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한 승 엽

Lipid Metabolism in Chronic Kidney Disease

Seung-Yeup Han

Department of Internal Medicine, Kidney Institute, Keimyung University School of Medicine, Daegu, Korea

Cardiovascular disease (CVD) is the leading cause of death in patients with chronic kidney disease (CKD). Mortality from CVD in this population is approximately 10 to 20 times higher than that in the general population. Several factors contribute to cardiovascular disease in CKD. Chief among them are oxidative stress, inflammation, hypertension and dyslipidemia.

CKD is associated with profound dysregulation of lipid and lipoprotein metabolism. CKD, per se, primarily affects the metabolism of high-density lipoprotein (HDL) and triglyceride (TG)-rich lipoproteins. Considerable progress has been made in the understanding of the molecular basis of lipid and lipoprotein disorders in chronic kidney disease during the last decade. These advances have been fueled by the availability of molecular tools and their application in the studies of animal models of renal insufficiency. In the artery wall, oxidized or otherwise modified lipoproteins are engulfed by macrophages via scavenger receptors, a process that can lead to foam cell formation and atherosclerosis. HDL plays a major role in mitigating this process by limiting lipid/lipoprotein oxidation and by retrieving surplus cholesterol for disposal in the liver, a process commonly known as reverse cholesterol transport. In addition, HDL plays a major role in metabolism of triglyceride-rich lipoproteins by serving as an ApoC and ApoE donor for the nascent chylomicrons and VLDL, a process which is vital in metabolism of these triglyceride-rich lipoproteins. Moreover, HDL serves as a potent endogenous inhibitor of inflammation, platelet adhesion, and LDL oxidation.

CKD is associated with profound dysregulation of lipid metabolism which leads to marked alterations of plasma lipid/lipoprotein profile and atherogenic diathesis. The associated lipid disorders are accompanied by and largely because of dysregulation to several key enzymes and receptors.