Additional Systemic Inflammation Exacerbates NAFLD and Atherosclerosis Induced by Cholesterol Overload

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Non-alcoholic fatty liver disease (NAFLD) is common in the general population, and it occurs even more frequently in patients with metabolic syndrome]. Patients with NAFLD have an increased risk of cardiovascular disease (CVD) because these diseases share several risk factors and surrogate markers. In addition, NAFLD is often associated with atherosclerotic signs including the presence of carotid plaques and coronary arterial calcium.

Inflammation can play an important role in atherosclerosis progression. Moreover, in NAFLD, steatohepatitis is an important factor that suggests progression to advanced liver disease. However, it is unclear if systemic inflammation affects both atherosclerosis and NAFLD induced by hypercholesterolemia.

Cholesterol accumulation in both the liver and arteries augments systemic inflammation in patients with hypercholesterolemia. Recent studies lend some clarity to the role of inflammation in driving the atherogenic response to hypercholesterolemia and suggest that this process is initiated in the bone marrow and spleen. In response to hypercholesterolemia, both the bone marrow and spleen overproduce inflammatory monocytes that enter the circulation, accumulate in atherosclerotic lesions, and differentiate into macrophages. The high-cholesterol diet not only increased proinflammatory cytokine and CRP levels in the blood, but also resulted in the development of early pathologic changes of NAFLD and atherosclerosis.

The addition of systemic inflammation to hypercholesterolemia simultaneously exacerbated atherosclerotic lesions within the aorta and hepatic lesions.