

Inflammation worsens danger due to atherosclerosis

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Current research suggests that inflammation increases the risk of plaque rupture in atherosclerosis. The related report by Ovchinnikova et al, "T cell activation leads to reduced collagen maturation in atherosclerotic plaques of ApoE-deficient- mice," appears in the February 2009 issue of *The American Journal of Pathology*.

Atherosclerosis is a disease of arterial blood vessels where fats, cholesterol, blood cells, and fibers form hardened plaques on the artery wall. These plaques restrict blood flow to tissues such as the heart and brain by narrowing the artery. Atherosclerosis can be caused by high blood pressure, high fat and high cholesterol diets, smoking, and diabetes. People with atherosclerotic plaques often show no symptoms for decades.

Atherosclerotic plaques consist of lipid cores covered by collagen fiber caps. These plaques can suddenly rupture, resulting in blood clots that completely block blood flow and lead to heart attack or stroke in otherwise healthy individuals. One potential cause of plaque rupture is the thinning of the collagen fiber cap covering the plaque.

Inflammatory cells are often observed at the site of plaque rupture. Researchers led by Dr. Göran K Hansson at the Karolinska Institute explored the role of inflammatory cells in atherosclerotic plaque rupture using an animal model of atherosclerosis with hyper-activated immune cells. They found that inflammation leads to a reduction of mature collagen in atherosclerotic plaques, leading to thinner caps that are more

likely to rupture. They then identified a collagen-maturing enzyme, lysyl-oxidase (LOX), which represents a novel target in inflammation-induced plaque rupture.

The data from Ovchinnikova et al suggest "a novel mechanism by which adaptive immunity can modulate plaque stability - impairment of collagen maturation by T cell-dependent inflammation." These studies help unraveling the cause of myocardial infarction and stroke, and provide "interesting new targets for plaque stabilization therapy." In future studies, Dr. Hansson's group will explore the role of insufficient collagen maturation in human atherosclerosis. They hope that components of collagen and the LOX enzyme will become useful both to identify patients at risk for plaque rupture and to develop new therapy to prevent plaque rupture and thrombosis.

Source: American Journal of Pathology

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