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ORIGINAL RESEARCH

48. The Lipid Paradox in 5,060 Hospitalized Patients: An Inverse Association Between C-Reactive Protein and Cholesterol in a Tertiary Care Cohort from 2020 to 2024

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Background. Systemic inflammation is theorized to alter lipid metabolism through cytokine-driven suppression of hepatic synthesis, changes in lipoprotein clearance, and impaired reverse cholesterol transport. These theoretical changes include reductions in low-density lipoprotein (LDL), high-density lipoprotein (HDL), and total cholesterol (TC) with elevations in triglycerides (TG). While these mechanisms are biologically plausible and supported in experimental settings, large-scale inpatient data confirming these patterns remain limited. Clarifying how lipid profiles shift during acute inflammation is essential for accurate cardiovascular risk assessment and interpretation of lipid panels during illness.

Methods. We conducted a retrospective analysis of electronic medical record data from a tertiary academic medical center between 2020 and 2024. A total of 5,060 hospitalized adult patients with C-reactive protein (CRP) >5 mg/L and at least one lipid panel were included. Patients were stratified into CRP categories: 5–20, 20–50, 50–100, and >100 mg/L. Mean values for TG, LDL, HDL, and TC were compared across groups using one-way ANOVA. Tukey HSD post-hoc testing was performed to evaluate between-group differences.

Results. Among the 5,060 patients included, th mean age was 50.5 years, and 53.4% were female. As CRP increased, LDL declined from 105.53 mg/dL in the 5-20 mg/L group to 87.94 mg/dL in the >100 mg/L group, and TC declined from 187.04 to 157.28 mg/dL. HDL also showed a consistent inverse relationship with CRP, decreasing from 48.89 to 38.68 mg/dL across categories. ANOVA revealed significant group differences for LDL ($F = 68.53$, $p < 0.0001$), HDL ($F = 94.83$, $p < 0.0001$), TC ($F = 99.39$, $p < 0.0001$), and TG ($F = 5.39$, $p = 0.0011$). Tukey post-hoc tests showed significant LDL reductions between the 5-20 and >100 mg/L groups (mean difference = –17.60 mg/dL, $p < 0.0001$) and the 20-50 and >100 mg/L groups (mean difference = –9.72 mg/dL, $p < 0.0001$). For TC, the 5-20 and >100 mg/L comparison showed a statistically significant reduction (mean difference = –29.77 mg/dL, $p < 0.0001$). For HDL, significant reductions were observed between the 5-20 and >100 mg/L groups (mean difference = –10.21 mg/dL, $p < 0.0001$), 20-50 and >100 (mean difference = –7.58 mg/dL, $p < 0.0001$), and 50-100 and >100 (mean difference = –3.09 mg/dL, $p = 0.0023$).

Conclusion. This study demonstrates a clear inverse association between CRP levels and lipid values, particularly LDL, HDL, and TC, in hospitalized patients. These patterns reflect inflammation-induced suppression of lipoprotein synthesis and metabolic remodeling. Lipid profiles obtained during inflammatory states may underestimate baseline atherosclerotic cardiovascular risk, highlighting the importance of contextual interpretation to avoid delayed or missed preventive interventions.

Figure 1. Mean Lipid Levels by CRP Category in Hospitalized Patients (N = 5,060)

CRP Category (mg/L)	Triglycerides (mg/dL)	LDL (mg/dL)	HDL (mg/dL)	Total Cholesterol (mg/dL)
5–20	174.31	105.53	48.89	187.04
20–50	163.55	97.66	46.26	174.11
50–100	169.16	89.57	41.78	160.45
>100	186.74	87.94	38.68	157.28
ANOVA F-value	5.39	68.53	94.83	99.39
ANOVA P-value	0.0011	<0.0001	<0.0001	<0.0001

Legend: Mean triglyceride, LDL, HDL, and total cholesterol levels are shown across four CRP categories (5–20, 20–50, 50–100, >100 mg/L). One-way ANOVA revealed significant inverse associations between CRP and LDL ($F = 68.53$, $p < 0.0001$) and total cholesterol ($F = 99.39$, $p < 0.0001$), supporting a lipid-lowering effect of systemic inflammation.

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