HDL cholesterol efflux capacity is impaired in acute systemic inflammation independently of the etiology

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Objective: one of the primary antiatherogenic properties of HDL is its role in promoting reverse cholesterol transport (RCT), a process whereby excess cholesterol is removed from peripheral tissues and transported to the liver for excretion. Objective of this study was to analyze whether acute systemic inflammatory disease (sepsis), that have been associated with an increase cardiovascular risk, may affects the capacity of HDL to promote cholesterol efflux (CEC), first step of RCT. **Results**: HDL from 17 patients with sepsis of various etiology and 8 control subjects were tested for their cholesterol efflux capacity (CEC) via the four main pathways by using in vitro cell-based assays. Patient with sepsis displayed a significant increase in the inflammatory markers erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), together with a reduction in plasma lipid levels. HDL subclass distribution analysis showed a reduction in medium sized HDL and an increase in large HDL in patients with sepsis; the small pre-beta HDL were unchanged in sepsis patients. Aqueous diffusion (AD)-, SR-BI-, ABCG1-mediated CEC were significantly reduced in all patients with sepsis compared to control subjects (percent reduction in efflux were -26%, -43% and -23%, respectively; $p<0.01$). ABCA1-mediated CEC remained unchanged between patients and controls (mean percentage efflux ± SEM were 2.33±0.28% compared to 2.49±0.35%). **Conclusion**: subjects with acute systemic inflammation, irrespectively of the etiology of the syndrome, showed impairment of HDL AD-, SR-BI- and ABCG1-mediated CEC; such impairment appear to be the result of structural HDL changes and contribute to explain the accelerated atherosclerosis in these patients.