Pathophysiology of atherosclerosis and novel imaging strategy

김 진 원
고려의대 순환기내과

Jin Won Kim, MD, PhD
The Cardiovascular Center, Korea University Guro Hospital, Seoul, Korea

Atherosclerotic plaque formation is initiated by the retention of retention of apolipoprotein B-containing cholesterol-rich lipoproteins, particularly at branch points of the arteries. The accumulation of these lipoproteins triggers resident macrophage activation and recruitment of monocyte-derived Ly6C^{high} cells from bone marrow and splenic hematopoietic stem cells into the subendothelial space, where they differentiate into mononuclear phagocytes that uptake the the lipoproteins. These cells become grossly engorged following ingestion of the lipids, transforming into foam cells. Vascular smooth muscle cells also become macrophage-like foam cells, although their behavior are largely not understood. While efficient efferocytosis coupled with apoptosis of foam cells in atheromatous plaque could be beneficial, however, in advanced stage, the ability of macrophages to clear their dying counterparts becomes compromised which attributed to necrotic core formation and thinning of protective fibrous cap causing fatal thrombotic complications.
Multi-modal biological imaging technology combined with morphological information, albeit still in its early stage, is expected to shed a light in the precise diagnosis of coronary atherosclerosis. To answer these unmet needs, our group has developed the fully integrated catheter system to simultaneously image plaque morphology and inflammation based on a double cladding optical fiber. In addition, we recently developed macrophage-targeted PPARγ activation strategy which is able to specifically activate PPARγ pathway within the inflamed atheroma. These novel imaging and treatment strategy could be a promising approach for the high risk atheroma.