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Admission LDL Cholesterol and Short-Term Outcomes in Acute Coronary Syndrome: A Single-Centre Descriptive Study

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Introduction: Although lowering LDL cholesterol (LDL-C) consistently reduces long-term cardiovascular risk[1], low LDL-C measured during acute coronary syndrome (ACS) has paradoxically been associated with worse outcomes[2,3]. Whether low admission LDL-C identifies a higher-risk clinical profile rather than genuinely lower risk remains uncertain.

Patients and Methods: We retrospectively analyzed consecutive patients hospitalized with ACS at Merkur University Hospital, Zagreb, between 2022 and 2024. Patients were grouped by admission LDL-C (<2.6 versus ≥2.6 mmol/L) and compared using the Mann–Whitney U and Fisher's exact tests. Major adverse cardiovascular events (MACE: stroke, reinfarction, cardiovascular death or heart-failure hospitalization)[4] were recorded until the last available visit, and all-cause survival was compared using Kaplan–Meier analysis and the log-rank test.

Results: Of 79 patients, 23 (29%) had LDL-C <2.6 mmol/L (median 2.0) and 56 had LDL-C ≥2.6 mmol/L (median 3.6). Patients with low LDL-C more often had hypertension (87% versus 46%; p=0.001), diabetes (48% versus 16%; p=0.009), statin therapy at admission (61% versus 9%; p<0.001), prior ACS (26% versus 4%; p=0.007) and NSTEMI presentation (22% versus 4%; p=0.02), and their admission LVEF was lower (median 45% versus 55%; p=0.04). Age (72 versus 62 years; p=0.06), HDL cholesterol (1.0 versus 1.2 mmol/L; p=0.09) and SYNTAX score[5] (19 versus 16.5; p=0.28) did not differ significantly. Over a median follow-up of 193 days (IQR 120–307), MACE occurred in 8/23 versus 5/56 patients (35% versus 9%; p=0.008), whereas mortality did not differ significantly (26% versus 16%; p=0.35), with six-month Kaplan–Meier survival of 78% versus 93% (log-rank p=0.33).

Conclusion: Contrary to conventional expectations, lower admission LDL-C was not associated with more favorable outcomes, which is consistent with the lipid paradox.[2,3] The excess risk appears to stem largely from the accompanying comorbidity burden, including established coronary disease, diabetes, reduced LVEF and prior statin therapy, while in acute illness low LDL-C may not be protective at all, reflecting instead frailty, malnutrition or advanced atherosclerosis.[2,3] Traditional lipid parameters may therefore be insufficient for risk assessment in patients with a high clinical burden but no overt dyslipidemia. Lipoprotein(a), an increasingly recognized contributor to residual risk despite statin therapy[6], was not yet measured routinely during the study period, so its contribution remains unknown. Admission LDL-C should be interpreted within the overall clinical context, without questioning the benefit of intensive lipid lowering.[1]

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