Low serum level of high-density lipoprotein cholesterol is a poor prognostic factor for severe sepsis.

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Abstract

OBJECTIVE: To assess the initial serum levels of lipids and lipoproteins and their correlations with the clinical outcome for patients with severe sepsis. The ability of high-density lipoprotein (HDL) to attenuate lipopolysaccharide (LPS)-induced cytokine production was also examined in vitro.

DESIGN: Prospective, observational cohort study.

SETTING: Medical intensive care unit (ICU) of a tertiary-level university hospital.

PATIENTS: Sixty-three consecutive patients with severe sepsis.

INTERVENTIONS: Blood samples were drawn within the first day of severe sepsis and the subsequent 14 days. Clinical outcome, including length of ICU stay, infection subsequent to hospital stay, and death, were monitored prospectively.

MEASUREMENTS AND RESULTS: Compared with the survivors, patients who died within 30 days had significantly lower levels of HDL cholesterol and apolipoprotein A-I during the first 4 days of severe sepsis. On day 1, HDL cholesterol levels correlated inversely with interleukin-6 (r = -0.72; p < .01) and tumor necrosis factor (TNF)-alpha (r = -0.70; p < .01) concentrations. Not only the overall and sepsis-attributable 30-day mortality rates but also the risk of prolonged ICU stay (>7 days) and the hospital-acquired infection rate were increased among patients with day 1 levels of HDL cholesterol of <20 mg/dL and apolipoprotein A-I of <100 mg/dL. Multivariate analysis identified an HDL cholesterol level of <20 mg/dL on day 1 (odds ratio, 12.92; 95% confidence interval, 2.73-61.29) and Acute Physiology
and Chronic Health Evaluation II score (odds ratio, 1.15; 95% confidence interval, 1.04-1.26) as independent predictors of the overall 30-day mortality rate. In human macrophages, LPS-induced TNF-alpha release was attenuated by incremental doses of HDL cholesterol added simultaneously (p < .01). However, HDL failed to suppress LPS-induced TNF-alpha production when administered after macrophages were exposed to LPS.

**CONCLUSIONS:** A low HDL cholesterol level on day 1 of severe sepsis is significantly associated with an increase in mortality and adverse clinical outcomes. In cultured macrophages, HDL can attenuate LPS-induced TNF-alpha production only if added concomitantly with, but not after, LPS exposure.

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