

**Lipid Composition Features in Elderly Patients with Ischemic Heart Disease and Chronic Kidney Disease**

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**Abstract:** As chronic kidney disease (CKD) progresses and the glomerular filtration rate (GFR) declines, lipid metabolism parameters deteriorate: levels of low-density lipoproteins (LDL), triglycerides, and the atherogenic index increase, while high-density lipoprotein (HDL) levels decrease. These changes emphasize the need for lipid profile monitoring in CKD patients, as dyslipidemia is a significant risk factor for cardiovascular complications. CKD treatment should include the correction of lipid metabolism disorders, particularly in patients with low GFR, to reduce the risk of atherosclerotic complications.

**Keywords:** ischemic heart disease (IHD), chronic kidney disease (CKD), blood lipids, urea, creatinine, GFR, cardiovascular diseases, elderly patients.

**Introduction:** The diagnosis and treatment of chronic ischemic heart disease (IHD) are well established, but managing patients with comorbid conditions often presents challenges. Among the associated diseases, chronic kidney disease (CKD) is one of the most prevalent and is considered a major risk factor for cardiovascular diseases. Mortality in patients with renal dysfunction is more often caused by IHD than by end-stage renal disease (ESRD). Studies indicate a high prevalence of renal dysfunction in IHD patients. For example, in stable IHD, the estimated GFR was below 75 mL/min/1.73 m<sup>2</sup> in 52% of cases according to the EUROPA study. In elderly patients over 65 years old with non-terminal kidney dysfunction, atherosclerotic heart disease (42.5% vs. 16.5%) and myocardial infarction (10% vs. 2%) were more frequent compared to those with normal kidney function, according to the USRDS registry. Coronary angiography results showed that three-vessel coronary artery disease (>50% stenosis) was observed in 53% of patients with moderate to severe renal dysfunction and in 28% of those with mild or normal renal function.

**Objective:** To study the lipid composition of the blood in elderly patients with IHD and CKD.

**Materials and Methods**

The study included 115 elderly patients (aged 60 and older) diagnosed with IHD, undergoing treatment at the cardiology department of City Clinical Hospital No. 7 in Tashkent. The duration of IHD was over five years, and CKD over three years. The study classified CKD according to KDIGO (2012), the Russian Nephrology Society's guidelines (2020), and the European Renal Association (ERA-EDTA, 2021). CKD was diagnosed based on the following criteria: GFR < 60 mL/min/1.73 m<sup>2</sup> for ≥3 months, albuminuria ≥30 mg/day, structural kidney changes on ultrasound, elevated serum creatinine and urea, and altered urine albumin/creatinine ratio. Patients were divided into two groups: Group 1 (n=65) – IHD patients with CKD of varying severity, and Group 2 (n=50) – IHD patients without CKD. The study analyzed lipid profiles (LDL, HDL, total cholesterol, triglycerides, atherogenic index), serum urea, creatinine, and GFR.

The Student's t-test was used for statistical analysis, with significance set at p<0.05. Pearson's correlation coefficient was used to analyze relationships between variables.

## Results

The blood lipid profile was assessed in all study patients to identify dyslipidemia and its impact on the development and progression of CKD in IHD patients. The analysis of lipid metabolism in the two groups revealed significant differences in atherogenic indicators, except for total cholesterol (Table 1)

**Table №1**  
**Blood lipid parameters**

Parameters	Group 1	Group 2	p
<b>Total cholesterol (mmol/l)</b>	6,2 ± 1,2 (95% ДИ: 5,9 – 6,5)	5,9 ± 1,1 (95% ДИ: 5,6 – 6,2)	>0,05
<b>LDL (mmol/l)</b>	4,1 ± 1,0 (95% ДИ: 3,9 – 4,3)	3,5 ± 0,9 (95% ДИ: 3,3 – 3,7)	<0,05

Total cholesterol in Group 1 was  $6.2 \pm 1.2$  mmol/L, while in Group 2, it was  $5.9 \pm 1.1$  mmol/L. The differences between the groups did not reach statistical significance ( $p > 0.05$ ), which may be due to the high variability of this parameter in CKD patients. LDL concentration was significantly higher in patients with both IHD and CKD ( $4.1 \pm 1.0$  mmol/L) compared to Group 2 ( $3.5 \pm 0.9$  mmol/L,  $p < 0.05$ ). This confirms more pronounced atherogenic changes in patients with comorbid conditions. HDL levels were significantly lower in Group 1 ( $0.9 \pm 0.2$  mmol/L) compared to Group 2 ( $1.1 \pm 0.2$  mmol/L,  $p < 0.05$ ), indicating reduced anti-atherogenic protection in CKD patients, which increases the risk of cardiovascular complications.

Triglyceride levels in Group 1 were  $2.3 \pm 0.5$  mmol/L, significantly higher than in patients with isolated IHD ( $1.8 \pm 0.4$  mmol/L,  $p < 0.05$ ). This indicates impaired lipid metabolism and an increased risk of atherosclerosis in patients with combined pathology. In patients with both IHD and CKD (Group 1), the atherogenic index was significantly higher at  $5.8 \pm 1.2$ , whereas in the group with isolated IHD (Group 2), this indicator was  $4.7 \pm 1.1$  ( $p < 0.05$ ). This confirms a more pronounced atherosclerotic process in Group 1.

Thus, patients with combined IHD and CKD exhibit more severe lipid metabolism disorders, characterized by elevated levels of atherogenic fractions (LDL, triglycerides) and decreased anti-atherogenic HDL. This underscores the necessity of aggressive dyslipidemia control and a comprehensive approach to the treatment of this patient category.

We analyzed urea, creatinine, and GFR data in the examined patients. Among the 65 patients with combined IHD and CKD, stratification by CKD stages was performed based on GFR according to KDIGO (2012): Stage II CKD (GFR 60–89 mL/min/1.73 m<sup>2</sup>) was observed in 15.4% of patients, Stage IIIa (GFR 45–59 mL/min/1.73 m<sup>2</sup>) in 33.8%, and Stage IIIb (GFR 30–44 mL/min/1.73 m<sup>2</sup>) in 50.8%. Stage III CKD (IIIa and IIIb) was present in 84.6% of patients,

reflecting moderate renal dysfunction often observed in patients with long-standing IHD. Stage II CKD was identified in 15.4% of patients and was mostly asymptomatic.

Thus, more than half of the patients (50.8%) had Stage IIIb CKD, indicating significant renal impairment requiring a comprehensive cardio-nephroprotective approach.

**Table №2.**

Indicator	CKD stage 2 (n=12)	CKD stage 3a (n=12)	CKD stage 3b (n=12)	p-value
Total cholesterol (TC), mmol/l	5,8 ± 1,0	6,0 ± 1,1	6,4 ± 1,3	>0,05
LDL, mmol/l	3,6 ± 0,8	3,9 ± 0,9	4,3 ± 1,0	<0,05
HDL, mmol/l	1,2 ± 0,3	1,0 ± 0,2	0,8 ± 0,2	<0,05

Total cholesterol did not show significant differences between groups ( $p > 0.05$ ), but a trend of increasing cholesterol levels with declining GFR was observed. LDL and triglycerides progressively increased with worsening kidney function, reaching their highest levels in Stage IIIb CKD ( $p < 0.05$ ). HDL significantly decreased with CKD progression ( $p < 0.05$ ), reflecting the decline in anti-atherogenic protection. The atherogenic index increased significantly with worsening renal dysfunction, indicating the intensification of atherosclerotic processes ( $p < 0.05$ ). These findings confirm the necessity of early dyslipidemia detection and correction in patients with IHD and CKD, particularly when GFR falls below 45 mL/min/1.73 m<sup>2</sup>.

As seen in the collected data, urea levels were significantly higher in Group 1 ( $p < 0.05$ ), indicating moderate azotemia characteristic of CKD Stage III. In Group 2, urea levels were within the normal range, confirming the absence of significant renal dysfunction. Serum creatinine was significantly elevated in Group 1 (142.5 µmol/L), corresponding to CKD Stage III according to the KDIGO classification. In Group 2, creatinine levels were within the normal range, indicating preserved kidney function. The estimated glomerular filtration rate (GFR) in Group 1 was approximately 45 mL/min/1.73 m<sup>2</sup>, which corresponds to CKD Stage III (moderate decline). In Group 2, GFR was 78.8 mL/min/1.73 m<sup>2</sup>. A decline in GFR in patients with both IHD and CKD is an important predictor of cardiovascular complications since GFR < 60 mL/min/1.73 m<sup>2</sup> increases the risk of cardiovascular events.

**Table №3.**

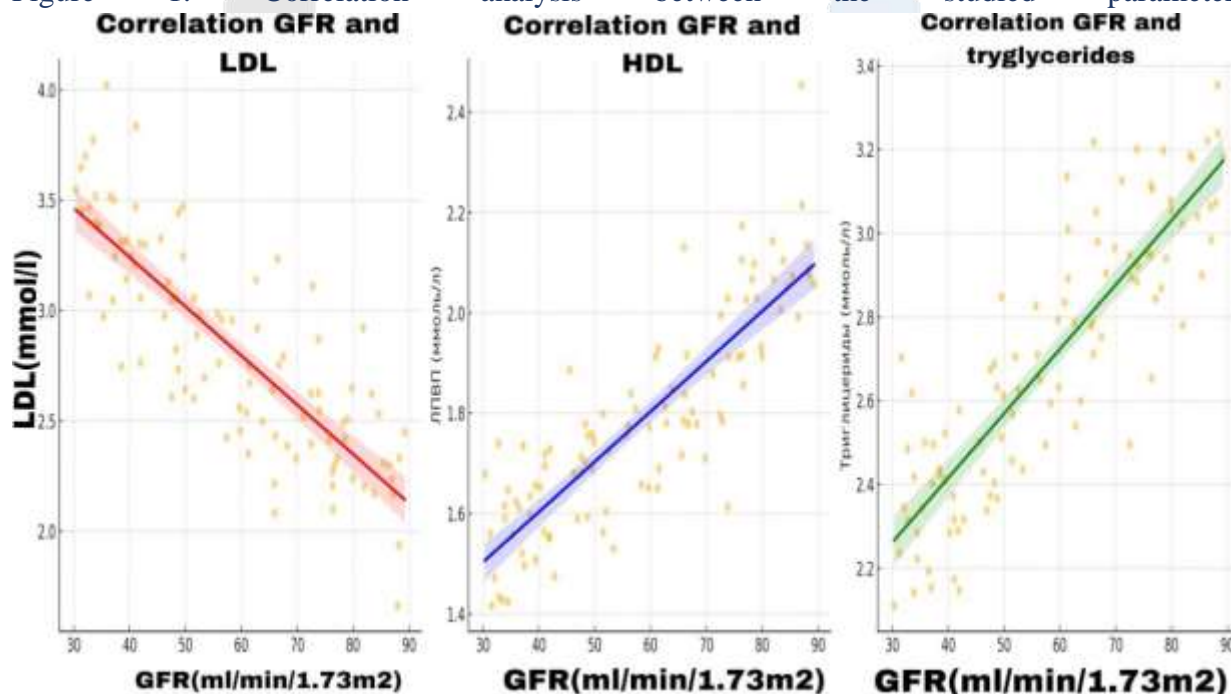
**Renal function indicators in patients with coronary artery disease and CKD**

Indicator	Group 1 (CHD + CKD)	Group 2 (CHD without CKD)	p (reliability)
Urea (mmol/l)	10,8 ± 2,9 (95% ДИ: 10,1 – 11,5)	5,8 ± 1,7 (95% ДИ: 5,3 – 6,3)	<0,05
Creatinine (µmol/l)	142,5 ± 28,3 (95% ДИ: 136,0 – 149,0)	86,5 ± 14,9 (95% ДИ: 82,2 – 90,8)	<0,05
GFR (ml/min/1.73 m <sup>2</sup> )	45,2 ± 6,8 (95% ДИ: 43,3 – 47,1)	78,8 ± 9,6 (95% ДИ: 76,0 – 81,6)	<0,05

Correlation analysis revealed the following key relationships between lipid metabolism markers in patients with different CKD stages:

A weak positive correlation with creatinine levels ( $r = 0.28$ ,  $p < 0.05$ ) suggests a potential influence of renal dysfunction on total cholesterol levels. However, this relationship is not strongly pronounced. A moderate negative correlation with GFR ( $r = -0.35$ ,  $p < 0.05$ ) indicates a tendency for total cholesterol levels to increase as kidney function declines. A significant negative correlation with GFR ( $r = -0.45$ ,  $p < 0.01$ ) confirms the atherogenic role of LDL in declining kidney function. The lower the glomerular filtration rate, the higher the LDL levels. A moderate positive correlation with creatinine levels ( $r = 0.39$ ,  $p < 0.05$ ) suggests a probable deterioration in lipid metabolism as CKD progresses. A significant positive correlation with GFR ( $r = 0.42$ ,  $p < 0.01$ ) indicates that HDL levels decrease with worsening kidney function, reducing anti-atherogenic activity. A negative correlation with creatinine levels ( $r = -0.37$ ,  $p < 0.05$ ) confirms the decline in protective HDL properties in patients with advanced CKD (Figure 1).

Figure 1. Correlation analysis between the studied parameters



Triglycerides (TG) exhibited a strong negative correlation with GFR ( $r = -0.52$ ,  $p < 0.01$ ), indicating that as kidney function declines, triglyceride levels increase significantly. A positive correlation with creatinine levels ( $r = 0.44$ ,  $p < 0.05$ ) suggests impaired fat metabolism in patients with renal dysfunction. The atherogenic index (AI) showed a strong negative correlation with GFR ( $r = -0.49$ ,  $p < 0.01$ ), demonstrating a substantial deterioration in lipid metabolism as kidney function declines. A significant positive correlation with creatinine levels ( $r = 0.41$ ,  $p < 0.05$ ) further indicates an increased atherogenic potential in patients with progressive renal failure.

#### Conclusion

As CKD progresses (with decreasing GFR), lipid metabolism parameters deteriorate: LDL, triglyceride levels, and the atherogenic index increase, while HDL levels decrease. The identified correlations emphasize the necessity of monitoring the lipid profile in CKD patients, as lipid

metabolism disorders are a significant risk factor for cardiovascular complications. CKD treatment strategies should include the correction of dyslipidemia, particularly in patients with low GFR, to reduce the risk of atherosclerotic complications.

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