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Hypertriglyceridemia in chronic kidney disease: prevalence and clinical spectrum

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Aim. To determine the prevalence of hypertriglyceridemia (HTG) and evaluate its association with markers of renal dysfunction in chronic kidney disease (CKD).
Material and methods. The study utilized clinical, laboratory, and instrumental data from 562 patients with non-diabetic CKD. The mean age of patients was 37.7 years (age range 17 to 72 years). Anthropometric, hemodynamic, and laboratory parameters were assessed. Patients were divided into two groups: Group 1 — women (n=185), Group 2 — men (n=377). A blood triglyceride (TG) concentration of ⩾ 1.7 mmol/L was considered as HTG.
Results. In the overall cohort of CKD patients, the prevalence of HTG was 55.3%. The frequency of HTG was higher at CKD stages 3B (72.00%), 3A (67.20%), 2 (60.90%), and 1 (57.50%) compared to stages 4 (49.20%) and 5 (39.50%). Levels of systolic, diastolic, pulse, and mean arterial pressure (MAP), as well as 24-hour proteinuria, were significantly higher, while high-density lipoprotein cholesterol (HDL-C) levels were significantly lower in men than in women. Lower values of peripheral blood laboratory parameters (hemoglobin, red blood cells, platelets) and nitrogen excretion function of the kidneys were characteristic of females. A strong correlation was found between estimated glomerular filtration rate (eGFR) and risk factors for chronic renal failure in both female and male patients. In the female CKD group, eGFR significantly correlated with age, systolic, diastolic and mean arterial pressure, hemoglobin (Hb) concentration, red blood cell and platelet counts. In men with CKD, eGFR significantly correlated with age, systolic, diastolic, pulse and mean arterial pressure, Hb concentration, red blood cell and platelet counts, as well as serum total cholesterol, HDL-C, and TG levels. In the overall patient cohort, a statistically significant relationship was observed between serum TG concentration and both eGFR and 24-hour proteinuria.
Conclusion. In patients with CKD, HTG is more common at the stage of renal insufficiency and is closely associated with markers of renal dysfunction. The association of HTG with cardiovascular risk factors is more pronounced in men. These findings highlight the need for early screening of triglyceride levels for timely correction of metabolic disorders and slowing the progression of chronic kidney disease. A differentiated approach to lipid profile assessment in men and women will allow for more accurate prediction of disease course and optimization of preventive therapy strategies.
Keywords: chronic kidney disease, hypertriglyceridemia, risk factors, chronic renal failure, prognosis, healthcare.

Conflict of Interest: none declared.

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Introduction

Patients with chronic kidney disease (CKD) belong to the high and very high cardiovascular risk (CVR) groups. The study of the role of non-immune factors in the development of cardiovascular disorders and the progression of CKD represents an important area of modern preventive medicine [1, 2]. A blood triglyceride (TG) concentration of ≥1.7 mmol/L is defined as hypertriglyceridemia (HTG) [3], which is widespread among patients with CKD. According to research data, HTG is accompanied by decreased lipoprotein clearance, increased concentrations of very low-density lipoproteins (VLDL), and accumulation of intermediate-density lipoproteins [3, 4]. Possible mechanisms of renal HTG include changes in enzyme expression and/or activity, impaired receptor apparatus function, and reduced high-density lipoprotein metabolism [5]. At the stage of chronic renal failure (CRF), insulin resistance is observed, which contributes to increased VLDL production in the liver and partially explains the development of HTG [3, 5]. Additionally, decreased lipase activity leads to impaired breakdown of TGs into free fatty acids necessary for the body's energy supply, which may also contribute to increased CVR.

Numerous studies have shown that HTG, in combination with other CVR factors, contributes to an increased frequency of cardiovascular events and the progression of CRF [6, 7]. Elevated blood TG concentrations in CKD, especially at the CRF stage, have specific features compared to the general population. For example, within the ARIC (Atherosclerosis Risk in Communities) registry, it was established that an increase in TG levels is accompanied by the progression of renal dysfunction [8]. Subsequent studies confirmed the contribution of HTG to the development of coronary artery disease (CAD), which is common in patients with CKD [9]. In one previously published study, it was shown that at TG concentrations >1.6 mmol/L, the incidence of CAD reaches 11.5% [10]. According to a meta-analysis by foreign colleagues, HTG is considered an independent risk factor (RF) for CAD: an increase in TG levels by 1.0 mmol/L is associated with a 32% increase in the risk of cardiovascular disease (CVD) in men and 76% in women [11]. Studies from the last five years have also demonstrated a link between elevated TG concentrations and a number of other pathologies, including arterial hypertension (AH), preeclampsia, cerebral ischemia, and acute pancreatitis [12].

Aim of the study. To determine the frequency of HTG and assess its relationship with markers of renal dysfunction in CKD.

Materials and methods

The study used clinical, laboratory, and instrumental data from patients with CKD due to chronic glomerulonephritis (without morphological verification) who underwent inpatient treatment in the nephrology department of the National Center of Cardiology and Therapy named after Academician Mirsaid Mirrakhimov between September 2008 and September 2016. This study is an observational, cross-sectional (retrospective) study conducted in accordance with the principles of the Declaration of Helsinki. All patients signed informed voluntary consent to participate. Inclusion criterion: presence of glomerular and tubulointerstitial kidney diseases.

Exclusion criteria: type 1 and type 2 diabetes mellitus (DM), AH, heart failure of functional class III–IV, hypothyroidism, systemic connective tissue diseases, gout, polycystic ovary syndrome, pregnancy and lactation, as well as patients receiving scheduled hemodialysis.

The analysis included 562 patients with an established diagnosis of chronic kidney disease. The mean age of the participants was 37.7 ± 12.7 years (range: 17 to 72 years).

Anthropometric parameters were recorded for all patients: height, body weight, and body mass index (BMI), with the determination of overweight (OW) and the degree of obesity. Systolic and diastolic blood pressure (SBP and DBP) were measured, mean and pulse pressure were calculated, and heart rate (HR) was recorded.

The laboratory part of the study included determination of hemoglobin (Hb) levels, erythrocyte and platelet counts, and erythrocyte sedimentation rate (ESR). Lipid profile analysis included determination of total cholesterol (TC), TGs, low-density lipoprotein cholesterol (LDL-C), and high-density lipoprotein cholesterol (HDL-C). Additionally, venous blood creatinine and glucose concentrations were measured.

The nitrogen-excretory function of the kidneys was assessed by estimated glomerular filtration rate (eGFR), calculated using the CKD-EPI (Chronic Kidney Disease Epidemiology Collaboration) formula. Daily proteinuria was also measured in all patients.

In accordance with Russian and international clinical guidelines, the following CVR factors were identified: OW (BMI 25.0–29.9 kg/m²); grade I obesity (BMI 30.0–34.9 kg/m²); grade II obesity (BMI 35.0–39.9 kg/m²); grade III obesity (BMI ≥40 kg/m²) [13]; systolic AH (SBP ≥140 mm Hg); diastolic AH (diastolic BP ≥90 mm Hg); tachycardia (resting HR ≥80 beats/min) [14]; renal failure (eGFR <60 ml/min); nephrotic proteinuria (daily proteinuria >3.5 g); anemia (Hb <120 g/L in women and <130 g/L in men); HTG (TG ≥1.7 mmol/L); hypercholesterolemia (TC >5.01 mmol/L) [1]. CKD stages were determined according to the recommendations of the Scientific Society of Nephrologists of Russia, published in 2021 [15].

The examined patients with CKD were divided into two groups: group 1 — women (n=185); group 2 — men (n=377). The groups were comparable in age: 38.7 ± 11.9 years and 37.2 ± 13.0 years, respectively (p=0.166).

Statistical analysis

Statistical data processing was performed using Microsoft Excel spreadsheets and the Statistica 10.0 software package. Qualitative characteristics are presented as absolute and relative values (n, %). Continuous variables were described as median (Me) and interquartile range [Q25; Q75] for non-parametric distribution, or as mean (M) and standard deviation (SD) for normal distribution.

Student's t-test was used to compare quantitative indicators between the two groups. The Mann–Whitney U-test was used for independent samples. Correlation analysis was performed using Pearson's correlation coefficient to assess relationships. The critical significance level for testing the null hypothesis was set at $p < 0.05$.

Results

According to the analysis results (Fig. 1), the majority of examined patients were at early stages of CKD: C1 (n=146) and C2 (n=110). The number of patients at later stages was: C3A — 58, C3B — 50, C4 — 56, and C5 — 129 individuals.

The number of women and men at CKD stages was: C1 — 31 and 115; C2 — 42 and 68; C3A — 24 and 34; C3B — 16 and 34; C4 — 24 and 45; C5 — 48 and 81, respectively. Among the examined patients, signs of CRF, i.e., a decrease in eGFR <60 ml/min, were observed in 306 (54.4%) individuals.

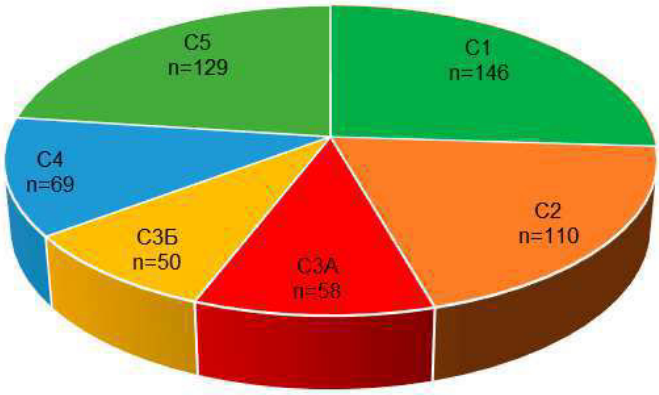

Fig. 1. Distribution of the examined patients with CKD

Table 1 presents the indicators of nitrogen-excretory renal function (median and interquartile range), daily proteinuria, and blood lipid spectrum parameters. According to these data, the examined patients had an initial stage of CRF (serum creatinine — 131.0 μmol/L, eGFR — 52.7 ml/min).

Proteinuria was characterized by a predominance of nephrotic variants of CKD (2.256 g/day). Blood lipid spectrum levels corresponded to the criteria for hyperlipidemia and dyslipidemia.

Table 1. **Characteristics of renal function and blood lipid spectrum in the examined patients with CKD, n=562**

Parameters	Me (Q25; Q75)
Serum creatinine, μmol/L	131.0 (87,0; 347,0)
eGFR, ml/min	52.7 (17.0; 91.5)
Daily proteinuria, g/day	2.256 (0.859; 4.817)
TC, mmol/L	5.27 (4.10; 6.70)
TG, mmol/L	1.81 (1.14; 2.57)
LDL-C, mmol/L	3.19 (2.35; 4.54)
HDL-C, mmol/L	1.00 (0.80; 1.30)

In accordance with the study objectives, a comparative analysis of clinical and laboratory data was performed (Table 2). The patient groups did not differ significantly in age, BMI, heart rate, fasting venous blood glucose, TC, LDL-C, TG, or creatinine levels.

In men, the values of systolic (144 ± 27 mm Hg vs. 137 ± 25 mm Hg, p=0.001), diastolic (91 ± 15 mm Hg vs. 88 ± 16 mm Hg, p=0.014), pulse (53 ± 17 mm Hg vs. 49 ± 17 mm Hg, p=0.001), and mean (48 ± 9 mm Hg vs. 45 ± 8 mm Hg, p=0.001) blood pressure, as well as daily proteinuria (2.542 [0.871; 5.566] g vs. 1.947 [0.781; 3.958] g, p=0.033), were statistically significantly higher, while HDL-C levels (1.04 ± 0.42 mmol/L

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Table 2. **Comparative characteristics of patients included in the study, n=562**

Parameter	Women with CKD, n=185	Men with CKD, n=377	p
Mean age, years	38.7±11.9	37.2±13.0	0.166
BMI, kg/m²	26.6±6.0	25.9±6.1	0.182
SBP, mm Hg	137±25	144±27	0.001
DBP, mm Hg	88±16	91±15	0.014
Pulse pressure, mm Hg	49±17	53±17	0.001
Mean BP, mm Hg	45±8	48±9	0.001
HR, beats/min	77±10	76±9	0.291
Hemoglobin, g/L	116.3±25.1	133.5±28.2	0.001
Erythrocytes, ×10¹²/L	4.03±0.62	4.39±0.64	0.001
Platelets, ×10⁹/L	233.6±25.9	245.1±25.0	0.001
ESR, mm/h	16.0 (10.0;28.0)	12.0 (5.0;24.5)	0.001
Glucose, mmol/L	4.76±0.78	4.71±1.08	0.642
TC, mmol/L	5.31 (4.24;6.42)	5.23 (4.07;6.80)	0.796
HDL-C, mmol/L	1.18±0.43	1.04±0.42	0.001
LDL-C, mmol/L	3.19 (2.42;4.27)	3.19 (2.32;4.63)	0.661
TG, mmol/L	1.82 (1.18;2.25)	1.78 (1.14;2.62)	0.383
Creatinine, μmol/L	129.5 (82.0;341.0)	131.6 (88.0;347.0)	0.309
eGFR, ml/min	46.5 (14.3;80.9)	57.5 (17.9;96.4)	0.013
Proteinuria, g/day	1.947 (0.781;3.958)	2.542 (0.871;5.566)	0.033

vs. 1.18 ± 0.43 mmol/L, p=0.001) were significantly lower compared to women.

In women, on the contrary, lower values of Hb (116.3 ± 25.1 g/L vs. 133.5 ± 28.2 g/L, p=0.001), erythrocyte count (4.03 ± 0.62 × 10^{12}/L vs. 4.39 ± 0.64 × 10^{12}/L, p=0.001), platelet count (233.6 ± 25.9 × 10^9/L vs. 245.1 ± 25.0 × 10^9/L, p=0.001), and eGFR (46.5 [14.3; 80.9] ml/min vs. 57.5 [17.9; 96.4] ml/min, p=0.013) were observed. At the same time, the ESR in women was statistically significantly higher (16.0 [10.0; 28.0] mm/h vs. 12.0 [5.0; 24.5] mm/h, p=0.001) compared to men with CKD.

A comparative analysis of CKD-associated risk factors for renal failure in women and men showed that the frequency of overweight (28.6% and 31.2%), grade I obesity (12.4% and 13.7%), grade II obesity (8.1% and 4.2%), grade III obesity (4.3% and 1.3%), diastolic hypertension (49.1% and 58.6%), tachycardia (34.5% and 26.2%), hypercholesterolemia (57.2% and 53.0%), HTG (58.3% and 53.0%), and nephrotic proteinuria (29.7% and 39.2%) did not differ significantly between the groups (Table 3).

As expected, the prevalence of anemia (51.3% and 38.9%, p<0.05) and CRF (83.2% and 69.4%, p<0.05) was statistically significantly higher in women compared to men. Overall, among the 562 patients with CKD, HTG was present in 311 individuals, accounting for 55.3% of cases.

Figure 2 presents the frequency of HTG in patients with CKD depending on the stage of the disease. The

Table 3. **Characteristics of risk factors in the examined patients, n=562**

Parameter	Women with CKD, n=185	Men with CKD, n=377
Overweight, n	53 (28.6%)	118 (31.2%)
Grade I obesity, n	23 (12.4%)	52 (13.7%)
Grade II obesity, n	15 (8.1%)	16 (4.2%)
Grade III obesity, n	8 (4.3%)	5 (1.3%)
Systolic hypertension, n	90 (48.6%)	226 (59.9%)*
Diastolic hypertension, n	91 (49.1%)	221 (58.6%)
Increased HR, beats/min, n	64 (34.5%)	99 (26.2%)
Anemia, n	95 (51.3%)*	147 (38.9%)
Hypercholesterolemia, n	106 (57.2%)	200 (53.0%)
HTG, n	108 (58.3%)	203 (53.8%)
CRF, n	154 (83.2%)*	262 (69.4%)
Nephrotic proteinuria, n	55 (29.7%)	148 (39.2%)

Note. * — p < 0.05 (statistically significant differences between men and women).

frequency of HTG at stages C3B (72%), C3A (67.2%), C2 (60.9%), and C1 (57.5%) was higher than at the later C4 and C5 stages (49.2% and 39.5%, respectively).

Additionally, blood TG concentrations in patients with CKD were assessed depending on the disease stage (Fig. 3). Statistically significantly higher TG levels were observed at stages C3A (2.20 [1.45; 3.05] mmol/L) and C3B (2.01 [1.52; 2.63] mmol/L) compared to stages C1 (1.84 [1.12; 2.62] mmol/L), C2 (1.91 [1.15; 2.82] mmol/L), C4 (1.60 [1.08; 2.57] mmol/L), and C5 (1.35 [0.89; 1.89] mmol/L).

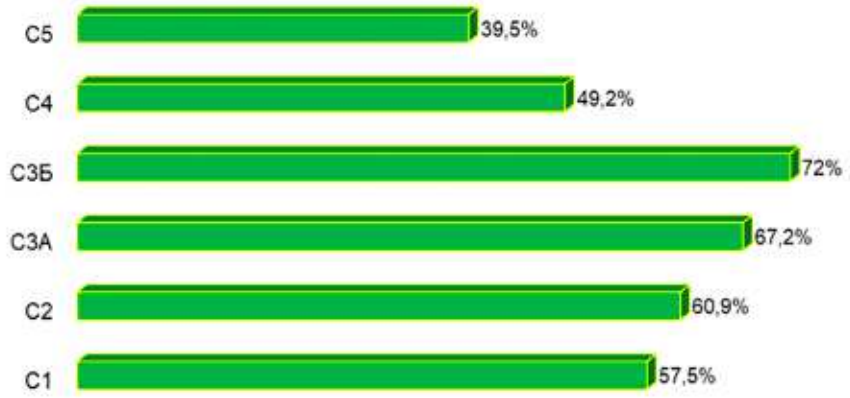

Fig. 2. Frequency of HTG in patients with CKD

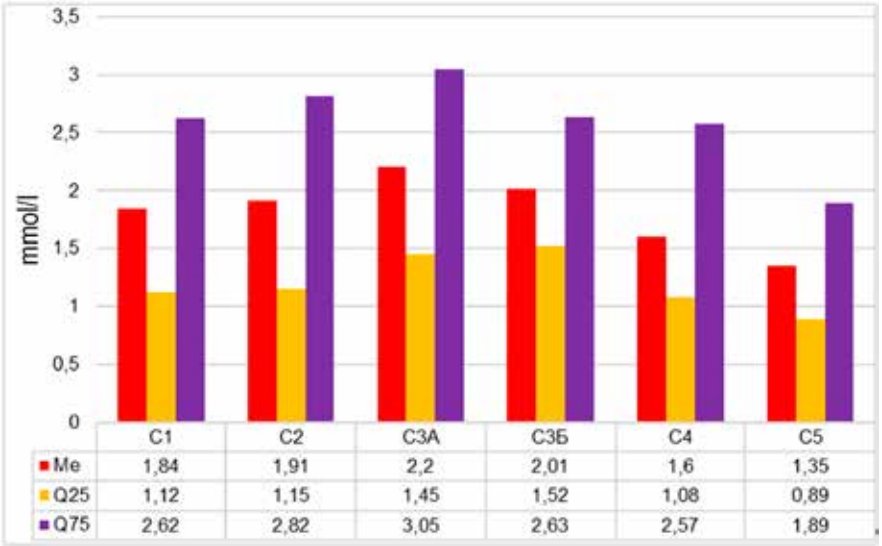

Fig. 3. Blood TG concentration in patients with CKD

Table 4. **Correlation of eGFR with clinical and laboratory data in the examined patients with CKD**

Parameter	Women with CKD, n=185		Men with CKD, n=377	
	eGFR, ml/min			
	r	p	r	p
Mean age, years	0.464	0.001	0.305	0.001
BMI, kg/m²	0.037	0.717	0.073	0.239
SBP, mm Hg	0.223	0.023	0.579	0.001
DBP, mm Hg	0.253	0.010	0.429	0.001
Pulse pressure, mm Hg	0.153	0.171	0.547	0.001
Mean BP, mm Hg	0.223	0.023	0.579	0.001
HR, beats/min	0.024	0.806	0.032	0.607
Hemoglobin, g/L	-0.46	0.001	-0.570	0.001
Erythrocytes, ×10¹²/L	-0.451	0.001	-0.567	0.001
Platelets, x109/L	-0.265	0.001	-0.446	0.001
ESR, mm/h	0.123	0.227	0.103	0.150
Glucose, mmol/L	0.074	0.464	0.119	0.096
TC, mmol/L	0.126	0.213	0.246	0.001
HDL-C, mmol/L	-0.171	0.092	-0.297	0.001
LDL-C, mmol/L	0.131	0.198	0.058	0.443
TG, mmol/L	0.143	0.147	0.103	0.046

Table 4 presents the results of the correlation analysis of eGFR with risk factors for CRF in women and men.

In the group of women with CKD, eGFR correlated significantly with age (r = 0.464; p = 0.001), systolic (r = 0.223; p = 0.023), diastolic (r = 0.253; p = 0.010), and mean (r = 0.223; p = 0.023) blood pressure, Hb concentration (r = 0.460; p = 0.001), erythrocyte count (r = 0.451; p = 0.001), and platelet count (r = 0.265; p = 0.001).

In men with CKD, eGFR also correlated significantly with age (r = 0.305; p = 0.001), systolic (r = 0.579; p = 0.001), diastolic (r = 0.429; p = 0.001), pulse (r = 0.547; p = 0.001), and mean (r = 0.579; p = 0.001) blood pressure, Hb concentration (r = 0.570; p = 0.001), erythrocyte count (r = 0.567; p = 0.001), and platelet count (r = 0.446; p = 0.001), as well as with TC (r = 0.246; p = 0.001), HDL-C (r = 0.297; p = 0.001), and TG (r = 0.103; p = 0.046).

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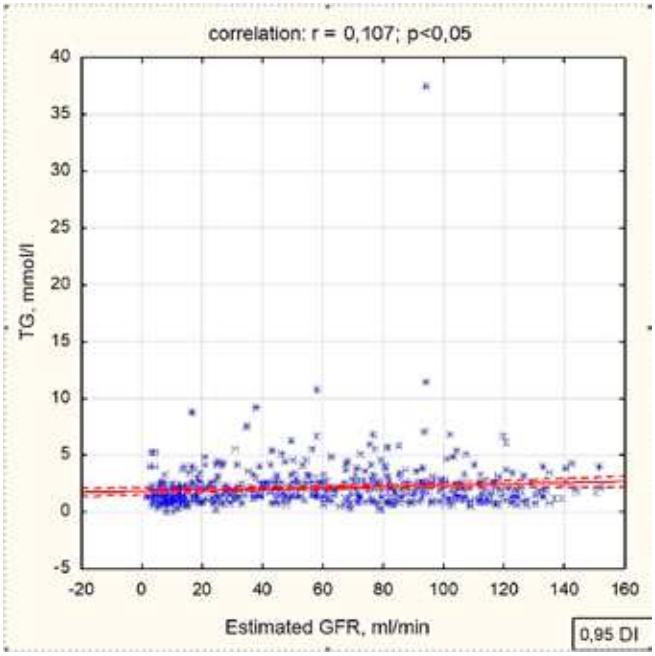

Fig. 4. Correlation of blood triglyceride concentration with eGFR in patients with CKD in the overall group

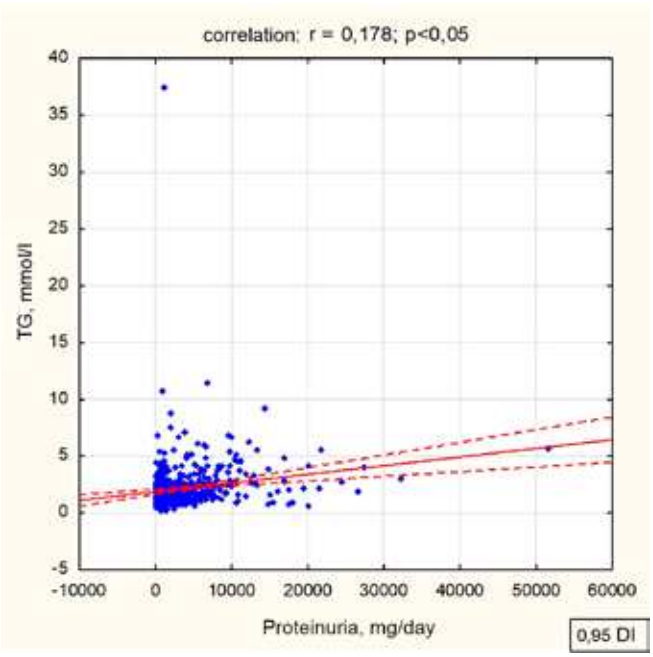

Fig. 5. Correlation of blood TG concentration with daily proteinuria in patients with CKD in the overall group

Given the absence of a statistically significant correlation between TG concentration and eGFR in the group of women, an additional assessment of the relationship between these parameters was performed in the overall sample. According to the data presented in Figures 4 and 5, TG concentration was statistically significantly correlated with eGFR ($r = 0.17$; $p < 0.05$) and daily proteinuria ($r = 0.178$; $p < 0.05$).

Discussion

Hypertriglyceridemia is frequently detected in patients with CKD, manifesting as an elevation of serum TG levels above reference values and associated with increased cardiovascular risk [15, 16]. Nephrogenic HTG is secondary in nature and is considered an indicator of the activity of the pathological process in the kidneys. Blood TG concentration particularly increases in the presence of nephrotic proteinuria and dysproteinemia, which are characteristic of early stages of CKD. In our study, the majority of patients were at the initial stage of CKD and had signs of early CRF.

The mechanisms of HTG formation in patients with CKD, especially in proteinuric variants, are complex and diverse. According to the literature, enhanced mobilization of free fatty acids from peripheral adipose tissue to the liver leads to increased concentrations of LDL and TG-enriched lipoproteins [17]. Additionally, in CKD, there is a decrease in lipoprotein lipase activity, a key enzyme responsible for the hydrolysis of TG-containing lipoproteins. As a result, patients with CKD and concomitant HTG exhibit reduced HDL-C levels and elevated LDL-C levels [15]. As shown in the present study, men with CKD were characterized by significantly higher blood pressure values, daily proteinuria, and lower HDL-C levels.

According to the literature, in the general population, blood TG levels are higher in men than in women, especially under the age of 70 [16]. The prevalence of HTG in the general population exceeds 10%, which is comparable to the frequency of CKD and type 2 diabetes mellitus [18]. In one study, the frequency of HTG in individuals with hypertension was 34.34% [19]. According to the NHANES (3rd National Health and Nutrition Examination Survey) registry, the prevalence of HTG among patients with CVD reaches 62% [20]. Previously published studies have noted that the frequency of HTG is higher among Mexican Americans compared to White Americans, while it was significantly lower in African Americans [21]. In our study, the prevalence of HTG among patients with CKD was 55.3%. According to other data, the frequency of HTG among individuals with CVD reaches 62% in Europe and 29.2% in the Russian Federation [22]. In our study, a high prevalence of HTG was identified at CKD stages C3B (72%), C3A (67.2%), C2 (60.9%), and C1 (57.5%). The median TG concentration was signifi-

cantly higher in patients with stage C3 CKD. It should be noted that the median and interquartile TG levels did not differ significantly between women and men.

With age, the frequency of HTG increases, reaching a maximum at 55–65 years, which is explained by the age-related increase in the incidence of type 2 diabetes mellitus, CKD, and CAD. In the present study, the median blood TG level was 1.81 mmol/L. It is known that in the general population, HTG is associated with hypertension, hypercholesterolemia, and atherogenic dyslipidemia. These data are fully reflected in our study. The results of a recent study allow us to identify a causal relationship between changes in blood lipid spectrum parameters and increased blood pressure [23]. In this regard, it can be assumed that elevated blood pressure is closely related to HTG, and their combination contributes to the development of renal dysfunction.

At the molecular-cellular level, elevated TG concentrations are associated with key manifestations of endothelial dysfunction, including impaired vasodilation, enhanced production of chemokines and cytokines, activation of the inflammatory response, and monocyte infiltration in renal tissue [24, 25]. These processes contribute to increased proteinuria and increase the risk of CRF progression. In our study group, the median values of serum creatinine and eGFR corresponded to the criteria for the initial stage of CRF. According to the literature, in CKD, the frequency of HTG increases as nitrogen-excretory renal function declines [26, 27]. In our previously published studies, we demonstrated a high prevalence of HTG among individuals with various cardiovascular risk categories [28], as well as an association between TG concentration and risk factors for CRF in patients with comorbid pathology [29].

According to the data of the present study, HTG is associated with the progression of CRF in CKD, especially in men. This is confirmed by a statistically significant correlation between elevated blood TG concentrations and decreased eGFR. It is likely that the presence of HTG in CKD exerts a synergistic effect on cardiovascular risk factors, as several studies have shown that the combination of elevated blood pressure with HTG increases the risk of all-cause mortality by 2.8 times [30].

On the other hand, the involvement of peripheral blood platelets in atherogenesis is an established fact. Under conditions of HTG, platelet aggregation and thrombus formation are enhanced [31], which potentiates the thrombotic component of atherogenesis and may contribute to the progression of vascular and renal complications. In this regard, the statistically significant association between platelet count and eGFR in both women and men, demonstrated in our study, logically fits into the pathophysiological model where platelet activation in the context of HTG contributes to a greater decline in eGFR through microvascular mechanisms.

The active involvement of TG-rich particles in atherosclerotic processes is explained by their ability to be readily taken up by macrophages migrating into the vascular wall [32]. TG accumulation in macrophages enhances the synthesis of reactive oxygen species, contributing to foam cell formation [33] and accelerating the development of atherosclerotic changes. Additionally, under conditions of HTG, increased vascular endothelial permeability is observed [34], making it more vulnerable to damage. When HTG is combined with anemia, hypertension, or electrolyte imbalance, the risk of endothelial cell damage and death increases significantly, which is accompanied by the progression of endothelial dysfunction, the development of CRF, and an increased frequency of cardiovascular events.

It should be emphasized that HTG in CKD has dual pathobiological significance: on the one hand, it contributes to the development of renal dysfunction and the progression of CRF, and on the other hand, it creates prerequisites for the occurrence of cardiovascular events [35]. The results of the correlation analysis presented in Figures 4 and 5 confirm the role of HTG in the development of renal dysfunction and the progression of CRF. In a prospective cohort study by Russian colleagues, it was shown that the presence of HTG increases the relative risk of cardiovascular mortality by 2.15 times. The authors note that HTG was found to be a significant risk factor for mortality exclusively in women [30].

Summarizing the accumulated research findings, it should be emphasized that a TG level >2.3 mmol/L is considered a risk factor for the development of CVD, for which pharmacological therapy is recommended [36, 37]. For patients with CVD and CKD, a TG level of < 1.7 mmol/L is considered the target, reflecting the need for strict lipid profile control in this group. These clinical benchmarks confirm that HTG acts not only as an independent risk factor but also as an element of a complex pathogenetic system that in-

teracts with other unfavorable factors. It follows that there is a close clinical and pathogenetic interrelationship between the studied cardiovascular risk factors, which represent not a simple sum of individual effects but possess a synergistic, mutually reinforcing impact on the progression of CRF and cardiovascular complications.

Conclusion

The results of the present study demonstrate a high prevalence of HTG in CKD at early stages of CRF. In both women and men with CKD, HTG is associated with hypertension, tachycardia, anemia, and reduced nitrogen-excretory renal function. The identified associations of HTG with cardiovascular risk factors and renal dysfunction underscore the need for early assessment of blood TG levels in patients with CKD, which will allow timely enhancement of therapeutic and preventive measures at the outpatient stage.

Thus, the obtained data confirm that HTG in CKD, especially in men, should be considered a priority target for the prevention of cardiovascular events and improvement of prognosis in this patient population.

Conflict of Interest: none declared.

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