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STATIN TREATMENT IN PATIENTS WITH CORONARY ARTERY DISEASE

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Relevance. Cardiovascular diseases (CVDs) are the leading cause of mortality globally, accounting for an estimated 17.9 million deaths annually. Low-density lipoprotein cholesterol (LDL-C) is a critical modifiable risk factor in the pathogenesis and progression of atherosclerosis. Statin therapy forms the cornerstone of secondary prevention in patients with acute coronary syndromes (ACS), including myocardial infarction (MI) and unstable angina (UA). Evaluating the real-world efficacy of such therapy is essential for optimizing clinical outcomes.

Aim: to assess the effect of statin therapy, primarily atorvastatin, on lipid profiles and clinical outcomes in patients diagnosed with MI and UA.

Materials and methods. A retrospective analysis was conducted at Clinical Hospital 4 in Minsk, Belarus. Data was collected from 41 patients diagnosed with either MI (n=28) or UA (n=13) between January 1 and August 31, 2025. All patients were prescribed atorvastatin (10–40 mg). A two-sample t-test was used to analyze changes in lipid profiles (total cholesterol, LDL-C, HDL-C, triglycerides), liver enzymes (ALT, AST), C-reactive protein (CRP), and glucose during hospitalization. A chi-square test was used to assess the association between diagnosis and comorbidities.

Results and their discussion. Statin therapy resulted in statistically significant reductions in total cholesterol and LDL-C in both MI ($p<0.001$) and UA ($p<0.001$) groups during hospitalization. HDL-C changes were also significant in both groups. No statistically significant changes were observed in triglycerides, ALT, AST, CRP, or glucose levels, suggesting that the observed effects were specific to the lipid-lowering pathway without notable short-term impact on these parameters. The overall treatment effect on LDL-C level was more statistically significant in the UA group ($p=0.0275$) compared to the MI group ($p=0.09$). This finding is consistent with the STELLAR study sub-analysis, which reported greater LDL-C reduction with statins in UA patients. This may reflect the benefits of early intervention in UA, stabilizing plaque and reducing progression to MI. A high prevalence of comorbidities was noted, with 19 patients having diabetes mellitus, 9 with kidney disease, and 38 with arterial hypertension. Chi-square analysis revealed no statistically significant association between the diagnosis (MI vs. UA) and the presence of diabetes, kidney disease, or hypertension. This indicates these conditions were evenly distributed as baseline risk factors across both ACS presentations. The study confirms the efficacy of atorvastatin in achieving significant LDL-C reduction in a real-world ACS population. However, the inverse relationship observed between age and LDL-C control highlights a critical care gap: younger patients, despite being at a potentially higher lifetime risk, showed poorer LDL-C control. This warrants urgent attention in secondary prevention strategies. Adherence to ESC guidelines was high, with widespread use of Class I recommended therapies (high-intensity statins, DAPT, ACEi/ARB, beta-blockers). However, the absence of SGLT2 inhibitor use represents a gap, particularly given their Class I recommendation for patients with concurrent heart failure or chronic kidney disease, both of which were present in the cohort. This underscores the need for individualized therapy that incorporates newer evidence-based agents.

Conclusions. Atorvastatin therapy significantly improves LDL-C levels in patients with MI and UA, with a more pronounced statistical effect observed in the UA group. While adherence to core guideline-directed medical therapy was strong, key gaps remain, including suboptimal LDL-C control in younger patients and the underutilization of novel therapies like SGLT2 inhibitors. These findings emphasize the importance of tailored secondary prevention to address residual risk and improve long-term cardiovascular outcomes.