristic of NAFLD acts as a catalyst in the development of atherosclerosis, hypertension, type 2 diabetes mellitus, and other chronic diseases [5]. In

this context, inflammatory biomarkers such as high-sensitivity C-reactive protein (hs-CRP), matrix metalloproteinase-9 (MMP-9), galectin-3, and monocyte chemoattractant protein-1 (MCP-1) may serve as indicators of inflammatory activity and organ dysfunction.

NAFLD is accompanied by so-called "metabolic inflammation," which can cause cellular dysfunction and pathological tissue remodeling throughout the body [7]. This process affects not only the liver but also other organs, including the heart, promoting structural changes in the left ventricle in patients with SCAD, especially when NAFLD is present as a comorbid condition.

Trimetazidine, an antianginal agent, exerts certain effects on the cardiovascular system and is used for the symptomatic treatment of stable CAD. However, its efficacy extends beyond cardiovascular conditions; in recent years, it has demonstrated antifibrotic properties that may be beneficial in managing NAFLD. Additionally, trimetazidine improves lipogenesis and reduces oxidative stress—mechanisms potentially relevant for NAFLD patients—though this aspect requires further investigation [13].

The purpose of the study was to evaluate the effectiveness of pharmacological therapy for stable coronary artery disease combined with non-alcoholic fatty liver disease , with a focus on the impact of trimetazidine on components of low-grade systemic inflammatory response, as well as the progression of fibrosis and steatosis.

Materials and methods. This was a single-center, prospective study conducted from January 2022 to May 2024, in accordance with the principles of the Declaration of Helsinki and approved by the Ethics Committee of Ivano-Frankivsk National Medical University (IFNMU). A total of 63 patients with stable CAD (exertional angina, NYHA class II–III) and confirmed NAFLD were enrolled and divided into two groups: Group 1 (n=30) received standard guideline-recommended therapy (SGT), while Group 2 (n=33) received SGT in combination with trimetazidine at a dose of 35 mg (2 tablets twice daily) for one month. All patients were evaluated at three time points: baseline (inclusion visit), after 3 months, and after 6 months. Patient recruitment took place at the Therapy Department of the City Clinical Hospital No. 1 of the Ivano-Frankivsk City Council. Liver ultrasound with assessment of steatosis using controlled attenuation parameter (CAP) and fibrosis via the METAVIR scale was performed on a TOSHIBA APLIO 500 system (Toshiba, Japan). Biochemical and immunoassay analyses were carried out using the HTI BioChem FC-120 analyzer (HTI, USA).

Inclusion criteria were: confirmed diagnosis of stable CAD (exertional angina, NYHA class II–III), verified NAFLD, age ≥18 years, and signed informed consent.

Exclusion criteria included: uncontrolled comorbidities, established liver cirrhosis or other severe liver disorders not related to NAFLD, and known allergies or intolerance to the study medications.

All participants underwent a general clinical examination, including measurement of anthropometric parameters (notably body mass index, BMI). The degree of liver involvement was characterized using the METAVIR fibrosis score and calculation of the FIB-4 and NAFLD Fibrosis Score (NFS). The complete blood count with leukocyte differentiation was used to calculate systemic inflammation indices: NLR (neutrophil-to-lymphocyte ratio), LMR (lymphocyte-to-monocyte ratio), PLR (platelet-to-lymphocyte ratio), and SII (systemic immune-inflammation index) = (neutrophils × platelets) / lymphocytes. Inflammatory and fibrotic biomarkers analyzed included serum concentrations of high-sensitivity C-reactive protein (hs-CRP), matrix metalloproteinase-9 (MMP-9), galectin-3, and monocyte chemoattractant protein-1 (MCP-1), measured by ELISA using the HTI ImmunoChem-2100 analyzer (HTI, USA).

Statistical analysis was conducted using Python 3.11 with the pandas, SciPy.stats, and NumPy libraries. Normality of distribution was tested using the Shapiro-Wilk test. Due to non-normal distribution the Mann–Whitney U test was used for comparing quantitative variables, and the chi-square test for categorical variables. Differences were considered statistically significant at $p<0.05$.

Results of the study and their discussion. At the stage of comparing baseline clinical and demographic characteristics between groups, no statistically significant differences were observed (Table 1). The median age of patients receiving SGT was 56.50 years (50.25; 60.75), while in the SGT + TMZ group it was 54.00 years (49.00; 58.00) ($p = 0.335$). The duration of disease was also comparable between groups: 7.45 years (5.75; 8.88) in Group 1 versus 7.10 years (6.00; 8.50) in Group 2 ($p = 0.665$).

In terms of gender distribution, men predominated in the SGT group (66.7 %) compared to 60.6 % in the SGT + TMZ group ($p > 0.05$); however, this difference was not statistically significant. The proportion of women was 33.3 % and 39.4 %, respectively. A similar lack of significant differences was noted in the analysis of CAD subtype (stable CAD vs. post-infarction cardiosclerosis), the time elapsed after stenting, and the distribution of coronary artery involvement (LAD, Cx, RCA, or multivessel disease).

The body mass index (BMI) in both groups indicated the presence of overweight or obesity: 29.66 kg/m² (27.48; 33.67) in the SGT group and 31.91 kg/m² (28.36; 35.53) in the SGT + TMZ group (p = 0.28), with no statistically significant difference.

Table 1

Clinical and demographic characteristics of patients with stable coronary artery disease combined with non-alcoholic fatty liver disease

Parameter		SGT (n=30)	SGT + TMZ (n=33)	p-value
Age, years		56.50 (50.25; 60.75)	54.00 (49.00; 58.00)	0.335
Disease duration, years		7.45 (5.75; 8.88)	7.10 (6.00; 8.50)	0.665
Sex	– Male	20 (66.7 %)	20 (60.6 %)	>0,05
	– Female	10 (33.3 %)	13 (39.4 %)	
Type of CAD	– Stable CAD	15 (50.0 %)	20 (60.6 %)	>0.05
	–Post-infarction cardiosclerosis	15 (50.0 %)	13 (39.4 %)	
Time since stenting	– <1 year	13 (86.7 %)	10 (76.9 %)	>0.05
	– 1–3 years	2 (13.3 %)	3 (23.1 %)	
Affected artery	LAD	9 (60.0 %)	6 (46.2 %)	>0.05
	Cx	1 (6.7 %)	1 (7.7 %)	
	RCA	2 (13.3 %)	3 (23.1 %)	
	– Multivessel disease	3 (20.0 %)	3 (23.1 %)	
Body mass index, kg/m²		29.66 (27.48; 3367)	31.91 (28.36; 35.53)	0.28

The dynamics of systemic inflammation indices (NLR, LMR, SII, PLR) in both groups are presented in Table 2.

The most notable therapeutic effect was observed for the NLR. Baseline NLR levels were 2.73 (2.64; 2.85) in the SGT group compared to 2.79 (2.64; 2.90) in the SGT + TMZ group (p = 0.57). After 3 months, the NLR was significantly lower in the SGT + TMZ group: 2.50 (2.35; 2.55) versus 2.67 (2.50; 2.87) in the SGT group (p = 0.002). The relative reduction in NLR from baseline was more pronounced in the combination therapy group (–10.34 %) compared to a slight increase in the control group (+2.78 %). A similar trend was observed at 6 months, with the NLR remaining significantly lower in the SGT + TMZ group: 2.39 (2.30; 2.59) versus 2.55 (2.46; 2.72) in the SGT group (p = 0.006), corresponding to a –13.16 % reduction versus –6.73 %, respectively.

At baseline, LMR values were comparable: 2.79 (2.39; 3.62) in the SGT group versus 3.21 (2.33; 3.80) in the SGT + TMZ group (p = 0.312). Although there was a trend toward increasing LMR at each follow-up visit in the SGT + TMZ group, the differences between groups at 3 months (p = 0.054) and 6 months (p = 0.085) did not reach statistical significance. Nevertheless, both groups demonstrated a positive trend of increasing LMR, with a 13.04 % increase in the SGT group and an 11.07 % increase in the SGT + TMZ group at 6 months.

A trend toward a more pronounced reduction in the SII was observed in the SGT + TMZ group. At baseline, the difference between groups was not significant: 657.74 (622.24; 672.59) in the SGT group versus 658.35 (625.90; 698.63) in the SGT + TMZ group (p = 0.172). SII levels decreased in both groups at 3 and 6 months, with a more substantial reduction in the TMZ group (–5.03 % and –6.94 %, respectively), approaching statistical significance at 6 months (p = 0.058), while the reductions in the SGT group were less pronounced (–0.97 % and –2.56 %).

No significant differences were found between groups regarding changes in PLR over time (p > 0.05). Although PLR decreased slightly in both groups, the reductions were not statistically significant. After 6 months, mean PLR decreased by 7.83 % in the SGT group and by 9.03 % in the SGT + TMZ group, with the intergroup difference remaining statistically non-significant (p = 0.698).

An analysis of the dynamics of liver fibrosis and steatosis using various scoring systems (FIB-4, NFS, CAP, METAVIR) revealed favorable changes in patients who received additional trimetazidine therapy. At baseline, FIB-4 levels did not differ significantly between groups: 1.71 (1.55; 1.94) in the SGT group versus 1.78 (1.51; 1.91) in the SGT + TMZ group (p = 0.596). However, by the 3-month follow-up, FIB-4 values were significantly lower in the SGT + TMZ group: 1.58 (1.53; 1.62) compared to 1.61 (1.56; 1.67) in the control group (p = 0.019). This difference became even more pronounced after 6 months: 1.14 (1.07; 1.21) versus 1.32 (1.23; 1.38), respectively (p < 0.001).

Table 2

Dynamics of integrated leukocyte inflammatory indices in patients with stable coronary artery disease combined with non-alcoholic fatty liver disease under the influence of trimetazidine therapy

Parameter	Time	SGT (n=30)	SGT + TMZ (n=33)	p-value
NLR	t0	2.73 (2.64; 2.85)	2.79 (2.64; 2.90)	0.57
	t1	2.67 (2.50; 2.87)	2.50 (2.35; 2.55)	0.002
	Δ %, p t1	-2.78 %, p = 0.177	-10.34 %, p < 0.001	
	t2	2.55 (2.46; 2.72)	2.39 (2.30; 2.59)	0.006
	Δ %, p t2	-6.73 %, p = 0.002	-13.16 %, p < 0.001	
LMR	t0	2.79 (2.39; 3.62)	3.21 (2.33; 3.80)	0.312
	t1	2.99 (2.57; 3.22)	3.15 (2.81; 3.74)	0.054
	Δ %, p t1	-1.53 %, p = 1.000	-2.42 %, p = 0.525	
	t2	3.42 (2.85;3.88)	3.63 (3.44; 3.94)	0.085
	Δ %, p t2	13.04 %, p = 0.043	11.07 %, p = 0.043	
SII	t0	657.74 (622.24; 672.59)	658.35 (625.90; 698.63)	0.172
	t1	645.70 (627.02; 660.11)	630.38 (606.50; 656.71)	0.157
	Δ %, p t1	-0.97 %, p = 0.529	-5.03 %, p < 0.001	
	t2	630.30 (616.19; 649.06)	618.37 (598.77; 635.78)	0.058
	Δ %, p t2	-2.56 %, p = 0.055	-6.94 %, p < 0.001	
PLR	t0	194.60 (179.23; 225.56)	193.04 (166.67; 226.67)	0.999
	t1	199.14 (178.31; 222.65)	192.91 (180.62; 210.17)	0.588
	Δ %, p t1	-0.77 %, p = 0.839	-2.67 %, p = 0.646	
	t2	185.75 (170.06; 205.60)	186.67 (172.22; 200.00)	0.698
	Δ %, p t2	-7.83 %, p = 0.213	-9.03 %, p = 0.090	

Baseline NFS values were also comparable between groups: –0.53 (–0.75; –0.38) in the SGT group versus –0.50 (–0.80; –0.39) in the SGT + TMZ group (p = 0.404). During treatment, both groups showed a trend toward more negative values (indicating regression of fibrotic changes); however, improvements were more significant in the SGT + TMZ group. At 3 months, NFS was –0.73 (–0.79; –0.67) compared to –0.52 (–0.58; –0.46) in the control group (p < 0.001), and this difference persisted at 6 months: –0.89 (–0.94; –0.83) versus –0.75 (–0.80; –0.68) (p < 0.001).

Assessment of hepatic steatosis using CAP showed similar baseline values in both groups: 8.43 (7.61; 9.32) kPa in the SGT group versus 8.73 (8.27; 9.86) kPa in the SGT + TMZ group (p = 0.446). However, after 6 months, significant differences emerged in favor of the TMZ group: 8.18 (8.02; 8.33) kPa versus 8.40 (8.27; 8.52) kPa (p = 0.004). This trend continued at the final measurement: 8.00 (7.81; 8.15) kPa in the SGT + TMZ group versus 8.24 (8.01; 8.52) kPa in the control group (p = 0.002).

Using the METAVIR scale, no significant between-group differences were observed at baseline: 283.29 (277.88; 293.00) dB in the SGT group versus 279.38 (268.57; 293.17) dB in the SGT + TMZ group (p = 0.407). However, by 3 months, this difference became statistically significant: 273.34 (267.62; 281.26) dB versus 281.29 (275.69; 292.66) dB (p < 0.001), and remained significant at 6 months: 266.90 (260.71; 273.55) dB versus 274.91 (269.76; 278.84) dB (p = 0.031).

Baseline levels of hsCRP were nearly identical between groups: 2.89 (2.69; 3.18) mg/L in the SGT group versus 3.04 (2.63; 3.33) mg/L in the SGT + TMZ group (p = 0.749). However, after 3 months, a significantly lower hsCRP level was observed in the SGT + TMZ group: 2.40 (2.32; 2.58) mg/L compared to 2.87 (2.70; 3.09) mg/L in the control group (p < 0.001). A similar trend persisted at 6 months, with hsCRP further decreasing to 2.21 (2.08; 2.42) mg/L in the TMZ group versus 2.51 (2.40; 2.75) mg/L in the SGT group (p < 0.001).

Mean MMP-9 concentrations at baseline were also comparable: 1015.02 (905.71; 1092.13) ng/L in the SGT group versus 975.73 (916.23; 1103.24) ng/L in the SGT + TMZ group (p = 0.786). At 3 months, a significant decrease in MMP-9 was documented in the TMZ group: 889.35 (849.44; 942.08) ng/L versus 954.05 (930.00; 993.43) ng/L in the SGT group (p < 0.001). At 6 months, the difference between groups approached statistical significance (p = 0.06), with MMP-9 remaining lower in the TMZ group: 901.98 (864.55; 937.32) ng/L versus 937.61 (912.59; 965.65) ng/L, indicating a tendency toward a more pronounced reduction.

Galectin-3 plasma concentrations did not significantly differ at baseline: 26.06 (24.36; 28.46) ng/L in the SGT group versus 26.83 (23.79; 29.79) ng/L in the TMZ group (p = 0.4). After 3 months, a statistically significant decrease in galectin-3 was observed in the SGT + TMZ group: 23.91 (23.26; 25.59) ng/L versus 26.23 (25.18; 26.64) ng/L in the control group (p < 0.001). By 6 months, galectin-3 levels had declined in both groups, but the reduction was significantly more pronounced in the TMZ group (–15.84 % vs –6.12 %, p < 0.001).

Baseline MCP-1 levels were similar between groups: 345.70 (336.17; 363.42) ng/L in the SGT group versus 358.16 (341.68; 370.26) ng/L in the TMZ group (p = 0.094). At 3 months, the between-group difference was not statistically significant (p = 0.383), though a more noticeable reduction was seen in the TMZ group (–6.70 % vs –1.55 %). After 6 months, the difference became statistically significant: 313.95 (271.97; 322.96) ng/L in the TMZ group versus 326.72 (311.63; 348.27) ng/L in the SGT group (p = 0.003).

This study evaluated the effects of trimetazidine on systemic inflammation, fibrosis, and steatosis in patients with stable coronary artery disease coexisting with non-alcoholic fatty liver disease. The results demonstrated significant reductions in systemic inflammatory markers, such as NLR and SII, and improvements in liver fibrosis and steatosis indices in patients receiving trimetazidine therapy.

The reduction in NLR and SII indicates decreased neutrophil activation and overall systemic inflammation. This aligns with findings from other studies demonstrating that lowering systemic inflammation may improve prognosis in patients with coronary artery disease and NAFLD.

A decrease in NLR supports the critical role of neutrophils in the pathogenesis of inflammatory processes in these conditions, while the reduction in SII reflects improved inflammatory control, which is essential for reducing the risk of cardiovascular complications [3, 12].

Regarding hepatic fibrosis and steatosis, the decline in FIB-4 and NFS scores, as well as improvements in METAVIR grading, point to the beneficial impact of trimetazidine on structural liver changes. The assessment of steatosis via CAP revealed a significant decrease in values at 6 months in the TMZ group, confirming findings from previous studies demonstrating the efficacy of trimetazidine in the treatment of non-alcoholic steatohepatitis and in improving liver function parameters [8, 9].

The reduction of systemic inflammation and improvements in liver condition may have a substantial influence on patient outcomes by lowering the risk of cardiovascular events such as myocardial infarction, stroke, and heart failure. These changes are associated with enhanced quality of life, as the reduction in hepatic inflammation and fibrosis alleviates symptoms and contributes to overall clinical improvement. This corresponds with research showing that attenuation of inflammation and fibrosis can significantly mitigate disease symptoms and reduce the risk of complications [6, 10].

Conclusion

The tested therapeutic regimen demonstrated a beneficial effect of trimetazidine in slowing the progression of fibrosis and steatosis in patients with combined pathology. Beyond its established antianginal action, trimetazidine appears to exhibit multiple pleiotropic effects, including a potential capacity to attenuate low-grade systemic inflammatory responses and modulate components of the innate immune system. However, the mechanisms underlying these effects remain unclear and warrant further investigation.

Prospects for further research. Further studies should explore the relationship between levels of low-grade systemic inflammatory biomarkers and both modifiable and non-modifiable risk factors for atherogenesis progression in patients with stable coronary artery disease and concurrent NAFLD, as well as assess the effectiveness of targeted pharmacological interventions.

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S.R. Yusubova, S.T. Huseynova, U.I. Ahmedzade, S.F. Niftalieva
Azerbaijan Medical University, Baku, Azerbaijan

STRUCTURE AND PREVALENCE OF DENTAL DISEASES IN PATIENTS WITH EARLY STAGES OF CHRONIC KIDNEY DISEASE IN AZERBAIJAN

e-mail: mic_amu@mail.ru

The purpose of the study was to investigate the features of dental diseases and their distribution in patients with early stages of chronic kidney disease in Azerbaijan. The study involved 179 patients with early stages of chronic kidney disease. Decayed, Missing due to caries, Filled Teeth index, Caries, Fillings and Extractions index, Müllemann bleeding index, Svrakov iodine index, Community Periodontal Index and Gingival Index, Fuchs and Rumford indices were measured. The results showed that in patients with chronic kidney disease generalized periodontitis was present in 81.8 % of cases, and localized periodontitis in 18.2 % of cases, which is 5.4 and 1.7 times more than in the control group, respectively. In the early stages of chronic kidney disease, the prevalence of periodontitis is higher than caries and chronic periodontal diseases. An increase in the DMFT index was observed as kidney function decreased. Fuchs and Svrakov iodine indices were different during chronic kidney disease compared to the control group ($p<0.01$).

Key words: chronic kidney disease, periodontitis, caries, Decayed, Missing due to caries, Filled Teeth index, Svrakov iodine index, gingival index.

С.Р. Юсубова, С.Т. Гусейнова, У.І. Ахмедзаде, С.Ф. Ніфталієва

СТРУКТУРА І ПОШИРЕНІСТЬ СТОМАТОЛОГІЧНИХ ЗАХВОРЮВАНЬ У ХВОРИХ ІЗ РАННІМИ СТАДІЯМИ ХРОНІЧНОЇ ХВОРОБИ НИРОК В АЗЕРБАЙДЖАНІ

Метою дослідження було вивчення особливостей стоматологічних захворювань та їхнього поширення у хворих із ранніми стадіями хронічної хвороби нирок в Азербайджані. У дослідженні взяли участь 179 пацієнтів із ранніми стадіями хронічної хвороби нирок. Було виміряно індекс «Decayed, Missing due to caries, Filled Teeth index», індекс «Caries, Fillings and Extractions index», індекс кровотечі Мюллеманна, йодний індекс Свракова, пародонтальний індекс спільноти і ясенний індекс, індекси Фукса і Рамфорда. Результати засвідчили, що у пацієнтів із хронічною хворобою нирок генералізований пародонтит був присутній у 81,8 % випадків, а локалізований пародонтит – у 18,2 % випадків, що в 5,4 і 1,7 раза більше, ніж у контрольній групі відповідно. На ранніх стадіях хронічної хвороби нирок поширеність пародонтиту вища, ніж карієсу і хронічних захворювань пародонту. Збільшення індексу DMFT спостерігалося в міру зниження функції нирок. Індекси Фукса і Свракова за хронічної хвороби нирок відрізнялися порівняно з контрольною групою ($p<0,01$).

Ключові слова: хронічна хвороба нирок, пародонтит, карієс, індекс DMFT, йодний індекс Свракова, ясенний індекс.

Chronic kidney disease (CKD) is considered one of the major public health problems of our time [5]. Various forms of oral diseases are observed in 82 % of patients with CKD [6]. Among these diseases, the most common oral diseases are periodontitis and gingivitis. Patients with CKD undergoing hemodialysis are more susceptible to severe periodontal diseases [1].

The increasing number of people suffering from this disease and the limited research in this area require special attention to the study of oral manifestations associated with chronic kidney disease. Chronic kidney disease, like other systemic diseases, is associated with oral problems as a result of the pathogenesis of the disease or the therapy used, or both. The role of inflammatory factors in the progression of chronic kidney disease has been demonstrated, and periodontal lesions found in this group of patients have been associated with chronic oral inflammation [9, 11].

According to some studies, oral hygiene and chronic kidney disease are associated. Thus, in some studies, maintaining oral hygiene reduced the risk of developing chronic kidney disease [2]. In general,