

Lipoprotein (a) levels in patients with acute coronary syndrome under the age of 60 – a single-center clinical study

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Introduction: Lipoprotein (a) [Lp(a)] is a particle consisting of lipids and proteins, structured similarly to low-density lipoprotein (LDL), with the addition of apolipoprotein (a). Lp(a) promotes atherogenesis – it increases release of proinflammatory cytokines and infiltration of monocytes to the arterial wall, and decreases stability of atherosclerotic plaques. Its levels are genetically determined and relatively stable during lifetime. Levels >125 nmol/L are considered to be elevated. It is recommended to measure Lp(a) levels in individuals with premature cardiovascular disease (CVD), recurrent CVD despite optimal therapy, SCORE risk ≥5% or aortic valve stenosis.¹⁻³ **Aim:** To analyze Lp(a) serum levels in patients with acute coronary syndrome (ACS) under the age of 60.

Patients and Methods: Patients hospitalized due to ACS at the University Hospital Center Osijek, under the age of 60, were enrolled into the study.

Results: Lp(a) levels were measured in 21 patients (14 male, 7 female). Median Lp(a) levels for all patients were 36 nmol/L (3–560 nmol/L) with no significant difference between male and female (p=0.551). Lp(a) levels did not correlate with widely known risk or protective factors for CVD: age (p=0.172); body weight (p=0.437); body mass index (p=0.204); serum levels of total cholesterol (p=0.312), LDL cholesterol (p=0.541), HDL cholesterol (p=0.942), triglycerides (p=0.074); and did not differ depending on prior statin treatment. Nineteen patients were treated with percutaneous coronary intervention, 1 was appointed to surgical revascularization, and 1 did not require invasive treatment. The number of surgically treated patients was low and did not enable statistical analysis, but it should be emphasized that Lp(a) levels of the patient treated surgically were 206 nmol/L, compared to the median Lp(a) levels of 36 nmol/L for all enrolled patients.

Conclusion: Lp(a) represents additional risk factor for CVD and its levels do not correlate with traditionally known risk and protective factors for CVD. Although specific Lp(a)-lowering therapies are not yet available in everyday clinical practice, lipoprotein apheresis may be considered in patients with very high Lp(a) levels and progressive atherosclerotic disease despite optimal control of all other modifiable risk factors.

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LITERATURE

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