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THE ROLE OF C-REACTIVE PROTEIN IN THE FORMATION OF INFLAMMATION-DRIVEN LIPID PARADOX

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Relevance. Cardiovascular diseases remain the leading cause of death worldwide. In patients with a high inflammatory response, particularly with elevated C-reactive protein (CRP), a «lipid paradox» is often observed: low levels of atherogenic lipids (low-density lipoproteins (LDL), and total cholesterol (TC)) are paradoxically associated with a more severe course and a higher risk of complications. This makes it harder to assess cardiovascular risk and to choose an appropriate lipid-lowering strategy, especially in hospitalized patients with multiple comorbidities.

Aim: to assess the influence of CRP levels and systemic inflammation on the relationship between lipid profile parameters and cardiovascular risk, as well as between lipid profile parameters and the presence of cardiovascular diseases in hospitalized cardiology patients.

Materials and methods. A single-center cross-sectional study of 65 patients was conducted at the 6th Minsk City Clinical Hospital among patients with arterial hypertension; 52,3% also had type 2 diabetes mellitus. Patients with available CRP values (n = 41) were divided into three groups according to CRP level: <3 mg/L (n = 14), 3–10 mg/L (n = 12), and >10 mg/L (n = 15). Clinical data, lipid profile, inflammatory markers, and cardiovascular risk (SCORE) were assessed. Statistical analysis included the Kruskal–Wallis test, Mann–Whitney U test, χ^2 test, and Spearman correlation analysis. Inclusion criteria: age ≥18 years, established diagnosis of hypertension, hospitalization in a therapeutic department. Exclusion criteria: acute infectious diseases, severe liver failure, malignant neoplasms, and renal replacement therapy.

Results and their discussion. In patients with the highest CRP level (>10 mg/L), lower LDL (1,91 mmol/L) and TC (3,51 mmol/L) levels were observed compared with the group with moderate inflammation (CRP 3–10 mg/L: 3.10 and 4,88 mmol/L, respectively), which is consistent with inflammation-driven lipid suppression. A significant inverse correlation was found between CRP and high-density lipoproteins (HDL) ($r = -0,420$; $p = 0,006$), indicating particular vulnerability of the antiatherogenic fraction during inflammation. The classical «lipid paradox» was confirmed: patients with coronary heart disease (CHD) had significantly lower LDL levels than patients without CHD (2,33 vs 3,31 mmol/L; $p = 0,001$). These findings are in line with current data on the mechanisms of inflammatory suppression of lipid synthesis and the transformation of HDL into dysfunctional particles, and they support the need to interpret the lipid profile strictly in the context of inflammatory status.

Conclusions. High CRP levels (>10 mg/L) are associated with decreased LDL and TC levels, reflecting inflammation-driven suppression of lipid synthesis. A significant inverse relationship was established between CRP and HDL, confirming the vulnerability of the antiatherogenic fraction in systemic inflammation. In patients with CHD, LDL and TC levels are lower than in patients without CHD, which represents a manifestation of the «lipid paradox». In hospitalized patients, the lipid profile should be interpreted with consideration of CRP level, and in the presence of inflammation, repeated lipid testing is recommended after its normalization.