

**O'ZBEKISTON RESPUBLIKASI  
SOG'LIQNI SAQLASH VAZIRLIGI**

**TOSHKENT TIBBIYOT AKADEMIYASI  
TERMIZ FILIALI**

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**“TIBBIYOTDAGI ZAMONAVIY ILMIY  
TADQIQOTLAR: DOLZARB MUAMMOLAR,  
YUTUQLAR VA INNOVATSIYALAR”  
MAVZUSIDAGI XALQARO ILMIY-AMALIY  
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**THE ROLE OF ALPHA-LIPOIC ACID IN THE TREATMENT OF  
DYSLIPIDEMIA IN PATIENTS WITH CORONARY ARTERY DISEASE**

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**Introduction:** Alpha-lipoic acid (ALA) is produced naturally in human organism. It plays an crucial role in [lipid metabolism](#). It is also a cofactor of enzymes in [carbohydrate metabolism](#). Recently, it has been shown that ALA supplementation might affect the lipid profile by influencing lipid metabolism. However, these findings remain controversial. The probable lipid-lowering mechanism might be explained by beneficial effects of ALA on mitochondrial fatty acid  $\beta$ -oxidation through activation of AMP-activated protein kinase.

**Materials and methods:** The total of 30 patients (12 males and 8 females) aged 46-67 with Coronary artery disease, FC II-III, patients without myocardial infarction and diabetes mellitus were chosen for the study. The study was carried out in Tashkent medical academy, in the department of vascular surgery in 2021-2022. We analyzed simple blood serum triglyceride (TGL), high density lipoprotein (HDL) and low-density lipoprotein (LDL) in patients with CAD before and after administration of alpha-lipoic acid, 300 mg capsule, twice daily, and atorvastatin 30 mg once daily at least a month.

**Results:** All the patients in the sample were divided into two groups based on the type of lipid lowering medicine received. 1<sup>st</sup> group consisted of 15 patients and average level of TGL, HDL and LDL are before therapy 212,4 mg/dl, 38,7 mg/dl and 185.8 mg/dl, 2<sup>nd</sup> group consisted of 15 patients and average level of TGL, HDL and LDL are 214,8 mg/dl, 36,7 mg/dl and 187,4 mg/dl. We give 1<sup>st</sup> group of patients atorvastatin 30 mg and 2<sup>nd</sup> group of patients atorvastatin 20 mg + alpha-lipoic acid 600 mg. After a 2 month we checked total level of TGL, HDL and LDL in blood serum in the both group of patients. 1<sup>st</sup> group of patients after therapy the level of TGL declined from 214,4 mg/dl to 175,7 mg/dl, HDL changed from 38,7 mg/dl to 44,7 mg/dl and LDL is dropped 135,3 mg/dl. 2<sup>nd</sup> group of patients after therapy the level of TGL declined from 214,8 mg/dl to 148,7 mg/dl, HDL changed from 36,7 mg/dl to 55,8 mg/dl and LDL is decreased from 187,4 mg/dl to 127,5. In the comparative analyses in both group of patients, we can see that from the obtained results in the 2<sup>nd</sup> group of patients the level of TGL and LDL significant declined compared to 1<sup>st</sup> group.

**Conclusion:** The high amount of TGL and LDL in the bloodstream may cause and increase the progression of atherosclerosis of coronary artery. Lipid lowering therapy plays an important role in the decreased the level of lipids. Through this prevalence of fatal outcomes such as myocardial infarction and ischemic stroke significant declined. It is evident that the noticeable reduction in 2<sup>nd</sup> group patients after combined therapy whereas there haven't been any noticeable changes in 1<sup>st</sup> group of patients after monotherapy. ALA might reduce TC or LDL by the following mechanisms: 1. Increased the activity of lipoprotein lipase: 2. Synthesis LDL receptors in the liver lead to transmission of cholesterol to the hepatic system and increased synthesis of Apo-lipoprotein A. 3. Increased plasma adiponectin levels, which improved free fatty acid  $\beta$ -oxidation.

Overall, our findings indicated that ALA supplementation did not show any significant effect on serum HDL.